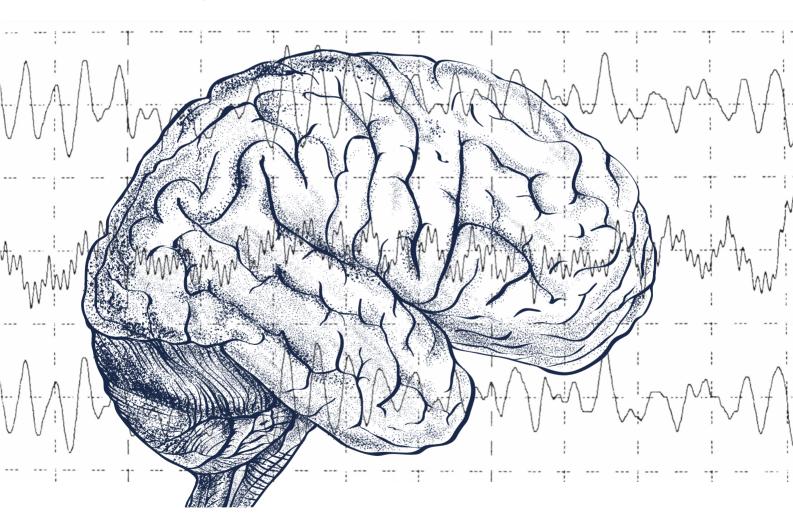
SLEEP Official Publication of the Sleep Research Society

VOLUME 43, 2020 | ABSTRACT SUPPLEMENT





34th Annual Meeting of the Associated Professional Sleep Societies



SLEEP

JOURNAL OF SLEEP AND SLEEP DISORDERS RESEARCH

Volume 43 Supplement 1 | April 10, 2020 | Pages 1-534

Official publication of the Sleep Research Socitety.

Editor-in-Chief

Ronald Szymusiak, PhD

Deputy Editors-in-Chief

Rachel Manber, PhD

Naresh M. Punjabi, MD, PhD

Executive Director

John A. Noel

Associate Editors

Alon Avidan, MD, MPH Fiona Baker, PhD Mary A. Carskadon, PhD Michael W. L. Chee, MD, PhD Ronald D. Chervin, MD Chiara Cirelli, MD, PhD Ian M. Colrain, PhD	Stephanie Crowley, PhD Thien Thanh Dang-Vu, MD, PhD Luis de Lecea, PhD Christopher Drake, PhD Raffaele Ferri, MD James E. Gangwisch, PhD Philip Gehrman, PhD	Namni Goel, PhD Daniel J. Gottlieb, MD, MPH David Gozal, MD Reut Gruber, PhD Andrew D. Krystal, MD Hans-Peter Landolt, PhD Jennifer Martin, PhD	Nathaniel Marshall, PhD Charles M. Morin, PhD John Peever, PhD Frank Scheer, PhD Carlos H. Schenck, MD Richard J. Schwab, MD Katie Stone, PhD	Mariana Szklo-Coxe, PhD Eus J. W. Van Someren, PhD Kenneth P. Wright Jr., PhD		
Editorial Board						
Monica L. Andersen, PhD J. Todd Arnedt, PhD Sara J. Aton, PhD Rashmi Aurora, MD M. Safwan Badr, MD Siobhan Banks, PhD Celyne H. Bastien, PhD Dean W. Beebe, PhD Bei Bei, PhD Richard B. Berry, MD Bjorn Bjorvatn, MD, PhD Donald L. Bliwise, PhD Orfeu Buxton, PhD Julie Carrier, PhD Peter Catcheside, PhD Phillip Cheng, PhD Yves Dauvilliers, MD, PhD	Jeffrey Donlea, PhD Leslie C. Dort, MSc, DDS Sean Drummond, PhD Jeanne F. Duffy, PhD Bradley Edwards, PhD Julio Fernandez-Mendoza, PhD Constance Fung, MD Peter C. Gay, MD Michael A. Grandner, PhD, MTR, CBSM Monica Haack, PhD Rosemary S. Horne, PhD Reto Huber, PhD Alex Iranzo, MD Shahrokh Javaheri, MD Dayna Johnson, PhD Athanasios Kaditis, MD	Leila Kheirandish-Gozal, MD Christopher Kline, PhD Gerrit Lammers, MD Miranda M. Lim, MD, PhD Peter Y. Liu, MBBS, PhD Steven W. Lockley, PhD Faith Luyster, PhD Mark Mahowald, MD Bryce A. Mander, PhD Janna Mantua, PhD George Mashour, MD, PhD Geert Mayer, MD W. Vaughn McCall, MD Thomas A. Mellman, MD David N. Neubauer, MD Seiji Nishino, MD, PhD Jason C. Ong, PhD	Thomas Penzel, PhD Michael L. Perlis, PhD Dante Picchioni, PhD Gina R. Poe, PhD Hengyi Rao, PhD Timothy A. Roehrs, PhD Aysa Rolls, PhD Jared Saletin, PhD Paula K. Schweitzer, PhD Kazue Semba, PhD Paul J. Shaw, PhD Renee Shellhaas, MD, MS Priyattam J. Shiromani, PhD Michelle Short, PhD Adam Spira, PhD Robert Stickgold, PhD Marie-Pierre St-Ong, PhD	Ariel Tarasiuk, PhD Robert J. Thomas, MD Liat Tikotzky, PhD Lynn-Marie Trotti, MD, MSc Adrienne Tucker, PhD Christa J. Van Dort, PhD Sigrid C. Veasey, MD Olivia J. Veatch, MS, PhD Vladyslov Vyazovkliy, PhD Arthur S. Walters, MD Emerson Wickwire, PhD Jonathan P. Wisor, PhD Amy R. Wolfson, PhD James K. Wyatt, PhD		

Statistical Editorial Board

Brendan Keenan, PhD Kwang-Youn A Kim, PhD Robert T. Krafty, PhD June C. Lo, Ph.D. Stefania Mondello, MD, MPH, PhD Lucia Peixoto, PhD Dale L. Smith, PhD Jacek K. Urbanek, PhD Meredith L. Wallace, PhD Wei Wang, PhD Joshua F. Wiley, PhD

Reviews Editors

Safwan Badr, MD

Michael Grandner, PhD

Table of Contents

Abstracts by Category (click on any section to jump to it)

A. Basic and Translational Sleep Science

- I. Mechanisms of Sleep and Circadian Disorders... 1 Abstracts 0001-0015

- Abstracts 0173-0263
- XI. Sleep Deprivation, Loss and Disruption...... 101 Abstracts 0264-0317
- XII. Sleep and Chronobiology Across the Lifespan ... 121 ABSTRACTS 0318-0355
- XIII. Disparities in Sleep and Circadian Health 136 Abstracts 0356-0379
- XIV. Population and Demographics 146 Abstracts 0380-0414
- XIV. Sleep and Neurodegeneration 159 Abstracts 0415-0430
- XV. Innovations in Sleep and Circadian Technologies.... 166 Abstracts 0431-0452

B. Clinical Sleep Science and Practice

I.	Insomnia	174
	Abstracts 0453-0554	
II.	Sleep-Related Breathing Disorders	213
	Abstracts 0555-0737	

III. Hypersomnia	1
IV. Circadian Rhythm Sleep-Wake Disorders 295 Abstracts 0775-0786	5
V. RLS, Movement Disorders and Parasomnias300 Abstracts 0787-0817	0
VI. Adults: Sleep and Aging, Sleep and Gender312 Abstracts 0818-0872	2
VII. Pediatrics	3
VIII. Sleep and Medical Disorders	2
IX. Sleep and Psychiatric Disorders	5
X. Sleep and Neurologic Disorders	6
XI. Healthcare Delivery and Education	4
XII. Consumer Technology	6
C. Case Reports	
Case Reports from Clinical Trainees	5
Indexes	
	_

Author Index	. 485
Keyword Index	. 522

iii

Welcome to your preview of SLEEP 2020, the 34th Annual Meeting of the Associated Professional Sleep Societies, which is scheduled to be held in Philadelphia, Pennsylvania on June 13-17, 2020.

This abstract supplement unites the journal *SLEEP*, and the science of SLEEP 2020. All abstracts presented at SLEEP 2020 are included in this special issue. This year 1,226 abstracts will be presented at the meeting. 196 will be presented in an oral presentation format, and the remainder will be presented in a poster format. Many authors of oral presentations will also be presenting their science in the poster hall, providing additional dedicated time to network with the authors of these important studies. In addition, this abstract supplement contains case reports submitted by individuals in Sleep Medicine Fellowship and other training programs.

Abstracts in this supplement are divided between Basic and Translational Sleep Science, and Clinical Sleep Science and Practice and then assigned to one of 28 subcategories. Each abstract has a unique four-digit number to facilitate identification and location both within this issue and at SLEEP 2020. The four-digit number in the abstract supplement matches the four-digit code published in the SLEEP 2020 Mobile App.

The SLEEP meeting fosters an environment in which members and attendees learn about the latest basic, translational and clinical science and technologies, promoting the continued growth of the field through the dissemination of new knowledge. As of the writing of this letter, the SLEEP meeting is still scheduled to be held. If the SLEEP meeting is canceled due to COVID-19, the dissemination of these abstracts through this supplement becomes even more important to the field as researchers and clinicians will not be able to present in person. We look forward to sharing this information with you in Philadelphia, Pennsylvania in June. But if this does not happen, we hope you stay healthy and look forward to seeing you in 2021.

Ronald Szymusiak, PhD

Editor-in-Chief

ACTIVATION OF NOCICEPTIN/ORPHANIN-FQ PEPTIDE (NOP) RECEPTORS PRODUCES AN INCREASE IN NON-REM SLEEP IN RATS AND CONSTITUTES A NOVEL AND ATTRACTIVE TARGET FOR THE TREATMENT OF INSOMNIA

*Whiteside, G. T.*¹ *Hummel, M.*² *Knappenberger, T.*² *Hiroyama, S.*³ *Itoh, T.*³ *Takai, N.*³ *Kyle, D. J.*²

¹Imbrium Therapeutics, Stamford, CT, ²Purdue Pharma L.P., Stamford, CT, ³Shionogi & Co., Ltd., Osaka, JAPAN.

Introduction: Treatments for insomnia have targeted GABA, histamine, serotonin, melatonin and orexin receptors. The nociceptin/ orphanin-FQ peptide (NOP) receptor is widely expressed in the nervous system. High doses of NOP agonists administered systemically or locally into the CNS can result in sedation, however, the utility of targeting this receptor to treat insomnia has not been fully described. Methods: V117957 is a recently described investigational oral, potent and selective NOP receptor partial agonist. We determined the brain Kp in whole brain and multiple sub-regions (50mg/kg) and receptor occupancy in the hypothalamus (30, 300mg/kg) via in vivo displacement using [3H]-NOP-1A. EEG/EMG were determined in rats chronically implanted with electrodes (cortex and dorsal neck muscle) and recorded via telemetry following dosing (3, 30, 300mg/kg); sleep stage was determined from visual analysis of EEG level. Sleep parameters were also assessed in NOP receptor knock-out rats (300mg/kg). The side-effect profile for V117957 was determined by functional observation battery, whole-body plethysmography, Morris water maze (MWM) (up to 600mg/kg) and conditioned place preference (CPP) assay (up to 300mg/kg).

Results: V117957 displayed limited distribution into the CNS but achieved a high level of receptor occupancy (75% at 30mg/kg). Administration of V117957 produced dose-dependent and statistically significant increases in non-REM sleep with a minimally efficacious dose of 30mg/kg; a coincident dose-dependent and statistically significant decrease in wakefulness and a non-dose-dependent effect on REM sleep occurred. These changes were not seen in knock-out animals demonstrating effects are via NOP receptors. At doses higher than those that increased non-REM sleep, V117957 had no effects in a functional observational battery, did not affect escape latency in MWM or produce CPP; additionally, V117957 did not affect respiratory parameters.

Conclusion: We conclude that activation of NOP receptors decreases wakefulness and increases non-REM sleep in rats with an improved preclinical profile compared to historical profiles of current treatments and, therefore, may represent a novel and attractive target for the treatment of insomnia.

Support: Funded by Shionogi and Imbrium Therapeutics, a subsidiary of Purdue Pharma L.P.

0002

DECREASED CONCENTRATION OF KLOTHO AND INCREASED CONCENTRATION OF FGF-23 IN THE CEREBROSPINAL FLUID OF PATIENTS WITH NARCOLEPSY

Oliveira, G. P.^{1,2} Elias, R. M.³ Fernandes, G. B.¹ Moyses, R.³ Tufik, S.¹ Bichuetti, D. B.¹ Coelho, F. M.¹

¹Universidade Federal de São Paulo, São Paulo, BRAZIL,

²Universidade Federal do Piauí, Teresina, BRAZIL,

³Universidade de São Paulo, São Paulo, BRAZIL, ⁴Universidade Federal de São Paulo, São Paulo, BRAZIL.

Introduction: Narcolepsy is a disorder characterized by hypersomnolence, cataplexy, sleep paralysis, hallucinations and sleep fragmentation. Patients with type 1 narcolepsy have cataplexy and/or hypocretin-1 deficiency. Klotho is a protein expressed by kidneys and choroid plexus, with anti-aging properties. Fibroblast growth factor 23 (FGF-23) is a hormone secreted by osteocytes with actions on mineral metabolism. The purpose of study was to explore the status of concentration of klotho and FGF23 in the cerebrospinal fluids (CSF) of patients with narcolepsy.

Methods: 59 patients with narcolepsy and 17 individuals were enrolled. We used a radioimmunoassay technique, human klotho enzyme-linked immunosorbent assay (ELISA), human intact FGF23 ELISA and spectrophotometry to measure hypocretin-1, klotho, FGF-23 and phosphorus, respectively. T-Student Test was used to compare klotho and phosphate concentrations and Mann-Whitney U Test was used to compare FGF-23 levels between groups. ANOVA Test was used to compare klotho and phosphate CSF concentrations among narcolepsy patients with CSF hypocretin-1 <110pg/ml (HCRT-) and narcolepsy patients with CSF hypocretin-1 >110pg/ml (HCRT+) versus control subjects.

Results: Klotho and phosphorus CSF levels were lower in narcoleptic patients than in control (908.18 ± 405.51 versus 1265.78 ± 523.26 pg/ml; p=0.004 and 1.34 ± 0.25 versus 1.58 ± 0.23 mg/dl; p= 0.001, respectively). We found higher median FGF-23 levels in narcoleptic patients (5.51 versus 4.00 RU/ml; p= 0.001). Klotho and phosphorus CSF levels were lower in both HCRT-/HCRT+ than controls (892.63 ± 388.34/ 925.95 ± 430.76 versus 1265.78 ± 523.26 pg/ml; p=0.014 and $1.35 \pm 0.28/1.33 \pm 0.22$ versus 1.58 ± 0.23 mg/dl; p= 0.004). Moreover, we found higher median FGF-23 levels in both HCRT-/HCRT+ groups versus controls (5.51/ 6.02 versus 4.00 RU/ml in controls), p= 0.009.

Conclusion: Patients with narcolepsy have decreased CSF concentration of klotho and increased CSF levels of FGF-23. These findings may play a role in understanding the pathogenesis of narcolepsy.

Support: .

0003

LGI1 AND CASPR2 AUTOIMMUNITY: SLEEP SYMPTOMS, POLYSOMNOGRAPHY, AND QUANTITATIVE REM SLEEP WITHOUT ATONIA

Devine, M. F. Feemster, J. C. Lieske, E. A. McCarter, S. J. Sandness, D. J. Steele, T. Boeve, B. F. Silber, M. H. McKeon, A. St. Louis, E. K. Mayo Clinic, Rochester, MN.

Introduction: Sleep disturbances, including rapid eye movement (REM) behavior disorder (RBD), are known manifestations of voltage-gated-potassium-channel-complex VGKC-IgG seropositivity (VGKC+). Discovery of leucine-rich, glioma inactivated protein 1 (LGI1) and contactin-associated protein 2 (CASPR2) have refined our understanding of VGKC+. VGKC+ without LGI1/CASPR2-IgG ("double-negative") has lost its clinical significance. Previous detailed sleep analysis of these subtypes has been limited.

Methods: We performed a retrospective study to characterize clinical and polysomnographic features of LGI1/CASPR2 sero-positive (LGI1+/CASPR2+) and VGKC double-negative patients, including quantitative REM sleep without atonia (RSWA). Quantified RSWA was compared to matched controls and normative RSWA percentiles.

Results: Eleven LGI1+/CASPR2+ (LGI1+, 9) and twelve VGKC double-negative patients were analyzed. Insomnia was seen in 55% of LGI1+/CASPR2+ and 8% of VGKC double-negative patients (p=0.05). The LGI1+/CASPR2+ group had reduced slow wave sleep compared to the VGKC double-negative group. Five LGI1+ patients had clinical dream enactment behavior (DEB). Eight LGI1+ patients met quantitative diagnostic levels of RSWA. Higher RSWA levels were seen in the LGI1+/CASPR2+ group. Ten LGI1+/CASPR2+ patients received immunotherapy; all ten neurologically benefited with sleep benefits in 6/10.

Conclusion: Sleep disorders such as insomnia and RBD are part of the LGI1/CASPR2 autoimmune phenotype. Objective sleep manifestations can be seen on polysomnogram in the form of reduced N3 and elevated RSWA as compared to controls. Quantitative RSWA analysis identified RBD in more LGI1+ patients than clinical report or qualitative RSWA. In this study, RBD was only seen with LGI1+, not CASPR2+. The intermediate RSWA levels of the VGKC double-negative patients may suggest a spectrum of abnormal motor activity in these related antibodies. Additional studies are needed to further explore the biomarker potential of quantitative RSWA in autoimmune neurological conditions.

Support: This project was supported by the National Center forResearch Resources, National Institutes of Health, through Grant Number 1 UL1 RR024150-01.

0004

TS-142: A NOVEL AND POTENT DUAL OREXIN RECEPTOR ANTAGONIST WITH SLEEP-PROMOTING EFFECTS IN RATS

Kambe, D. Hikichi, H. Tokumaru, Y. Ohmichi, M. Konno, Y. Hino, N.

Taisho Pharmaceutical Co., LTD., Tokyo, JAPAN.

Introduction: The orexin system plays a pivotal role in regulating sleep and wakefulness, thus, orexin receptors (OX1 and OX2 receptors) have gained much attention as promising therapeutic targets for the treatment of insomnia. We synthesized a novel and potent dual orexin receptor antagonist (DORA), ORN0829 (investigation code name as TS-142), which was designed to have short-acting effects. Here we report pharmacological and pharmacokinetic profiles of ORN0829 in rats.

Methods: The antagonistic activities of ORN0829 were assessed using calcium mobilization assays. Ala-orexin A-induced [Ca2+] i response was measured with CHO-K1 cells stably expressing human/rat orexin receptor. Rats implanted the EEG/EMG electrodes were orally administrated ORN0829 at doses of 1, 3 or 10 mg/kg at the dark onset and sleep-wake stages were inspected visually. In addition, pharmacokinetic profiles of ORN0829 were investigated in rats.

Results: ORN0829 inhibited Ala-orexin A-increased [Ca2+]i response with a Kb of 0.67/0.44 nmol/L (for human/rat OX1 receptor), and with a Kb of 0.84/0.80 nmol/L (for human/rat OX2 receptor), respectively, indicating that ORN0829 is a potent DORA with no species differences. ORN0829 dose-dependently increased total sleep time and reduced sleep onset latency at doses of 1, 3 and 10 mg/kg. Importantly, the ORN0829 levels in plasma and cerebrospinal fluid rapidly reached a maximum concentration, and decreased with an elimination half-life of less than 1 h.

Conclusion: The present study indicates that ORN0829 is a novel and potent DORA with sleep-promoting effects, and that it exhibits ideal pharmacokinetic profiles (rapid absorption and short half-life) in rats. A phase 2a study of TS-142 using patients with insomnia has been completed, which is presented in a separate poster.

Support: Taisho Pharmaceutical. Co., Ltd.

0005

CATAPLEXY TRIGGERED BY SOCIAL CUES: A ROLE FOR OXYTOCIN IN THE AMYGDALA

Mahoney, C. E.¹ Zhao, W.¹ Coffey, A.¹ Woods, C.¹ Kroeger, D.¹ Scammell, T.¹

¹BIDMC/Harvard Medical School, Boston, MA, ²BIDMC/ Harvard Medical School, Boston, MA.

Introduction: People with narcolepsy type 1 report that cataplexy is triggered most often by positive social experiences such as laughing with friends, yet the mechanisms through which social interaction promotes cataplexy are unknown. We hypothesize a subpopulation of central amygdala neurons that are sensitive to the prosocial neuropeptide, oxytocin (CeA^{OTR}), respond to positive valence and trigger cataplexy.

Methods: We have used in vivo calcium imaging, chemogenetic and optogenetic approaches to characterize the activity pattern of these neurons and to manipulate their activity state.

Results: Cre-dependent anterograde tracing of the CeA^{OTR} neurons of the central amygdala indicate a moderate to dense projection to the REM sleep-regulatory region of the ventral lateral periaqueductal gray (vlPAG). Additionally, Channel Rhodopsin Assisted Circuit Mapping (CRACM) experiments show that CeA^{OTR} neurons inhibit vlPAG neurons that innervate the REM atonia-promoting region, the sublaterodorsal nucleus. Targeted photostimulation (15Hz (10ms) for 20sec every hour) of the CeA^{OTR} fibers in the vlPAG doubled the amount of cataplexy. Preliminary *in vivo* calcium imaging indicates that the CeA^{OTR} are active just prior to the onset of cataplexy. Chemogenetic and optogenetic activation of CeA^{OTR} neurons increased cataplexy.

Conclusion: We conclude that the CeA^{OTR} subpopulation is sufficient to promote cataplexy. Our future directions include determining the necessity of these oxytocin sensitive neurons in cataplexy under different conditions of positive valence. **Support:** R01 NS106032 and WakeUp Narcolepsy.

0006

THE ROLE OF STRESS IN SLEEP IN NIGHT SHIFT WORKERS: GOING BEYOND CIRCADIAN MISALIGNMENT

Schaap, E. Sagong, C. Cuamatzi Castelan, A. S. Sayed, J. Roth, T. Drake, C. L. Cheng, P.

Henry Ford Health System, Detroit, MI.

Introduction: Despite a growing need for nighttime work, few studies have characterized the causes of sleep disturbance in night shift workers beyond circadian misalignment. Recent research suggest that high sleep reactivity to stress (a predisposition for sleep disturbance due to stress) may also lead to sleep difficulties in shift workers. This study investigated if sleep reactivity is an independent predictor of daytime sleep disturbances after controlling for circadian phase.

Methods: Night shift workers (N= 48) completed an 8 hour polysomnography (PSG) during the daytime following a night shift (9am - 4pm). Circadian phase was measured using melatonin assays of saliva samples collected over 24 hours under dim light (<10 lux; Dim Light Melatonin Onset [DLMO]). Sleep reactivity

was measured using the Ford Insomnia Response to Stress Test (FIRST). Linear regressions were conducted with PSG sleep parameters as outcome variables: difficulty falling asleep (Sleep Onset Latency [SOL] and Latency to Persistent Sleep [LPS]), difficulty staying asleep (Wake After Sleep Onset [WASO]), and sleep duration (Total Sleep Time [TST]). FIRST was tested as a predictor controlling for DLMO.

Results: After controlling for circadian phase, higher FIRST scores was associated with more difficulty staying asleep (WASO: t[45]=4.059, p<0.001) and shorter sleep duration (TST: t[45] = -4.403, p<0.0001), but not predictive of difficulty falling asleep (SOL: p>0.05). However, higher FIRST scores did predict a longer latency to persistent sleep (LPS: t[45]=2.272, p<0.05).

Conclusion: These results suggest that sleep reactivity to stress and circadian misalignment are independent processes that are both associated with disrupted daytime sleep in night shift workers. Given that night shift work can also cause psychosocial stress, treatments focused on circadian misalignment alone may not be sufficient. Our study highlights the need to consider sleep reactivity in the clinical management of shift work disorder.

Support: Support for this study was provided to PC by NHLBI (K23HL138166).

0007

DAYTIME SLEEP IN NIGHT SHIFT WORKERS: QUANTIFYING THE ROLE OF CIRCADIAN MISALIGNMENT

Mann, E. Sagong, C. Cuamatzi Castelan, A. Singh, M. Roth, T. Drake, C. L. Cheng, P.

Henry Ford Health System, Detroit, MI.

Introduction: Circadian misalignment is commonly cited as a culprit of daytime sleep disturbances in night shift workers; however, the specific impact and magnitude that circadian misalignment has on daytime sleep has not been well-characterized in larger samples of night shift workers.

Methods: Participants included fixed-night shift workers (n=52, ages 18–50) who completed an 8-hour daytime polysomnography (PSG) in the lab following a night shift. Measures of sleep disturbances included: difficulty falling asleep (sleep onset latency [SOL], latency to persistent sleep [LPS]), difficulty staying asleep (sleep efficiency [SE], wake after sleep onset [WASO]), and sleep duration (total sleep time [TST]). Melatonin samples were collected hourly for 24 hours under dim light (<10 lux) and used to determine dim light melatonin offset (DLMOff). Circadian misalignment (CM) was calculated as the time difference between bedtime and DLMOff (higher values represented sleeping after DLMOff), and correlated with PSG sleep variables.

Results: CM was significantly associated with difficulty staying asleep (WASO: r=0.48, p<0.001; SE: r=-0.45, p<0.001), and sleep duration (TST: r=-0.38, p<0.01). Specifically, every 3 hours of CM on average added 19.2 minutes of WASO and reduced TST by 15 minutes. In contrast, CM was not significantly correlated with sleep onset difficulties (SOL: r=-0.27; LPS: r=-0.02).

Conclusion: These data suggest that circadian misalignment in shift workers may be a better predictor of difficulties staying asleep and sleep duration during the day relative to difficulties falling asleep. Because longer work hours (10–12 hours) are common in night shift worker, it may be that sleep initiation difficulties associated with circadian misalignment is masked by elevated fatigue or an increased homeostatic drive from prolonged wakefulness. These results may help guide decisions about the magnitude of phase

shifts required (e.g., with light therapy) for the desired improvement in daytime sleep.

Support: Support for this study was provided to PC by the NHLBI (K23HL138166)

0008

SUVN-G3031, A HISTAMINE H3 RECEPTOR INVERSE AGONIST PRODUCES ROBUST WAKE PROMOTING AND ANTICATAPLECTIC ACTIVITY IN OREXIN KNOCKOUT MICE

Benade, V. Daripelli, S. Petlu, S. Subramanian, R. Bhyrapuneni, G. Shinde, A. Rasheed, M. Jayarajan, P. Choudakari, P. Nirogi, R. Suven Life Sciences, Hyderabad, INDIA.

Introduction: Narcolepsy is a sleep disorder characterized by excessive daytime sleepiness, sleep paralysis, hallucinations, and in some cases episodes of cataplexy. Results from animal studies indicate the involvement of deficient orexin transmission in narcolepsy which can be circumvented by the activation of histaminergic neurons. SUVN-G3031 is a potent and selective histamine H3 receptor inverse agonist with hKi of 8.7 nM and shows less than 50% inhibition at 1 µM against 70 other targets. SUVN-G3031 exhibited excellent pharmacokinetic properties and brain penetration in preclinical species. Oral administration of SUVN-G3031 produces significant increase in histamine, dopamine and norepinephrine levels in the rat cortex. Long-term safety studies in animals have been successfully completed without any concern for further development of SUVN-G3031. In the present study, the effects of SUVN-G3031 were evaluated in orexin knockout mice, a reliable animal model of narcolepsy as a proof-of-concept study for the treatment of narcolepsy with and without cataplexy.

Methods: Male orexin knockout mice (10 - 15 weeks old, 25 - 35 g at the time of surgery) were implanted with telemetric device for simultaneous monitoring of electroencephalography (EEG) and electromyography. Animals were allowed surgical recovery of 3 weeks prior to EEG recording. Effects of SUVN-G3031 (3 and 10 mg/kg, *p.o.*) were evaluated during active period of animals.

Results: SUVN-G3031 produced significant increase in wakefulness with concomitant decrease in non-rapid eye movement sleep in orexin knockout mice. SUVN-G3031 also significantly decreased the number of cataplectic episodes in orexin knockout mice.

Conclusion: Results from the current preclinical study provide a strong basis for the utility of SUVN-G3031 for the treatment of narcolepsy with and without cataplexy. SUVN-G3031 is currently being evaluated in a Phase 2 study as monotherapy for the treatment of narcolepsy with and without cataplexy (ClinicalTrials.gov Identifier: NCT04072380).

Support: None

0009

ANTI-STREPTOCOCCAL ANTIBODIES IN CHINESE PATIENTS WITH TYPE -1 NARCOLEPSY

Ding, Q. Li, J. Xiao, F. Zhang, C. Dong, X. Han, F. Peking University People's hospital, Beijing, CHINA.

Introduction: Narcolepsy type 1 (NT1) is considered to be an autoimmune disease, and streptococcal infection may be an environmental trigger. However, previous studies from Asian narcolepsy patients did not reveal elevated anti-streptolysin O [ASO]. The aim is to investigate whether large sample Chinese patients with NT1 have an increase in antistreptococcal antibody titers. **Methods:** A total of 214 narcolepsy patients and 360 healthy controls were recruited. All patients were DQB1*0602 positive with clear-cut cataplexy or had low CSF hypocretin-1. Participants were tested for ASO and anti DNAse B [ADB]. These patients were divided into five groups according to disease duration, including 29 patients less than 3 months; 25 from 3 months to 1 year; 40 from 1 to 3 years; 61 from 3 to 10 years and 59 patients over 10 years. Comparison was also made between children and adults with age matched controls, respectively.

Results: There were no significant differences between patients and healthy controls in regard to both ASO \geq 200 IU (19.2% vs. 16.9%, p = 0.50) and ADB \geq 480IU (9.8% vs. 10.3%, p = 0.86). For children narcolepsy patients, ASO positive rates(19.8% vs. 18%, p = 0.68) and ADB positive rates(10.4% vs. 12%, p = 0.72) had no differences compared to age matched controls. And no difference was observed in adult narcolepsy patients either, with ASO positive rates (18.5% vs. 13.8%, p = 0.39) and ADB positive rates (9.3% vs. 5.3%, p = 0.42) compared to age matched controls, respectively. ASO (ADB) positive rates had no significant differences among different disease duration groups(p= 0.55, 0.9).

Conclusion: It is indicated that positive rates of ASO and ADB were not significantly different between Chinese patients with NT1 and healthy controls, including recent onset cases and children. **Support:** The study was supported by the National Natural Science Foundation of China (No. 81420108002 and NO. 81570083)

0010

FUNCTIONAL BRAIN CONNECTIVITY ALTERATIONS IN RESTLESS LEGS SYNDROME ARE MODULATED BY DOPAMINERGIC MEDICATION

Tuovinen, N.¹ Stefani, A.¹ Mitterling, T.³ Heidbreder, A.¹ Frauscher, B.⁴ Gizewski, E. R.⁵ Poewe, W.¹ Högl, B.¹ Scherfler, C.¹ ¹Department of Neurology at Innsbruck Medical university, Innsbruck, AUSTRIA, ²Department of Neurology at Innsbruck Medical university, Innsbruck, AUSTRIA, ³Department of Neurology at Johannes Kepler University, Linz, AUSTRIA, ⁴Montreal Neurological Institute, Montreal, QC, CANADA, ⁵Department of Neuroradiology at Innsbruck Medical university, Innsbruck, AUSTRIA.

Introduction: Functional brain connectivity studies revealed alterations within thalamic, salience, and default mode networks in patients with restless legs syndrome. The objective of this study was to characterize functional connectivity and network topology in a large cohort of patients with restless legs syndrome compared to healthy controls, and to investigate the modulatory effect of dopaminergic treatment upon connectivity.

Methods: 82 patients with restless legs syndrome (untreated, n=30; on dopaminergic medication, n=42; on alpha-2-delta ligands as mono- or polytherapy combined with dopaminergic medication, n=10) and 82 individually age and gender matched healthy controls were studied with resting state functional MRI. Connectivity of twelve resting-state networks was compared with independent component analysis, and among 410 brain regions with graph theoretical modeling.

Results: Patients with restless legs syndrome showed significantly higher connectivity within salience (P=0.029), executive (P=0.001), somatomotor (P=0.050), and cerebellar (P=0.041) networks, as well as significantly (P<0.05) lower cerebello-frontal communication compared to healthy controls. Untreated patients had significantly (P<0.05) lower cerebello-parietal communication compared to healthy connectivity between the thalamus and

frontal regions were significantly increased in patients on dopaminergic medication compared to untreated patients and healthy controls (P<0.05).

Conclusion: Networks with higher intra-network connectivity (i.e. salience, executive, somatomotor, cerebellar) and lower between regions connectivity (i.e. cerebello-frontal, cerebello-parietal) in rest-less legs syndrome correspond to regions associated with attention, response inhibitory control, and processing of sensory information. Dopaminergic medication normalizes the altered cerebello-parietal communication and increases thalamic connectivity to the prefrontal cortex suggesting that these regions are associated with the emergence of symptoms in restless legs syndrome.

Support: The study was funded by a Grant from Translational Research

Fund of the government of Tyrol, Austria, and in-kind resources of the Medical University of Innsbruck.

0011

THE INFLUENCE OF OBSTRUCTIVE SLEEP APNEA SEVERITY AND SEX ON CEREBRAL PERFUSION

Turner, A. D.¹ Bubu, O. M.¹ Rapoport, D. M.² Varga, A. W.² Ayappa, I.² de Leon, M.³ Rusinek, H.¹ Glodzik, L.³ Jean-Louis, G.¹ Osorio, R.¹

¹New York University, New York, NY, ²Icahn School of Medicine at Mount Sinai, New York, NY, ³Weill Cornell Medical College, New York, NY, ⁴New York University, New York, NY, ⁵New York University, New York, NY.

Introduction: Obstructive Sleep Apnea (OSA) has been shown to initiate a pathological cascade negatively affecting the cardiovascular system, including cerebral circulation. There is limited data on OSA effects on regional brain function, though reduced global cerebral blood flow (CBF) has been observed among patients with OSA. However, there are few precise assessments. We hypothesized that regional CBF values are altered in OSA, and that sex influences the hypothesized relationship.

Methods: Participants from the NYU Center for Brain Health cohort (n=68; 57.4% female; mean age=66.32±6.84), representing cognitively healthy volunteers with OSA (AHI4% > 5/hr) and without, from several NIA-supported studies, completed evaluations including clinical, structural & functional high-resolution arterial spin labeling 3 tesla MRI scans. Hippocampal and temporal cortex CBF was assessed at baseline and after CO₂ challenge using a rebreathing protocol. Analyses were completed using one-way ANCOVA controlling for age and BMI.

Results: More men had OSA (82.8% vs 56.4%). Men without OSA showed a larger change in CBF after challenge in left (t=2.6, p=0.014) and right (t=2.4, p=0.021) hippocampus. Although the main analyses by severity level only boarded significance, pairwise comparisons indicated men with severe OSA (AHI4%>30/hr) exhibited a larger change in CBF after challenge in the hippocampus overall compared to those with mild OSA (AHI4%5–15/hr; p=0.015) and without OSA (p=0.017). Women with severe OSA showed a reduced change in CBF after challenge in the right hippocampus compared to those with mild (p=0.016), moderate (AHI4%16–29/hr; p=0.008), and without OSA (p=0.015).

Conclusion: This study suggests a possible differential effect of OSA severity and sex on regional CBF in response to a CO_2 challenge, specifically in the hippocampus. Further studies will examine cognitive consequences of these sex-specific hippocampal perfusion abnormalities in OSA.

Support: NIH/NIA (1R01HL118624) Osorio, RS 07/01/13-04/30/17 Sleep Disordered Breathing in normal elderly and risk for Alzheimer's disease (AD).NIH/NIA (R01AG056031S1) Osorio, RS 8/01/2019-7/31/2020 Sleep Aging and Risk for Alzheimer's disease - Research Supplement to Promote Diversity in Health-Related Research

0012

REM SLEEP IN OSTRICH CHICKS

Lyamin, O.^{1,2} Borshenko, V.³ Bakhchina, A.^{2,4} Siegel, J.¹ ¹UCLA and VA GLAHS Sepulveda, North Hills, CA, ²A.N. Severtsov Institute of Ecology and Evolution RAS, Moscow, RUSSIAN FEDERATION, ³Samara National Research University, Samara, RUSSIAN FEDERATION, ⁴Institute of Psychology RAS, Moscow, RUSSIAN FEDERATION.

Introduction: It was reported that adult ostriches displayed the longest REM sleep episodes (up to 5 min) and more REM sleep (24% of the nighttime) than any other avian species. In all mammals studied so far REM sleep predominates at early age suggesting it promotes development of the brain. The aim of this study was to examine REM sleep in ostrich chicks.

Methods: EEG, electrooculogram and electromyogram of the neck muscles were recorded in 4 chronically implanted 2–3 month old ostrich chicks over 3 nights. The last night was scored in 4-sec epochs for waking, nonREM and REM sleep.

Results: NonREM sleep and REM sleep in the ostrich chicks occurred when they were sitting or lying with the head held above the ground or rested on the ground. REM sleep was characterized by distinct rapid eye movements, head drops and eye closure. The amplitude of the EEG during episodes of REM sleep ranged between low voltage EEG, as recorded during quiet waking and high voltage slow waves, as recorded during nonREM sleep EEG. The ostrich chicks spent on average 70.7 + 2.2% of the nighttime in nonREM sleep and 12.3 + 3.9% in REM sleep. The episodes of REM sleep lasted on average 9 + 1 sec and ranged between 4 and 36 sec.

Conclusion: Similar to adult birds, 2–3 mo old ostrich chicks displayed a "mixed" sleep state which has features of both slow wave sleep / nonREM and REM sleep, as we have described in the platypus and echidna. An unexpected result of this study is the total amount and duration of episodes of REM were considerably smaller than has been reported in adult ostriches. More studies need to be done on the developmental and environmental determinants of REM sleep in the ostrich.

Support: The Russian Foundation for Basic Research (18-04-01252) and HL148574

0013

MOBILE DEVICE USE IN BED AND RELATIONSHIPS TO WORK PRODUCTIVITY: IMPACT OF ANXIETY

Gozar, A.¹ Seixas, A.² Hale, L.³ Branas, C.⁴ Barrett, M.⁵ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹

¹University of Arizona, Tucson, AZ, ²New York University, New York, NY, ³Stony Brook University, Stony Brook, NY, ⁴Columbia University, New York, NY, ⁵University of Pennsylvania, Philadelphia, PA.

Introduction: Mobile phone use at night is associated with worse sleep quality. It may also be associated with daytime productivity, possibly via anxiety.

Methods: Data were obtained from the Sleep and Healthy Activity, Diet, Environment, and Socialization (SHADES) study, including N=1007 adults age 22–60. Mobile device use in bed was assessed as the frequency that participants reported: a device in the bedroom, use of the device in bed, texting, emails, internet browsing, calls, and/or social networking in bed, being woken up by the device in a planned (alarm) or unplanned (alert/call/message) way, and checking the phone at night. Each of these were coded as "never," "rarely," or "often." Work productivity was assessed with the Well-Being Assessment of Productivity (WBA-P; scores 0–22 measure productivity loss). Regressions with WBA-P score as outcome and mobile phone variables as predictors were adjusted for age, sex, race/ethnicity, education, and income level. Post-hoc analyses included GAD7 score to examine the mediating role of anxiety.

Results: The presence of a device was not associated with productivity loss, but frequent use ("often") was (B=1.26,p=0.01). Increased productivity loss was also seen in those who frequently ("often") sent texts (B=1.20,p=0.008), browsed internet (B=1.14,p=0.01), emailed (B=2.09,p<0.0005), called (B=1.42,p=0.004), and used social media (B=1.26,p=0.004). Productivity loss was associated with being woken by a call/alert "rarely" (B=1.20,p=0.001) or "often" (B=1.72,p=0.005), but not by alarm. Checking the phone at night "rarely" (B=0.89,p=0.01) and "often" (B=1.73,p<0.0005) were also associated with productivity loss. When anxiety was entered into the model, all relationships except those with frequent emails and calls in bed became nonsignificant.

Conclusion: Anxiety may be the underlying cause for both increased mobile phone usage and reduced productivity. Reducing anxiety levels may indirectly aid in decreasing nighttime mobile phone use and increasing daytime productivity.

Support: The SHADES study was funded by R21ES022931 Dr. Grandner is supported by R01MD011600

0014

REDUCED CORTICAL THICKNESS AS A BIOMARKER OF DAYTIME SLEEPINESS IN MILD TRAUMATIC BRAIN INJURY

Dailey, N. S. Raikes, A. C. Alkozei, A. Grandner, M. A. Killgore, W. D. University of Arizona, Tucson, AZ.

Introduction: Sleep disruptions, including the increase of daytime sleepiness, are reported in roughly 70% of all individuals who have suffered a mild traumatic brain injury (mTBI). Prior research using magnetic resonance imaging (MRI) has identified associations between functional brain changes and daytime sleepiness following mTBI. In the present study, we aimed to identify whether structural differences in cortical thickness are associated with increased daytime sleepiness in adults with mTBI.

Methods: A total of 58 adults between 18 and 45 years of age (M=23.58 \pm 5.31) participated in the study, including 19 healthy controls and 39 individuals with a documented mTBI. Individuals with mTBI were further divided based on time-since-injury into a sub-acute (n=22) or chronic (n=17) group. Daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS) and cortical thickness was measured using high-resolution T1-weighted structural MRI. Whole-brain vertex-wise estimations of cortical thickness were calculated using FreeSurfer (v.6.0) and entered into a GLM to identify between-group differences in cortical thickness and the association with ESS.

Results: Significant differences in cortical thickness were found between the two mTBI groups (cluster-forming threshold p<.01;

cluster-wise threshold p<.05; two-tailed; FWE-corrected). Specifically, lower cortical thickness in the left hemisphere was found in the inferior parietal lobule (p=.01), precuneus (p=.03), and pars triangularis (p=.04) for the sub-acute, compared to chronic group. Furthermore, a significant negative correlation was found between ESS and cortical thickness in the inferior parietal lobule (r=-.55, p=.009) for the sub-acute mTBI group.

Conclusion: More daytime sleepiness was associated with reduced inferior parietal cortical thickness in those 2 to 12-weeks postinjury, an association not observed in those 6 to 12-months postinjury or healthy controls. The inferior parietal lobule is part of the frontoparietal attention network and has been associated with vulnerability to sleep loss. Our findings suggest structural damage to the attention network following mTBI may be one factor affecting daytime sleepiness in mTBI. These findings may reflect a potential biomarker of sleep disturbances in mTBI.

Support: USAMRMC grant (W81XWH-12-0386).

0015

MANIPULATING BODY TEMPERATURE: EFFECTS ON SLEEP IN POSTMENOPAUSAL WOMEN

*Reid, K. J.*¹ *Kräuchi, K.*² *Grimaldi, D.*¹ *Sbarboro, J.*¹ *Attarian, H.*¹ *Zee, P. C.*¹

¹Northwestern University, Center for Circadian and Sleep Medicine, Chicago, IL, ²Psychiatric University Clinics, Basel, SWITZERLAND.

Introduction: A decline in sleep quality and reduction in slow wave sleep (SWS) and slow wave activity (SWA) are common in older adults. Prior studies have shown that manipulating body temperature during sleep can increase SWS/SWA. The aim of this study was to determine the effects of manipulation of body temperatures during sleep, using a high heat capacity mattress, on SWS/SWA and heart rate variability in post-menopausal women.

Methods: Twenty-four healthy postmenopausal women between 40–75 years of age (mean age 62.4 ± 8.2 years, mean BMI 25.4 \pm 3.5 kg/m²) were randomized in a single-blind, counterbalanced, cross-over manner to sleep on either a high heat capacity mattress (HHCM) or a low heat capacity mattress(LHCM) a week apart. Sleep was recorded using polysomnography during an 8-hour sleep opportunity. Core and peripheral temperatures were recorded using Equivital and ibutton respectively.

Results: In comparison to the LHCM, sleep on HHCM exhibited a selective increase in SWS (average increase in Stage N3 of 9.6 minutes (2.1%), p = 0.04) and in slow oscillatory activity (0.5-1Hz) in the first NREM/REM cycle (p=0.04). In addition, the HHCM induced a greater reduction in core body temperature (p=0.002), and delayed the increase in mattress surface temperature (maximal difference LHCM-HHCM: 4.66±0.17°C). Average heart rate was 2.7 beats/minute lower across the night on the HHCM compared to the LHCM (p=0.001).

Conclusion: The results of this study indicate that manipulation of body temperature during sleep may be a useful approach to enhance SWS sleep and cardiovascular function in postmenopausal women. **Support:** Technogel

GENETIC BASIS OF DAYTIME NAPPING AND CONSEQUENCE ON CARDIOMETABOLIC HEALTH

Dashti, H. S.¹ Daghlas, I.¹ Lane, J.¹ Udler, M.¹ Garaulet, M.² Saxena, R.¹

¹Massachusetts General Hospital, Boston, MA, ²University of Murcia, Murcia, SPAIN.

Introduction: Although daytime napping is a common, evolutionarily conserved behavior, its genetic basis is unknown. Elucidating its genetic basis may clarify relevant underlying biological pathways and determine causal links with cardiometabolic health.

Methods: We performed a genome-wide association study of selfreported daytime napping using linear regression in adults of European ancestry in the UK Biobank (n=452,633) and assessed robustness of signals with accelerometer-derived daytime inactivity duration (n=84,671). Next, we conducted a phenome-wide association study in a hospitalbased clinical biobank (n=30,683) using napping genome-wide polygenic score (GPS), and Mendelian randomization (MR) with cardiometabolic traits. To deconstruct the napping genetic variants, we applied a novel "soft clustering" Bayesian nonnegative matrix factorization method and generated partitioned cluster-specific polygenic risk scores (PRS).

Results: We identified 121 distinct genome-wide significant loci for daytime napping, with lead signals at or near genes KSR2 (kinase-suppressor of ras 2), HCRTR1/HCRTR2 (hypocretinreceptor 1/2), SKOR2 (SKI family transcriptional-corepressor 2), and MAPT (microtubule-associated protein tau), among others. The loci associated with accelerometer-derived daytime inactivity duration. Gene enrichment analyses pointed to pathways involved in neurogenesis and others including nervous system development and opioid signaling. Genetic overlaps were evident in a clinical biobank where highest, compared to lowest, decile of napping GPS associated with 30%, 40%, and 50% higher odds for essential hypertension, obesity, and nonalcoholic liver disease, respectively (P<0.0001). In MR, potential causal links were identified with higher diastolic blood pressure (2.67 mmHg per napping categoryincrease, 95% CI 1.62-3.23, P=6.80e-07), systolic blood pressure (3.65mmHg, 1.86-5.44, P=6.40e-05), and waist circumference (0.28 SD-units, 0.11-0.45, P=0.0015). The clustering of variants identified 3 robust clusters (cluster-1: "higher sleep propensity"; cluster-2: "more fragmented/inefficient night sleep"; cluster-3: "early sleep timing"). Only clusters 2 and 3 PRSs were associated with worse cardiometabolic health outcomes, including higher BMI, waist circumference, CRP, and triglycerides (all P<0.05).

Conclusion: These findings expand our understanding of the genetic architecture of napping implicating multiple biological pathways, indicating possible genetic overlap and causal links to cardiometabolic traits, and suggesting distinct nap-promoting mechanisms with differential associations with health outcomes. **Support:** This work is supported by grants NIH-F32DK102323, NIH-4T32HL007901, NIH-R01DK107859, NIH-R35HL135818, and MGH Research Scholar Fund.

0017

TRANSMEMBRANE TNF- SOLUBLE TNF RECEPTOR REVERSE SIGNAL TO INDUCE A WAKE-LIKE STATE IN VITRO

Dykstra-Aiello, C. J. Koh, K. Nguyen, J. Krueger, J. M. Washington State University, Spokane, WA.

Introduction: Tumor necrosis factor (TNF) has sleep regulatory roles. Neuronal action potentials enhance TNF expression.

Neuron/glia co-cultures exhibit more intense local sleep-like states after TNF administration in vitro. Both TNF and TNF receptors (Rs) are produced as transmembrane (tm) proteins that can subsequently be cleaved to produce soluble (s) forms. With immunocytes, sTNFR can bind tmTNF and induce reverse signaling within the cell expressing the tmTNF. This is opposite of conventional signaling induced by soluble ligands (e.g. sTNF) binding to transmembrane receptors. Having previously shown sleep inhibition after sTNFR administration in vivo, we hypothesized that tmTNFsTNFR binding would induce wake-like states in vitro through reverse signaling.

Methods: Somatosensory cortical neurons/glia, from wildtype (WT) mice and mice lacking either TNF (TNF-KO) or both TNFRs (TNFR-KO), were co-cultured on multi-electrode arrays. Daily one-hour recordings were taken consecutively on incubation days 4 - 13 for development analyses. On day 14, a one-hour baseline was recorded prior to treatment with sTNFR (0.0 ng/µL-120 ng/µL). Immediately after treatment, recordings resumed for one hour. Synchronization of electrical activity (SYN), action potentials, slow wave power (SWP; 0.25–3.75 Hz), and burstiness index (measures used to define sleep in vivo) were used to characterize the ontological emergence of these electrophysiological properties and sTNFR-induced changes in vitro.

Results: Development rates were reduced in TNF-KO cells and increased in TNFR-KO cells relative to each other and to WT mice. Additionally, after sTNFR treatments, cells from TNFR-KO mice, which still express TNF, exhibited dose-dependent decreased SYN and SWP, indicative of a wake-like state. In contrast, cells from TNF-KO mice lacked a response to sTNFR treatment.

Conclusion: To our knowledge, this is the first demonstration of reverse TNF signaling with respect to sleep/wake states. As such, it provides a new way of viewing state regulation and associated potential clinical applications.

Support: This work was supported by grant NS096250 awarded to JK by NIH/NINDS.

0018

WHOLE GENOME SEQUENCING STUDY IDENTIFIES NOVEL VARIANTS ASSOCIATED WITH INTRINSIC CIRCADIAN PERIOD IN HUMANS

Smieszek, S. P.

Vanda Pharmaceuticals Inc., District of Columbia, DC.

Introduction: Non-24 is a circadian rhythm disorder in which the master body clock runs either slightly earlier or, more commonly in the disorder, longer than 24 hours.

Methods: We conducted the first whole genome sequencing study of a non-24 population of 174 individuals that we identified as being totally blind with Non-24 Disorder. We have directly tested the association between SNPs and circadian period length (tau) (n=69). Linear regression corrected for PCs and covariates identified a strong signal in HCN1, Brain Cyclic Nucleotide-Gated Channel 1, HCN1.

Results: HCN1 channel is responsible for the feedback on the rods regulating the dynamic range of light reactivity under dim or intermediate light conditions. Minor allele rs72762058 associated with longer tau, a difference of 12 minutes, and mean tau of 24.71. In *Drosophila* there is only one HCN channel encoding gene, *DmIh*. Interestingly, *DmIh* mutant flies display alterations in the rest:activity pattern, and altered circadian rhythms, specifically, arrhythmic behavior or a shorter period in constant darkness.

We report a variant that associated with longer tau. In addition, we identify others variants that strongly associate with tau, such as a missense variant (rs16989535), (minor allele associated with longer tau), within *DEPDC5*, GATOR Complex Protein). Subjects carrying the rare allele have a period > 25.2. DEPDC5 is part of GATOR1 complex, together with NPRL2 and NPRL3acts to inhibit the mTORC1 pathway. The GATOR1 seizure phenotype consists mostly of focal seizures, often sleep-related and drug-resistant and is associated with focal cortical dysplasia (20%). mTOR signaling is part of the photic entrainment pathway in the SCN, it regulates autonomous clock properties in a variety of circadian oscillators. Light-induced mTORC1 activation appears to be important for photic entrainment of the SCN clock, as rapamycin modulates light-induced phase shifts of wheel-running and body temperature rhythms in mice.

Conclusion: We identify variants in HCN1 and DEPDC5 implicated in significantly longer tau. Knowledge of the circadian clock and period length is not only essential for understanding of the basic clockwork mechanisms but also could provide insights into mechanistic links between circadian dysfunctions and human diseases such as epilepsy.

Support: Vanda Pharmaceuticals

0019

INVESTIGATING NOVEL-SLEEP RELATED GENES IN DROSOPHILA MELANOGASTER: A FOLLOW UP ON KOMP2 IDENTIFIED GENES IN MUS MUSCULUS

Guerriero, R.¹ Shaw, P.² O'Hara, B.¹

¹University of Kentucky, Lexington, KY, ²Washington University St. Louis, St. Louis, MO.

Introduction: Sleep is well-conserved across phylogeny, yet the function of sleep and its underlying mechanisms are currently poorly understood. Novel-sleep related genes were previously identified by our lab in part of the Knockout Mouse Phenotyping Program (KOMP2). This international effort generated single-gene knockouts on a *Mus musculus* C57BL6/NJ background and proceeded to gather data on over 200 phenotypes, including five days of baseline sleep and wake parameters. Sleep data was gathered using the non-invasive, high-throughput PiezoSleep System (Signal Solutions, LLC) which uses a piezoelectric film to gather movement data which then can be assigned to be wake or sleep. These data identified 122 novel genes that influence sleep phenotypes such as sleep duration and bout length.

Methods: Homologous proteins were identified and a subset of these genes are under investigation in *Drosophila melanogaster*, including myosin heavy chain (Mhc) and spinophillin (Spn). Using both genetic mutants and RNAi knockdowns, the effect of gene reduction on activity profiles and sleep are being analyzed. Sleep and activity data is recorded using DAM2 monitors (TriKinetics Inc.) while being maintained on a 12:12 light:dark cycle.

Results: Preliminary data analysis show that aberrations in Mhc and Spn impact sleep percentage. Both Mhc and Spn are known to be involved in structure and development of synapses. Spn is involved in the neurexin scaffolding of presynaptic neurons and also help with maintaining these synapses once formed. Synaptic reorganization and regulation is known to take place during sleep, showing a potential connection of these proteins and sleep.

Conclusion: These genes that show effects on sleep in both *D. melanogaster* and *M. musculus* show a conservation of the underlying sleep machinery. **Support:**

0020

DIFFERENTIALLY EXPRESSED GENES USING SALIVA SAMPLES FROM NURSES ROTATING SHIFTS

Imes, C. C. Monica, M. A. Chasens, E. R. Conley, Y. P. University of Pittsburgh, Pittsburgh, PA.

Introduction: Globally, millions of people work night or rotating shifts (i.e., shift work), including nurses and other healthcare providers. Shift work can cause insufficient and mistimed sleep which disrupts the normal circadian rhythm. Shift work is associated with an increased risk for cardio-metabolic disorders and certain cancers. This descriptive, single group, within-subject, repeatedmeasures study explored the effect of shift work on gene expression levels in a sample of female nurses engaged in rotating shifts. Methods: Saliva samples were collected from ten nurses without sleep or alertness medication use or a sleep disorder. The samples were collected using DNA Genotek RNA stabilizing saliva kits after participants worked at least 3 consecutive day shifts (~ 7:30 pm) and 3 consecutive night shifts (~7:30 am). Takara Smarter Stranded Total RNA Seq Kit was used following manufacturer's instructions on an Illumina NextSeq500. CLC Genomic Workbench 12 (Qiagen) was used for quality control, aligning the sequence reads, normalization, and differential expression analyses. Genes with log2 fold changes of \pm 2.0 were included in gene set enrichment and pathway analyses using Ingenuity Pathway Analysis (IPA; Qiagen).

Results: Participants were all female, white, and mostly healthy with a mean \pm SD age of 27.2 \pm 4.5 years. Compared to the postday shift samples, a total of 287 genes were differentially expressed at a log2 fold change of \pm 2.0 in the post-night shift samples. The genes with the greatest increase in expression levels were: *PRDX5*, *SLC7A5*, *FCGR1A*, *DNAJC7*, *PSMD4*, and *PER1*. The genes with the greatest decrease in expression levels were: *PPIP5K2*, *SCART1*, *CASP10*, *SLC24A4*, and *OSBP*. Based on the IPA analyses, the differentially expressed genes play a role in gene expression, cell signaling, cell death and survival, and RNA damage and repair.

Conclusion: Significant differential gene expression in pathways associated with poor health were observed among female nurses engaged in rotating shifts. Potential molecular and cellular functions were identified that may be the mechanisms resulting in the increased health risks associated with shift work.

Support: University of Pittsburgh School of Nursing Center for Research and Evaluation Pilot/Feasibility Study Program

0021

RNA SEQUENCING REVEALS TRANSCRIPTOMIC CHANGES IN INDIVIDUALS WITH INSOMNIA

Mithani, S.¹ Yun, S.² Pattinson, C.¹ Kim, H.¹ Guedes, V.¹ Fink, A.³ Weljie, A.⁴ Gehrman, P.⁵ Gill, J.¹

¹National Institutes of Health, Bethesda, MD, ²Yotta Biomed, LLC., Bethesda, MD, ³University of Illinois at Chicago, Chicago, IL, ⁴University of Pennsylvania, Department of Psychiatry, Philadelphia, PA, ⁵University of Pennsylvania, Department of Pharmacology, Philadelphia, PA.

Introduction: Insomnia affects 10–20% of the US population and is associated with negative health and psychosocial sequelae. Despite the public health impact of insomnia little is known about its underlying molecular mechanisms. The purpose of this study

is to examine differentially expressed genes in 15 patients with chronic insomnia and age- and sex-matched good sleepers (n=15). **Methods:** We performed total RNA-seq on 30 whole blood samples collected at 09:00 at 150 bp paired-ends on the Illumina NovaSeq-6000 platform. Alignment was performed using the STAR version 2.7.2a software on the human reference genome (GRCh38). Differential gene expression analysis was performed using DESeq2 version 1.24.0. Pathway analysis was performed using IPA, release 2019-08-30.

Results: An average of 86.7 million paired end reads per sample were sequenced. We found that 289 genes were differentially expressed in insomnia patients with a log fold change (LFC) ± 0.50 and had a FDR p-value < 0.05. Top dysregulated genes include CSMD1 (L2FC=-2.78; p=1.35E-06), DUX4L9 (L2FC=3.40; p=2.81E-06) and GRM4 (L2FC=2.45; p=4.50E-05). Among the functionally relevant genes, CSMD encodes a complement control protein that is known to participate in the complement activation and inflammation in the developing central nervous system. UTS2 (L2FC=1.778; p=8.94E-06) is involved in regulation of orexin A and B activity and rapid eve movement during sleep. Ingenuity Pathway Analysis revealed 3 associated networks: Hematological, Hereditary Disorder, Organismal Injury and Abnormalities (score: 46), Developmental, Hereditary Disorder, Metabolic Disease (score: 43), and Cell Cycle, Cell mediated Immune Response, Cellular Development (score: 43).

Conclusion: Overall, our study revealed dysregulated genes in individuals who suffer from insomnia. Notably, dysregulation of these functionally relevant genes could impair functional brain connectivity and synaptic function. Further investigation of these biological pathways will be useful to elucidate the pathogenesis of insomnia and identify novel biomarkers or drug targets for developing improved diagnostics and therapeutics.

Support: National Institutes of Nursing Research, Graduate Partnership Program

0022

KLOTHO GENETIC VARIANTS MEDIATE THE ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA AND SHORT TELOMERE LENGTH

Tempaku, P. F. D'Almeida, V. Andersen, M. L. Belangero, S. I. Tufik, S.

Universidade Federal de Sao Paulo, São Paulo, BRAZIL.

Introduction: The core features of obstructive sleep apnea (OSA) can potentially contribute to the acceleration of telomere shortening mechanisms. Among these factors, klotho reduction can contribute since it is associated with accelerated systemic inflammation and oxidative stress and has recently been associated with OSA. Also, decreased levels of klotho are implicated in the regulation of telomerase activity. Therefore, we aimed to evaluate the effect of common genetic variants (SNPs) on KLOTHO gene on the association between OSA and short telomere length.

Methods: As part of the Sao Paulo Epidemiologic Sleep Study cohort, 1,042 individuals answered questionnaires, underwent polysomnography and had blood collected for DNA extraction. OSA was defined according to AHI equal or greater than 15 events per hour. Leukocyte telomere length (LTL) was measured through qPCR and SNPs were genotyped by microarray.

Results: LTL was significantly shorter in OSA compared to controls in a severity-dependent manner (B=0.055, CI=0.007–0.102, p=0.02). Among the 43 SNPs analyzed, we observed that 4 SNPs (rs525014, rs7982726, rs685417 and rs9563124) significantly

mediated the association between OSA and short LTL (B=0.046, df=1, p=0.005; B=0.044, df=1, p=0.007; B=0.045, df=1, p=0.006; B=0.044, df=1, p=0.007; respectively). Furthermore, this association was under an additive model since having one or two alleles of the alternative variants were significantly associated with shorter LTL.

Conclusion: We could conclude that klotho opens a new venue in OSA research and would be applicable to prevent the consequences of short telomeres in individuals with OSA.

Support: This work was supported by grants from AFIP, FAPESP and CAPES.

0023

NEUROPEPTIDERGIC REGULATION OF DROSOPHILA LARVAL SLEEP

Poe, A. R. Szuperak, M. Kayser, M. S. University of Pennsylvania, Philadelphia, PA.

Introduction: Sleep during early life is thought to be important for brain development. Indeed, disruptions in sleep during development have long-lasting effects on cognitive functioning. Recently, our lab has developed the LarvaLodge platform for monitoring sleep in developing *Drosophila* larvae. Using this system we can investigate the neural circuits and signals controlling sleep during early neurodevelopmental periods. Neuropeptides play critical roles in regulating many behaviors in both larvae and adult flies. While several neuropeptides modulate sleep in adult flies, it is not known what role neuropeptides play in controlling larval sleep.

Methods: To identify peptidergic neurons that regulate 2nd instar larval sleep, we activated neurons labeled by 34 independent Gal4 driver lines corresponding to 25 different neuropeptide genes using the heat-sensitive cation channel, TrpA1.

Results: Of the 34 Gal4 driver lines, we determined that 2 lines are wake-promoting and 7 lines are sleep-promoting. A subset of these exert effects on sleep without associated changes in wake activity levels. We also observed sleep fragmentation (increase in sleep bout number and decrease in sleep bout length) in 3 lines. Subsequent analysis indicated that manipulation of activity in *Diuretic hormone 44* (Dh44)-labeled neurons bidirectionally modulates sleep-wake. Additionally, pan-neuronal knockdown of Dh44 altered sleep duration.

Conclusion: This work indicates that neuropeptidergic signaling modulates sleep during early development and provides a platform to examine how neuropeptidergic regulation of sleep/wake changes throughout the lifespan.

Support: NIH T32

0024

PTSD WITH CONCURRENT EXCESSIVE DAYTIME SLEEPINESS ALTERS GENE EXPRESSION IN MILITARY PERSONNEL AND VETERANS; AN RNA-SEQUENCING STUDY

Pattinson, C. L.^{1,2} Edwards, K.² Guedes, V. A.² Mithani, S.² Yun, S.² Taylor, P.³ Dunbar, K.³ Lai, C.² Roy, M. J.⁴ Gill, J. M.² ¹The University of Queensland, Institute for Social Science Research, Brisbane, AUSTRALIA, ²National Institutes of Health (NIH), National institutes of Nursing Research, Bethesda, MD, ³Henry M. Jackson Foundation for the Advancement of Military Medicine, Bethesda, Bethesda, MD, ⁴Department of Medicine, Uniformed Services University of the Health Sciences, Bethesda, MD., Bethesda, MD. **Introduction:** Up to 91% of military personnel and veterans with posttraumatic stress disorder (PTSD) report co-occurring sleep disturbances, including. insomnia and excessive daytime sleepiness (EDS). Sleep disturbances have been shown not only to increase the risk of developing PTSD, but to exacerbate and maintain PTSD symptomology. The aim of this study was to examine gene expression in active duty military personnel and veterans with PTSD, with and without EDS. Participants were categorized into three groups; 1) PTSD with EDS (PTSDwEDS; n=21), 2) PTSD without EDS (PTSDnoEDS; n=25), or 3) Controls (no PTSD and no EDS; n=57).

Methods: Participants were 79% male, mean age of 37.6years (SD=11.2years). PTSD symptoms were measured using the PTSD checklist for civilians (PCL-C); participants were classified as PTSD-present using DSM-IV-TR criteria of "moderate-to-severe". Daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS), high sleepiness was indicated by an ESS score >13. We performed RNA-seq with Illumina's HiSeq 2500 in paired-end. We conducted quality control using FastQC and aligned to GRCh38 reference genome using STAR (v2.5.3a). Differentially expressed genes identified using DESeq2 (v1.20.0) with False Discovery Rate of 0.10. Finally, Ingenuity Pathway Analysis (IPA) was conducted to identify dysregulated gene networks.

Results: Between the Controls and PTSDnoEDS groups, two genes were significantly dysregulated. In controls and PTSDwEDS groups, 251 genes were dysregulated. The IPA networks showed that genes associated with inflammation were significantly dysregulated. Finally, between PTSDwEDS and PTSDnoEDS there were 1,873 significantly dysregulated genes. The IPA networks identified dysregulation of genes related to sleep, fatigue, circadian, and mitochondrial function.

Conclusion: Taken together this data indicates that EDS that is co-morbidly experienced with PTSD is associated with significant gene dysregulation, above and beyond that observed in participants with PTSD without significant EDS and controls. Treating EDS in military personnel and veterans with PTSD is important. **Support:** This work was supported by the Center for Neuroscience and Regenerative Medicine (CNRM)

0025

CIRCADIAN DYSREGULATION OF DNA REPAIR AND INCREASED ENDOGENOUS AND EXOGENOUS SENSITIVITY TO DNA DAMAGE PRECIPITATE ELEVATED CANCER RISK ASSOCIATED WITH NIGHT SHIFT WORK

Van Dongen, H.^{1,2} Koritala, B.^{1,3} McDermott, J. E.^{4,5} Porter, K. I.^{1,3} Arshad, O. A.⁴ Gajula, R. P.^{1,3} Mitchell, H. D.⁴ Arman, T.³ Manjanatha, M.⁶ Gaddameedhi, S.^{1,3}

¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ³Department of Pharmaceutical Sciences, College of Pharmacy and Pharmaceutical Sciences, Washington State University, Spokane, WA, ⁴Computational Biology and Bioinformatics, Pacific Northwest National Laboratory, Richland, WA, ⁵Department of Molecular Microbiology and Immunology, Oregon Health and Science University, Portland, OR, ⁶Division of Genetic and Molecular Toxicology, National Center for Toxicology Research, US Food and Drug Administration, Jefferson, AR.

Introduction: The elevated cancer risk associated with night shift work is poorly understood. To investigate whether circadian

disruption may play a role, we assessed the circadian transcriptome and its association with hallmark cancer pathways, as well as sensitivity to endogenous and exogenous sources of DNA damage, after in-laboratory simulated shift work.

Methods: N=14 healthy humans (aged 22-34y; 10m, 4f) were exposed to a simulated night shift schedule (daytime sleep: 10:00-18:00) or a simulated day shift schedule (nighttime sleep: 22:00-06:00) for 3 days (n=7 in each condition). After the simulated shift schedule, subjects underwent a 24h constant routine protocol, during which blood was collected every 3h. Lymphocytes were extracted from the blood samples and subjected to transcriptome analysis using a NanoString multiplex assay. We evaluated 726 mRNA cancer hallmark targets (NanoString PanCancer Pathway Panel) and 17 circadian clock genes, with 18 arrhythmic internal controls. Gene expression was analyzed for circadian rhythmicity using mixed-effects cosinor analysis. Further, lymphocytes were investigated for DNA damage using an alkaline comet assay and immunofluorescence assessment of DNA damage response biomarkers BRCA1 and yH2AX. Lymphocytes collected at 07:30 and 19:30 were also exposed to ionizing radiation (2.5Gy) and DNA damage response assessments were repeated.

Results: Simulated night shift caused widespread disruption of circadian rhythmicity, as measured under constant routine, for core clock genes and the transcriptome of cancer hallmark pathways. The DNA repair pathway showed significant enrichment of rhythmic genes (p<0.05) after the simulated day shift schedule only. Following simulated night shift, lymphocytes showed induction of endogenous DNA damage, with extended tail in the comet assay (p<0.001), and higher percentage of lymphocytes with BRCA1 and γ H2AX foci (p<0.01). Lymphocytes collected at 19:30 showed enhanced impact of ionizing radiation as indicated by increased prevalence of cells with BRCA1 and γ H2AX foci (p<0.05).

Conclusion: Circadian dysregulation of DNA repair mechanisms and increased sensitivity to DNA damage following night shift work may increase genomic instability and precipitate elevated cancer risk in night shift workers.

Support: NIH grants ES022640 and CA227381, CDMRP award W81XWH-18-1-0100, and Pacific Northwest National Laboratory BRAVE investment under DOE contract DE-AC05-76RL01830.

0026

STRONG GENE-ENVIRONMENT INTERACTIONS OF TRANK1 GENE POLYMORPHISMS WITH BIRTH DIFFICULTIES IN KLEINE LEVIN SYNDROME

Ambati, A.¹ Hillary, R.² Semenescu, S. L.³ Lin, L.¹ Ollila, H.¹ Farber, N.⁵ Huang, Y.⁶ Dauvilliers, Y.⁷ KLS Working Group, -.¹ Arnulf, I.³ Mignot, E.¹

¹Stanford University, Palo Alto, CA, ²Stanford University, Palo Alto, CA, ³Sleep Disorders (Department "R ³S"), Pitié-Salpêtrière Hospital, APHP, National Reference Center for Narcolepsy, Idiopathic Hypersomnia and Kleine-Levin Syndrome, Sorbonne Universités, IHU@ICM, INSERM, Paris, FRANCE, ⁴Stanford University, Palo Alto, CA, ⁵Kleine-Levin Syndrome Foundation, Boston, MA, ⁶Department of Child Psychiatry and Sleep Center, Chang Gung Memorial Hospital at Linko, Taoyuan,, Taoyuan, TAIWAN, ⁷Centre de référence nationale narcolepsie et hypersomnie idiopathique, Montpellier, FRANCE.

Introduction: Kleine-Levin Syndrome (KLS) is a rare disorder affecting adolescents and characterized by relapsing-remitting episodes of severe hypersomnia, cognitive impairment, and

SLEEP, Volume 43, Abstract Supplement, 2020

behavioral disturbances such as hyperphagia and sexual disinhibition. Pathophysiology is unknown, although imaging studies indicate decreased activity in hypothalamic/thalamic areas and in cortical areas during episodes. Familial occurrence is increased, and risk is associated with reports of complicated birth.

Methods: A worldwide Genome wide association (GWA) study was conducted in 673 KLS patients and ethnically matched 15,341 control individuals.

Results: We found a strong genome-wide significant association (OR=1.48 at rs150168018, p=8.6x10⁻⁹) with 24 single nucleotide polymorphisms (SNPs) encompassing a 35kb region located in the 5' region of TRANK1 gene previously associated with bipolar disorder and schizophrenia. Strikingly, KLS cases with TRANK1 had statistically increased reports of difficult birth. As perinatal outcomes have dramatically improved over the last 40 years, we further stratified our sample by birth years, and found that recent cases had a significantly reduced TRANK1 association. These findings were confirmed in an independent replication cohort of 171 new patients where polygenic risk scores constructed on the discovery cohort replicated (r2=0.15; $p < 2.7 \times 10^{-22}$ at p=0.1 threshold) and the TRANK1 association was found to be dependent on reports of birth difficulties (OR=1.54, p=0.01 versus OR=1.12, p=0.4). Pathway analysis of the overall GWAS association revealed significant association (p=0.02) with 19 genes in a pathway modulating rhythmic behaviors.

Conclusion: Our results demonstrate links between hypersomnia, behavioral rhythmicity and bipolar disorder and indicate that a polymorphism in the TRANK1 region affect brain development in the presence of a perinatal injury, with pathophysiological consequences such as KLS, bipolar disorder and schizophrenia.

Support: NIH NIMH 1R01MH080957 to EM PHRC 070138 to IA

0027

ALTERNATIONS IN CONTRACTILITY, CALCIUM SIGNALING AND NUCLEAR FACTOR KAPPA B ACTIVATION IN STEM CELLS DERIVED CARDIOMYOCYTES FOLLOWING EXPOSURE TO OBSTRUCTIVE SLEEP APNEA CHILDREN'S SERUM, AND INTERMITTENT HYPOXIA

Goldbart, A.¹ Haddad, H.² Etzion, S.³ Gopas, J.⁴

¹Dept. of Pediatrics,Saban Pediatric Medical Center,Soroka University Medical Center, Beer Sheva, ISRAEL, ²Faculty of Health Sciences, Ben Gurion University, Beer Sheva, ISRAEL, ³Regenerative Medicine & Stem Cell Research Center, Ben Gurion University, Beer Sheva, ISRAEL, ⁴Department of Microbiology, Immunology and Genetics and Oncology Department,Faculty of Health Sciences,Ben Gurion University, Beer Sheva, ISRAEL.

Introduction: There are cardiovascular morbid effects of OSA in adults and children. We have previously reported over-expression of Nuclear-Factor-kappa B in tonsils of children with OSA, and NF- κ B activation after exposure to sera of children with OSA. In order to investigate NF-kB activation and the physiology of cardiomyocytes of human origin (CM's), we have established an ex-vivo model where CM's derived from human embryonic stem cells H-9.1 clone (hES) and from Induced pluripotent stem cells (iPSC) are either incubated with human sera, or exposed to intermittent hypoxia(IH) or room air (RA).

Methods: Serum samples were drawn following overnight polysomnography (PSG) from children with OSA (AHI>5) or control (AHI<1). Differentiated human cardiomyocytes were

incubated with 5% OSA sera or control. Average cell beating/min was determined and NF-kB p50 and p65 cytoplasmic or nuclear localization were detected by immunofluorescence (operetta). CM's were exposed to either RA or to IH (21% alternating with 1% O_2 , 6 cycles/h, 24 h) in a chamber (BioSpherix Instruments, Redfield, NY)Intracellular calcium transient signal (ionoptix) and mechanical contraction of CM (Musclemotion) were assessed.

Results: 1. Cell beating/min was reduced in cells incubated with OSA (n=10) as compared to control (n=10);(p<0.01), and following IH exposure vs RA (p<0.01),). 2. There is an over-expression of NF- κ B p50, p65 subunits as a result of exposing CMs to OSA sera, and following IH exposure 3. a rapid decrease (up to 100%) was measured in calcium transient amplitude as well as contraction amplitude (p<0.05) after adding OSA sera, that was completely restored after washing.

Conclusion: We revealed human cardiomyocytes NF-kB activation and decreased contractility following exposure to OSA sera and to IH. The rapid decrease in calcium signaling and contractility infer the presence of factors that reversibly affects CMs contraction. This ex-vivo model enables the study of functional and molecular alterations in human CM

Support: Israel science Foundation (ISF) 1344/15

0028

SLEEP DURATION INFLUENCES THE KINETICS OF STRESS GRANULE FORMATION

Dougherty, M. K. Saul, C. Carman, L. Nelson, M. D. Tudor, J. C. Saint Joseph's University, Philadelphia, PA.

Introduction: Stress granules are non-membrane bound aggregates of messenger ribonucleoproteins that are biomarkers of cellular stress. It has been shown in cells *in vitro* that suppression of the mammalian target of rapamycin (mTOR) pathway and its non-mammalian orthologue target of rapamycin (TOR) is associated with an increase in stress granule formation. It has also been shown that the mTOR pathway is suppressed in response to sleep deprivation in mice. Despite the possible connection via the TOR/mTOR pathway, there has not been any previous evidence linking sleep deprivation with stress granule formation.

Methods: Our present investigation uses the nematode *Caenorhabditis elegans* to model how stress granule formation and clearance are modified by sleep duration. We developed novel strains of *C. elegans* that model each type of sleep deprivation or enhancement and have RFP-labeled PAB-1 protein, a key component of stress granules. In addition to modifying sleep duration via genetic means, we also sleep deprived wildtype fluorescently labeled animals using mechanical disturbances.

Results: Animals with enhanced stress-induced sleep have stress granules that are smaller in size and cleared faster than wildtype, while sleep deprived animals have granules that are slower to clear ($F_{11,473} = 7.752$, ***p < 0.0001, one-way ANOVA). Animals that were manually deprived of stress-induced sleep were similarly slower to clear stress granules ($F_{5,209} = 5.476$ ***p < 0.0001, one-way ANOVA). Interestingly, animals genetically deprived of developmentally-timed sleep does not appear to have more stress granules in the middle of their sleep period than the sleeping wildtype stage ($F_{2,42} = 2.659$, p = 0.0729, one-way ANOVA).

Conclusion: This work demonstrates that the amount of sleep affects stress granule kinetics, which impacts the flow of genetic information inside cells.

Support: This work was supported by an R15GM122058 (NIH), John P. McNulty scholars program (SJU) and summer scholars program (SJU).

III. Circadian Rhythms Mechanisms and Physiology

0029

LIGHT EMITTED FROM MEDIA DEVICES AT NIGHT IS ASSOCIATED WITH DECLINE IN SPERM QUALITY

Green, A.^{1,2} Barak, S.³ Shine, L.⁴ Kahane, A.⁵ Dagan, Y.⁶ Dagan, Y.^{1,2} ¹The Sleep and Fatigue Institute, Assuta Medical Center, Tel-Aviv, ISRAEL, ²The Research Institute of Applied Chronobiology, The Academic College of Tel-Hai, Tel-Hai, ISRAEL, ³Reproductive Services, Assuta University Hospital, Ashdod, ISRAEL, ⁴The Andrology Laboratory, Assuta Medical Center, Rishon Le-Zion, ISRAEL, ⁵The IFV Unit, Assuta Medical Center, Rishon Le-Zion, ISRAEL, ⁶The Sleep and Fatigue Institute, Assuta Medical Center, Tel Aviv, ISRAEL.

Introduction: The last several decades have been characterized by the widespread usage of digital devices, especially smartphones. At the same time, there have been reports of male fertility decline. The aim of this study was to assess the relationship between evening exposure to the light-emitting screens of digital media devices and sperm quality.

Methods: Semen samples were obtained from 116 men adults aged between 21 and 59 (35.2 ± 7.2) undergoing fertility evaluation for the following sperm variables: volume (mL), pH, sperm concentration (n/mL), motility percentage (progressive% + non-progressive motility%) and total sperm count. Exposure to the screens of electronic devices and sleep habits were obtained by means of a questionnaire.

Results: Smartphone and tablet usage in the evening and after bedtime was negatively correlated (p<0.05) with sperm motility, sperm progressive motility, and sperm concentration, and positively correlated with the percentage of immotile sperm. In addition, sleep duration was positively correlated with sperm total and progressive motility and negatively correlated with semen pH (p<0.05). A significant negative correlation was observed between subjective sleepiness and total and progressive motility as well as total motile sperm number (p<0.05).

Conclusion: The results of this study revealed a link between evening and post-bedtime exposure to light-emitting digital media screens and sperm quality. To the best of our knowledge, this is the first study to report these types of correlations between sperm quality and exposure time to SWL emitted from digital media, especially smartphones and tablets, in the evening and after bedtime. **Support:** No Support

0030

EFFECT OF A SIMULATED SUNSET VERSUS TYPICAL INDOOR LIGHTING ON EVENING MELATONIN LEVELS

Lanza, S. M.¹ Kindel, B. C.² Sprecher, K. E.¹ Trainer, M. M.¹ Wright Jr., K. P.¹

¹Sleep and Chronobiology Laboratory, Department of Integrative Physiology, University of Colorado at Boulder, Boulder, CO, ²Laboratory for Atmospheric and Space Physics, University of Colorado at Boulder, Boulder, CO.

Introduction: The human circadian clock evolved in the presence of the natural light-dark solar cycle. Exposure to artificial light at night suppresses endogenous melatonin levels and delays the timing of the circadian clock. The advent of tunable LED (light emitting diode) technology presents an opportunity to develop and implement circadian based practices for healthy lighting. Here we determined the influence of a simulated sunset with tunable LED technology on evening melatonin levels. Methods: Nine healthy adults (3 females, 24.0 ± 5.3 years) completed a 15-day crossover study comparing typical artificial indoor lighting versus a simulated sunset using tunable LEDs (Acuity Brands-Rubik). After 1 week maintaining an ~8h sleep schedule, participants arrived at the laboratory 6h prior to habitual bedtime. Typical electrical indoor home lighting was <90 lux in angle of gaze until scheduled bedtime (<467 lux maximum at 183 cm in the direction of the ceiling mounted light fixtures; 3,500K). To simulate sunset, a simple least square fit was used to match relative spectral irradiance of the laboratory LED lighting to solar spectral irradiance of a standard mid-latitude summer atmosphere in Boulder, Colorado with solar elevation angles ranging from 3.9 degrees to 0 degrees (sunset). The first 3h30min of the simulation was typical indoor lighting of <90 lux (angle of gaze; 3,500K) followed by a 25 min transition in spectral irradiance and then 2h5min at ~7 lux in the angle of gaze (<38 lux maximum at 183 cm; 2,700K). **Results:** Melatonin levels were initially similar between conditions but were significantly higher (p < 0.05) after the sunset transition in the simulated sunset condition compared to the typical electrical indoor home lighting condition.

Conclusion: These preliminary findings suggest that simulating a sunset transition with tunable LED technology prior to habitual bedtime in the evening has potential to benefit circadian health. **Support:** This work was supported in part by NIH R01 HL135598 and NASA Award 80NSSC17K0569.

0031

PHOTOPERIODIC EFFECTS ON DIURNAL RHYTHMS IN THE IMMUNE SYSTEM, REST-ACTIVITY BEHAVIOR, AND CORTISOL REVEALED IN A DIURNALLY ACTIVE LARGE ANIMAL MODEL - THE DOMESTIC PIG

Engert, L. C. Weiler, U. Pfaffinger, B. Stefanski, V. Schmucker, S. S.

Behavioral Physiology of Livestock, Institute of Animal Science, University of Hohenheim, Stuttgart, GERMANY.

Introduction: Diurnal variations in immune cell number and function are regarded important for immune competence and are thought to be mediated by rest-activity rhythms and hormones. Moreover, the photoperiod is also known to modulate the immune system and considered to affect seasonal disease susceptibility. Whereas few studies investigated seasonal effects, the present study is the first investigating the specific effect of the photoperiod on diurnal rhythms in cell numbers of peripheral leukocyte types in any species.

Methods: Domestic pigs were held either under long day (LD, 16L:8D, lights-on 07:00-23:00, n=9) or short day conditions (SD, 8L:16D, lights-on 07:00-15:00, n=11) and fed concentrate at 07:30 + 14:00 (ad libitum hay/water). Blood samples were taken every 2 h over periods of 50 h via indwelling vein catheters. Restactivity behavior of the pigs was analyzed using continuous camera recordings.

Results: Cosinor analyses (p<.05) revealed photoperiodic differences in diurnal rhythms of cell numbers of various peripheral leukocyte subtypes, rest-activity behavior, and cortisol concentration. Cell numbers of total leukocytes, NK cells, T cells, and eosinophils in blood, rest-activity behavior, and cortisol concentration peaked earlier relative to lights-on under SD (p<.05). Relative amplitudes in rest-activity behavior and cell counts of total leukocytes, NK cells, T cells, and monocytes were higher under SD (p<.05). However, there was no photoperiodic effect on diurnal rhythms in neutrophil counts and mesor in any leukocyte type.

SLEEP, Volume 43, Abstract Supplement, 2020

Generalized linear mixed models revealed associations of leukocyte counts with rest-activity behavior and cortisol concentration in most cell types (p<.05). Moreover, the found photoperiodic effects on diurnal rhythms in rest-activity behavior and cortisol concentration are in agreement with research in humans and primates. **Conclusion:** The present study revealed photoperiodic effects on diurnal rhythms in the immune system, rest-activity behavior, and

cortisol concentration in pigs and strengthens the importance of the domestic pig as suitable model for chronoimmunology. **Support:** German Research Foundation DFG (grant provided to SS, grant number SCHM3162/1-1), Federal Ministry of Education and Research, Germany (grant number 01PL16003), Faculty of Agricultural Sciences, University of Hohenheim (scholarship provided to LE).

0032

TIMING OF DAILY RHYTHM OF CARDIAC AUTONOMIC CONTROL CONTRIBUTES TO WEIGHT LOSS RESISTANCE, INDEPENDENT OF DAILY ENERGY INTAKE AND PHYSICAL ACTIVITY LEVEL

Yang, H.¹ Garaulet, M.² Li, P.¹ Bandin, C.² Lin, C.⁵ Lo, M.⁵ Hu, K.¹ ¹Medical Biodynamcis Program, Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, Boston, MA, ²Department of Physiology, University of Murcia, Murcia, SPAIN, ³Medical Biodynamcis Program, Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, Boston, MA, ⁴Department of Physiology, University of Murcia, Murcia, SPAIN, ⁵Institute of Translational and Interdisciplinary Medicine and Department of Biomedical Sciences and Engineering, National Central University, Taoyuan, TAIWAN.

Introduction: Obesity is a major health problem. Many treatments have been designed to help overweight/obese people to lose weight, but their effectiveness is highly variable. The same treatments may work for some persons while others have no responses — weight loss resistance. We tested whether the daily rhythm of cardiac autonomic control contributes to weight loss resistance.

Methods: We studied 39 overweight/obese Caucasian women (BMI>25; age: 21–62 years old) who completed (1) an obesity dietary treatment of up to 30 weeks with weekly assessments of body weight, and (2) ambulatory monitoring of electrocardiogram (ECG) for up to 3.5 days. Heartbeat intervals were derived from ECG. Cardiac autonomic control was assessed in each 1-h bin by examining the temporal correlation in heartbeat fluctuations — a nonlinear measure that quantifies the delicate dynamic interplay between sympathetic and vagal outflows. Daily rhythm was estimated using the cosinor analysis.

Results: Weight loss was highly variable (range: 0.68%-21.78 % of initial body weight). The correlation in heartbeat fluctuations displayed a 24-h rhythm (p<0.0001) with fewer correlations (more random) during the nighttime. The phase (peak timing) of the rhythm was highly variable, i.e., 10AM to 8PM for most participants, and after midnight in four participants. Weight loss evolution depended on the phase (p=0.006) in a nonlinear manner. Specifically, participants with the phase between 2PM-8PM lost weight faster than those with phases before 2PM and those after 8PM. The effect was independent of total energy intake, physical activity level, and sleep/wake schedules.

Conclusion: Cardiac autonomic control in overweight/obese women displayed a daily rhythm. The timing of the rhythm had previously un-identified contributions to weight loss. The inter-individual differences in the timing may reflect different circadian regulation of autonomic function and its interaction with the daily behavioral cycle. **Support:** This work was supported by NIH grants R01AG048108, RF1AG059867, RF1AG064312, R01AG017917, and R01NS078009.

0033

RECURRENT CIRCADIAN DISRUPTION WHILE MINIMIZING SLEEP LOSS IN HUMANS IMPAIRS GLUCOSE TOLERANCE ONLY IN THE PRESENCE OF HIGH-FAT DIET

Zitting, K.^{1,2} Yuan, R. K.^{1,2} Vujovic, N.^{1,2} Klerman, E. B.^{1,2,3} Quan, S. F.^{1,2,4} Scheer, F. A.^{1,2} Wang, W.^{1,2} Buxton, O. M.^{1,2,5} Williams, J. S.^{1,2} Duffy, J. F.^{1,2} Czeisler, C. A.^{1,2}

¹Brigham and Women's Hospital, Boston, MA, ²Harvard Medical School, Boston, MA, ³Massachusetts General Hospital, Boston, MA, ⁴University of Arizona College of Medicine, Tucson, AZ, ⁵Pennsylvania State University, University Park, PA.

Introduction: Nearly 14% of Americans experience chronic circadian disruption due to shift work, increasing their risk of obesity and cardiometabolic disorders. These disorders are also exacerbated by modern eating habits such as frequent snacking and consumption of high-fat foods. Here we used a forced desynchrony protocol to investigate the effect of 3 weeks of recurrent circadian disruption (RCD) with minimal sleep loss on glucose metabolism in humans on a lower or higher fat diet (LFD and HFD, respectively). Methods: Six healthy adults (38-69yrs; 3f) participated in a 37-day inpatient protocol with LFD (25-27% fat) and 15.67-hr fasting duration, or HFD (45-50% fat) and 13-hr fasting duration. The protocol included three weeks of RCD consisting of 28-hr "days" with 11.67-hr sleep opportunities (=10hrs/24hr). Glucose and insulin responses to a standardized breakfast were conducted at baseline, at an aligned circadian phase after 2-3 weeks of exposure to RCD, and after 1 week of recovery. Frequent blood samples were assayed for glucose and insulin; the Area-Under-Curve was calculated from start of breakfast through postprandial minute 180.

Results: Total Sleep Time was similar in Baseline and RCD in both groups. Participants on the LFD showed no change in glucose AUC during RCD compared to Baseline. Insulin AUC was lower during RCD (p=0.0269) and Recovery (p=0.0443) than Baseline. In contrast, participants on the HFD showed a significant increase in glucose AUC during RCD compared to Baseline (p<0.0001); AUC returned to Baseline during Recovery. There was no significant change in insulin AUC on the HFD.

Conclusion: RCD (in the absence of sleep loss) led to impaired glucose tolerance when combined with HFD, but not when combined with LFD. These results suggest that LFD may be part of healthy strategies for people experiencing RCD.

Support: Study supported by P01AG009975 and conducted in the Brigham and Women's Hospital Center for Clinical Investigation, part of Harvard Clinical and Translational Science Center supported by UL1TR001102. Authors supported by a fellowship from the Finnish Cultural Foundation (KMZ); T32HL007901 and F32HL143893 (RKY); T32HL007901 and F32AG051325 (NV); K24HL105664 (EBK); R01HL118601 (FAJLS).

WHEN SHOULD YOU SLEEP TO MAXIMIZE ALERTNESS?

Vital-Lopez, F.^{1,2} Doty, T. J.³ Balkin, T. J.³ Reifman, J.¹ ¹DoD Biotechnology High Performance Computing Software Applications Institute, Fort Detrick, MD, ²Henry M. Jackson Foundation for the Advancement of Military Medicine, Inc., Bethesda, MD, ³Behavioral Biology Branch, Walter Reed Army Institute of Research, Silver Spring, MD.

Introduction: Working under sleep-restricted conditions may curtail safety and productivity. We could potentially minimize the negative effects of sleep restriction by optimizing the timing of sleep. However, to date, there are no algorithms that can determine the optimal sleep time to maximize alertness when most needed.

Methods: Our previously validated unified model of performance predicts the recuperative effects of sleep on alertness. Here, we extended this model to predict the likelihood of an individual falling and remaining asleep at any given moment, as a function of recent sleep history and time of day. Then, we combined the model with an optimization algorithm to provide optimal sleep recommendations for a given work/rest schedule. Specifically, using the model to predict the effectiveness of different sleep schedules, the algorithm determines when to sleep and for how long, so as to maximize alertness at desired times. The algorithm takes as inputs the 1) user-provided sleep history, 2) periods when the user has an opportunity to sleep, and 3) desired periods for maximum alertness, and provides as outputs sleep recommendations that are physiologically feasible and optimize alertness for the desired period. We assessed the algorithm by computing and comparing sleep recommendations for five previously published experimental studies of sleep restriction, including diurnal and nocturnal sleep.

Results: Compared to the original sleep schedules in the studies, our algorithm identified sleep recommendations that increased the predicted alertness by up to 33% and by 18% on average. These results suggest that the algorithm can tailor the timing of sleep to each specific sleep-restriction condition so as to maximize its benefits.

Conclusion: Our algorithm provides automated, customized guidance to enhance the recuperative benefits of limited sleep opportunities to maximize alertness at the most needed times. As such, it is the first quantitative sleep optimization tool for fatiguemanagement systems.

Support: This work was sponsored by the Military Operational Medicine Research Area Directorate of the U.S. Army Medical Research and Development Command, Ft. Detrick, MD.

0035

RESTING METABOLISM AND THE METABOLIC RESPONSE TO EXERCISE FOLLOW CIRCADIAN PATTERNS WITH DAY/NIGHT DIFFERENCES IN SUBSTRATE UTILIZATION BETWEEN LEAN AND OBESE ADULTS

McHill, A. W.¹ Thosar, S. S.¹ Bowles, N. P.¹ Emens, J. S.¹ Purnell, J. Q.³ Gillingham, M.⁴ Shea, S. A.¹

¹Oregon Health and Science University, Portland, OR, ²Oregon Health and Science University, Portland, OR, ³Knight Cardiovascular Institute, Oregon Health and Science University, Portland, OR, ⁴Department of Molecular and Medical Genetics, Oregon Health and Science University, Portland, OR.

Introduction: Resting energy expenditure (EE) follows a circadian rhythm in healthy lean participants, with a nadir in the early morning hours. We determined: (1) whether this pattern persists (or how substrate utilization may change), when challenged with exercise, and; (2) whether obesity affects these responses.

Methods: Fourteen participants (aged $48.5\pm12.8y$; 6-female; 5-obese, BMI $31.9\pm1.4kg/m^2$ [avg±SD]) underwent a 5-day inpatient forced desynchrony protocol, comprised of ten 5h 20min 'days' in dim-lighting and free of time cues. Resting EE was measured immediately prior to a 15-minute cycle ergometer exercise bout at 50% of estimated heart rate maximum. Substrate utilization was determined from the respiratory quotient (RQ). Circadian phase was calculated using the salivary dim-light melatonin onset (>3pg/mL threshold). EE data were analyzed using a mixed-effect model with group (lean vs. obese) and circadian phase as fixed factors; subject was a random factor. RQ was analyzed using t-tests to determine day/night differences in groups at rest and in response to exercise.

Results: Resting and exercising EE both displayed endogenous circadian rhythms (p<0.05) with nadirs in the early morning (~5:30am), without any differences between groups (p>0.22). Resting RQ was similar between the day and night in the lean group (p=0.66), but decreased (suggesting lower carbohydrate utilization) at night within the obese group (-2.5 \pm 1.6%, p=0.02). The lean group increased RQ in response to exercise both during the day (+8.9 \pm 2.8%) and night (+8.0 \pm 2.8%) (both p<0.001), but there was no increase in RQ in the obese group during either day or night exercise (p>0.16).

Conclusion: These data demonstrate that EE during rest and exercise follows a circadian pattern, with limited influence of obesity. Circadian differences in substrate utilization between lean and obese in the resting state and in response to exercise may play a role in expression and maintenance of unwanted weight gain and impaired metabolic health.

Support: R01HL125893, R01HL140577, KL2TR002370, K01HL146992, F32HL131308, Medical Research Foundation of Oregon, Ford Foundation, and CTSA grant (UL1TR000128)

0036

INCONSISTENT BEDTIMES ASSOCIATED WITH UNHEALTHIER BODY SIZE IN OLDER ADULTS

Zendels, P. Gaultney, J.

Psychological Sciences department, University of North Carolina Charlotte, NC.

Introduction: A variety of sleep related variables have been shown to impact measures of health, including duration of sleep, consistency of sleep, quality of sleep, and sleep disorders. These can impact respiratory health, metabolic health, immune function, and more. Older adults have been shown to have different sleep schedules, with mostly consistent weeknight and weekend bedtimes and more opportunities to nap. The study investigated which aspects of sleep best predicted body size (averaged standardized body mass index and percent body fat indices).

Methods: A sample of 304 older adults (55+) participated in surveys and health measures in an urban area in the southeastern United States. Survey data collected were reports of sleep during the last month, including duration at night, nap duration, measures of quality, typical weeknight and weekend bedtime, and reports of possible sleep disorders. Physiological measures, including height, weight, BMI, body fat percentage, blood sugars, blood fats, and fitness tests were conducted. Sleep data were weighted ((5*weeknight+2*weekend)/7) across the week. A hierarchical multiple regression model was run with

SLEEP, Volume 43, Abstract Supplement, 2020

a standardized average of BMI and body fat percentage with multiple sleep variables as a predictor, controlling for age and socioeconomic status.

Results: After controlling for age and socioeconomic status (SES), symptoms of obstructive sleep apnea, night sleep duration, nap duration, difficulty initiating and maintaining sleep, sleep midpoint, duration inconsistencies and midpoint inconsistencies were added to the regression. SES and duration inconsistencies were significant predictors, explaining 16% of the variance in body size.

Conclusion: Inconsistent bedtimes may be associated with larger body size. Encouraging older adults to have consistent sleep schedules could help preserve health as they age. This may also reduce rates of disorders like obstructive sleep apnea, which are associated with higher BMI, and help promote better overall sleep quality and health. However, these older adults reported fairly consistent sleep midpoint, limiting interpretation of this variable.

Support: Psychological Sciences department funding

0037

MELANOPSIN DRIVEN PUPIL RESPONSES AND PHYSICAL ACTIVITY: STABILITY OF ACTIVITY FROM DAY-TO-DAY IN WINTER IN SEASONAL AFFECTIVE DISORDER

*Roecklein, K. A.*¹ *Wescott, D. L.*¹ *Smagula, S. F.*² *Soehner, A. M.*² *Franzen, P. L.*² *Hasler, B. P.*²

¹University of Pittsburgh Department of Psychology, Pittsburgh, PA, ²University of Pittsburgh Department of Psychiatry, Pittsburgh, PA.

Introduction: The post-illumination pupil response (PIPR) is a measure of the responsivity of intrinsically photosensitive retinal ganglion cells (ipRGCs), and reflects the cell biology of the photoentrainment pathway projecting from the retina to the circadian clock. Adequate signaling from the ipRGCs in the retina to the circadian clock is necessary to result in robust circadian output which we hypothesize would increase inter-daily stability (IS), a non-parametric modeling technique that examines stability of rest activity rhythms across successive days.

Methods: Participants were aged 18–66 years and recruited from the greater Pittsburgh area during the Winter with Seasonal Affective Disorder who completed both actigraphy and pupillometry (n = 16). PIPR measures were collected after a 1 second red or blue light pulse, and are calculated as the Net difference between red and blue at multiple time frames: at 6 seconds post stimulus (PIPR 6), from 10–30 seconds post-stimulus (PIPR 20), or from 10–40 seconds post-stimulus (PIPR 30). Using actigraphy, inter-daily stability (IS) was calculated as the amount of overall variability in the recording that is accounted for by the typical 24-hour profile, and reflects stability of the mean 24-h profile day-to-day.

Results: Inter-daily stability (IS) was associated with Net PIPR 20 (B = 0.561; p = .031) and Net PIPR 30 (B = 0.551; p = .034; all *B*'s are standardized), but not Net PIPR 6 (B = 0.298; p = .304). Retinal irradiance was calculated for each participant based on age and pupil diameter, to account for age-related differences in transmission of the stimulus to the retina. All raw Net PIPR values were adjusted for calculated retinal irradiance, and gender and time since wake were included as covariates.

Conclusion: Inter-daily stability (IS) values indicate greater stability of 24-hour activity profiles across days. If reduced responsivity to entraining pulses of light is associated with day-to-day instability in activity rhythms, as shown here, we might expect that amplifying entraining light through environmental changes or bright light therapy would normalize inter-daily stability in SAD, or the reverse, stabilizing activity profiles across days could improve depression and/or normalize retinal ipRGC responsivity. **Support:** NIMH K.A.R. MH103303

0038

THE EFFECTS OF MORNING BLUE LIGHT THERAPY ON INSOMNIA SEVERITY AND PTSD SYMPTOMS IN A CLINICAL SAMPLE

Jecmen, D. King, R. Gould, J. Mitchell, J. Ralston, K. Burns, A. I. Bullock, A. Grandner, M. A. Alkozei, A. Killgore, W. D. University of Arizona, Tucson, AZ.

Introduction: Individuals with Post Traumatic Stress Disorder (PTSD) often present with insomnia, which may exacerbate other symptoms of the disorder. Morning Blue Light Therapy (BLT) can regulate circadian rhythms and may even improve sleep and mood in individuals with major depressive disorder. However, it is unclear whether morning BLT could also be an effective treatment for the insomnia associated with PTSD. We investigated whether 6 weeks of daily morning BLT would improve insomnia severity and symptom presentation in individuals with PTSD in comparison to a placebo condition of amber light (ALT). We hypothesized that changes in insomnia severity would correlate with improvement in PSTD symptom severity.

Methods: Forty-one participants with a clinical diagnosis of PTSD were randomized to receive 6 weeks of either daily morning BLT (n=22) or ALT (n=19). Insomnia and PTSD symptom severity were evaluated at pre- and post-treatment using the Insomnia Severity Index (ISI) and the Clinician-Administered PTSD Scale (CAPS) for DSM-5, respectively.

Results: Both groups showed a significant decrease in their PTSD symptom severity (p<0.001) and insomnia severity (p<0.001) over the 6-week treatment period. However, improvement in insomnia severity significantly predicted improvements in PTSD symptom severity for the BLT group only (BLT: r =0.542, p=0.009; ALT: r=-0.095, p=0.699). The difference between the two correlation coefficients was significant (Z=-2.07, p=0.039).

Conclusion: The results suggest that morning BLT may be effective in improving PTSD symptoms by regulating the circadian rhythm and improving sleep. While ALT also led to improved PTSD symptom severity, it appears that those changes cannot be explained by improved sleep and may have other underlying mechanisms (e.g., placebo effect). Morning BLT may be a promising adjunctive method to bolster current treatment approaches for PTSD. Because of its ease of administration, it could be easily added to ongoing treatment as usual. This approach warrants further research.

Support: US Army Medical Research and Materiel Command: W81XWH-14-1-0570

THE CLINICAL UTILITY OF DIM LIGHT MELATONIN ONSET IN TREATMENT OF DELAYED SLEEP-WAKE PHASE DISORDER: PRELIMINARY FINDINGS

Swanson, L. Arnedt, J. DuBuc, K. de Sibour, T. Burgess, H. University of Michigan, Ann Arbor, MI.

Introduction: Delayed sleep-wake phase disorder (DSWPD) is common, debilitating, and challenging to treat. In an ongoing randomized trial, we are comparing exogenous melatonin treatment outcomes in DSWPD participants for whom dim light melatonin onset (DLMO) is measured objectively vs. estimated.

Methods: Thus far, 13 participants (27±6 years old, 67% female) have completed a randomized, controlled, double-blind 4-week trial of 0.5 mg of exogenous melatonin timed to either 3 h before measured DLMO (M-DLMO, n = 6) or 3 h before DLMO estimated at 2 h before average sleep onset time based on at least 7 days of wrist actigraphy and sleep diary (E-DLMO, n = 7). All participants met International Classification of Sleep Disorders-3 diagnostic criteria for DSWPD and were otherwise healthy. Participants completed 4 weekly treatment sessions with a blinded psychologist; time of melatonin administration and bed-rise schedule were advanced up to 1 h/week. A validated home saliva collection kit measured DLMO in all participants. Between-group t-tests and Hedges' g effect sizes (ES) were calculated at post-treatment for the following outcomes: DLMO; Pittsburgh Sleep Quality Index (PSQI) global score; Morningness-Eveningness Questionnaire (MEQ); and the actigraphy parameters sleep efficiency (SE) and clock time of sleep onset and offset. A paired-sample t-test compared the measured vs. estimated DLMO at baseline.

Results: The M-DLMO group had a 65 ± 88 mins DLMO advance vs. 27 ± 30 mins in the E-DLMO group (ES=0.51 p=.381). PSQI scores were similar between groups (M-DLMO=6.67±2.06, E-DLMO=7.1± 1.57, ES=-0.24, p=.646), as were MEQ scores (M-DLMO=43±4.98, E-DLMO=48±12.72, ES=-0.47, p=.387). Sleep onset time (M-DLMO=0:32±1:02, E-DLMO=0:31±1:38, ES=0.01, p=.98) and offset time (M-DLMO=8:05±1:03, E-DLMO=8:08±2:14, ES=-0.02, p=.968) were similar between the groups, although sleep was more efficient in M-DLMO vs. E-DLMO (84%±3% vs. 76%±10%, ES=0.94, p=.096). On average, baseline measured DLMO occurred 123±83 mins earlier than estimated DLMO (p=.001).

Conclusion: We are continuing to enroll participants in this trial. Preliminary results suggest some potential benefit of measuring the DLMO, but results will need to be clarified in a larger sample.

Support: American Sleep Medicine Foundation Strategic Research Award

0040

RELATIONSHIP BETWEEN INFLAMMATORY MARKERS AND SLEEP IN HEALTHY ADOLESCENTS

Reddy, A.¹ Li, L.¹

¹Univ of Alabama at Birmingham, Birmingham, AL, ²Univ of Alabama at Birmingham, Birmingham, AL.

Introduction: Multiple studies in different countries show a trend of adolescents having insufficient sleep. Review of literature strongly suggests role of cytokines in sleep regulation. Different inflammatory markers like tumor necrosis factor (TNF) α , C-reactive protein (CRP) and interleukins (IL) are sleep regulatory substances. Most of the studies showing relation between cytokines and sleep are seen in adults. In our study, we were interested in finding the relationship between sleep quality and inflammatory markers in healthy adolescents.

Methods: Twenty eight female and male, African American and White, healthy adolescents aged 15–18 completed the study. Sleep quality was measured using the Pediatric Sleep Questionnaires (PSQ), including snoring, daytime sleepiness and hyperactive behavior. Blood sample was collected from each participant for measuring the inflammatory factors.

Results: Partial Pearson correlation analysis showed that global PSQ score and hyperactive behavior were significantly correlated with TNF α (r=0.37 for both). Snoring was significantly correlated with leptin, CRP and IL-6 in healthy adolescents. No other correlations were observed.

Conclusion: Consistent with findings in adults, we have observed an association between inflammatory markers and poor sleep in healthy adolescents. Our findings suggest the importance to improve sleep quality in adolescents for better health outcomes.

Support: None of the authors have any conflict of interest. This research was supported by awards, P30DK056336 and P30DK079626, from the National Institute of Diabetes And Digestive And Kidney Diseases to Nutrition Obesity Research Center and Diabetes Research Center, respectively, at the University of Alabama at Birmingham.

0041

HEART RATE VARIABILITY DIFFERS IN RESILIENT VS. VULNERABLE ADULTS FROM TOTAL SLEEP DEPRIVATION AND PSYCHOLOGICAL STRESS AND PREDICTS COGNITIVE PERFORMANCE

Yamazaki, E. M.¹ Rosendahl-Garcia, K. M.² MacMullen, L. E.³ Ecker, A. J.³ Kirkpatrick, J. N.⁴ Goel, N.¹

¹Biological Rhythms Research Laboratory, Department of Psychiatry and Behavioral Sciences, Rush University Medical Center, Chicago, IL, ²Wyle Science, Technology and Engineering, Houston, TX, ³Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, ⁴Division of Cardiology, Department of Medicine, University of Washington, Seattle, WA.

Introduction: There are substantial individual differences (resilience and vulnerability) in neurobehavioral performance from psychosocial stress and sleep loss. However, the time course of heart rate variability (HRV) across baseline, total sleep deprivation (TSD), the combination of TSD + psychological stress, and recovery has not been investigated; in addition, it remains unknown whether HRV and blood pressure (BP) differ in resilient vs. vulnerable individuals and predict individual differences in cognitive performance.

Methods: Thirty-one healthy adults (ages 27–53; mean \pm SD, 35.4 \pm 7.1y; 14 females) participated in a five-day experiment consisting of two 8h time-in-bed (TIB) baseline nights, 39h TSD, and two 8h-10h TIB recovery nights. A modified Trier Social Stress Test (TSST) induced psychological stress on the TSD day. Systolic and diastolic BP and HRV (derived from echocardiographic R-R interval) were obtained at six time points (pre-study, baseline, during TSD, during TSD after the TSST, after recovery, and post-study). Cognitively resilient (n=15) and vulnerable (n=16) groups were defined by a median split on 10-minute Psychomotor Vigilance Test (PVT) TSD performance [total lapses (>500ms response time) and errors]. Repeated measures ANOVA and post-hoc comparisons corrected for multiple testing, examined BP and HRV across time points between groups.

Results: HRV showed a significant time*group interaction: while resilient individuals had significantly lower HRV at pre-study compared to vulnerable individuals, their HRV increased above that of vulnerable individuals with TSD and with TSD + psychological stress. By contrast, systolic and diastolic BP did not show significant time*group interactions and did not predict cognitive vulnerability during TSD.

Conclusion: HRV differed between resilient and vulnerable individuals across TSD, psychological stress and recovery sleep and predicted individual differences in cognitive performance, whereby lower HRV during full-rested conditions predicted resilience to TSD and TSD + psychological stress. HRV, but not BP, is a reliable biomarker of sleep deprivation, psychological stress, and neurobehavioral vulnerability.

Support: NASA NNX14AN49G.

0042

STROKE VOLUME AND CARDIAC INDEX ARE DIFFERENTIALLY ALTERED BY TOTAL SLEEP DEPRIVATION AND PSYCHOLOGICAL STRESS IN RESILIENT VS. VULNERABLE INDIVIDUALS AND PREDICT COGNITIVE PERFORMANCE

Yamazaki, E. M.¹ Rosendahl-Garcia, K. M.² MacMullen, L. E.³ Ecker, A. J.³ Kirkpatrick, J. N.⁴ Goel, N.¹

¹Biological Rhythms Research Laboratory, Department of Psychiatry and Behavioral Sciences, Rush University Medical Center, Chicago, IL, ²Wyle Science, Technology and Engineering, Houston, TX, ³Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, ⁴Division of Cardiology, Department of Medicine, University of Washington, Seattle, WA.

Introduction: Individuals show robust resilience and vulnerability in neurobehavioral performance to sleep loss and stress. For the first time, we investigated the time course of two cardiovascular measurements, stroke volume (SV) and cardiac index (CI), both derived from echocardiography, across baseline, total sleep deprivation (TSD), the combination of TSD+psychological stress, and recovery. We also determined whether these variables differ in resilient vs. vulnerable individuals and whether they predict differential vulnerability in cognitive performance.

Methods: Thirty-one healthy adults (ages 27–53; mean \pm SD, 35.4 \pm 7.1y; 14 females) participated in a five-day experiment consisting of two 8h time-in-bed (TIB) baseline nights, 39h TSD, and two 8h-10h TIB recovery nights. A modified Trier Social Stress Test (TSST) was conducted on the TSD day to induce psychological stress. Echocardiographic measures of SV and CI were obtained at six time points (pre-study, baseline, during TSD, during TSD after the TSST, after recovery, and post-study). A median split of TSD performance [total lapses (>500 ms response time) and errors] on the 10-minute Psychomotor Vigilance Test (PVT), defined cognitively resilient (n=15) and vulnerable (n=16) groups. Repeated measures ANOVA and post-hoc comparisons corrected for multiple testing, examined SV and CI across time points between groups.

Results: There was a significant time*group interaction for SV: cognitively resilient individuals had greater SV during the fiveday experiment. In addition, in both resilient and vulnerable individuals, SV increased with TSD and with TSD+psychological stress compared with baseline. Like SV, there was a significant time*group interaction for CI: resilient individuals had greater CI at all points of the experiment.

Conclusion: SV and CI differed between resilient and vulnerable individuals across TSD, psychological stress and recovery sleep. Greater SV and greater CI at baseline predicted resilience to TSD and TSD+psychological stress. CI and SV are novel physiological biomarkers of sleep loss, stress, and individual differences in cognitive performance.

Support: NASA NNX14AN49G.

0043

BDNF GENOTYPE MODULATES CIRCADIAN RESPONSE TO SIMULATED NIGHTWORK IN IL-6

Satterfield, B. C.^{1,2} Savenkova, M. I.³ Karatsoreos, I. N.^{1,3} Van Dongen, H.^{1,2}

¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ³Department of Integrated Physiology and Neuroscience, Washington State University, Pullman, WA.

Introduction: Brain Derived Neurotrophic Factor (BDNF) is involved in synaptic plasticity and may be involved in sleep and circadian regulation. The BDNF gene has a single nucleotide polymorphism (Val66Met) resulting in decreased BDNF secretion. The Met allele is associated with reduced nocturnal slow wave activity following sleep loss, and may also enable more flexible adaptation to circadian misalignment. We used IL-6, a pleiotropic cytokine with biphasic circadian secretion, to compare the response of Val and Met carriers to circadian misalignment.

Methods: 15 healthy men (26.7±4.9y) participated in a 16-day laboratory study of simulated nightwork with two nighttime duty cycles and an intervening 58h restart break. The study began with two baseline 10h time in bed (TIB) nighttime sleep opportunities (22:00-08:00) and a 5h TIB afternoon nap (15:00-20:00). Each duty cycle consisted of five nighttime waking periods and four 10h TIB daytime sleeps (10:00-20:00). The restart break began with a 5h TIB morning nap (10:00-15:00), followed by two 10h TIB nighttime sleeps (22:00-08:00). The middle two daytime sleep periods from each duty cycle were monitored polysomnographically and visually scored. During the baseline and restart periods, blood samples were collected at 08:25 and then at 2h intervals until 20:00. Plasma IL-6 was measured by means of ELISA. BDNF Val66Met genotype was determined from whole blood.

Results: The genotype distribution was in Hardy-Weinberg equilibrium (Val/Val:5; Val/Met:10; Met/Met:0). On average, Met carriers slept 52.2min longer (p=0.052) and displayed 27.7min more REM (p=0.013) than Val/Val subjects during the 10h TIB day-time sleeps. Baseline IL-6 concentrations were similar between genotypes. However, during the restart break, Val/Met subjects showed reduced IL-6 compared to baseline and to Val/Val subjects (p=0.022).

Conclusion: BDNF Val66Met heterozygotes exhibited blunted IL-6 levels following simulated nightwork, suggesting this genotype experienced a substantial circadian shift and/or altered response to sleep loss associated with daytime sleep.

Support: FMCSA award DTMC75-07-D-00006; Elliot D. Weitzman, M.D. Research Grant from the Sleep Research Society Foundation.

ASSOCIATION BETWEEN CHRONOTYPE AND CIRCULATING LEVELS OF INTERLEUKIN-6 IN COLORECTAL CANCER PATIENTS: PRELIMINARY RESULTS FROM THE COLOCARE STUDY

Peoples, A. R.^{1,2} Gigic, B.³ Ose, J.^{1,2} Himbert, C.^{1,2} Hardikar, S.^{1,2} Boehm, J.^{1,2} Schrotz-King, P.⁴ Ulrich, A. B.³ Schneider, M.³ Li, C. I.⁵ Shibata, D.⁶ Siegel, E. M.⁷ Figueiredo, J. C.⁸ Toriola, A. T.⁹ Ulrich, C. M.^{1,2}

¹Huntsman Cancer Institute, Salt Lake City, UT, ²Department of Population Health Sciences, University of Utah, Salt Lake City, UT, ³Department of General, Visceral and Transplantation Surgery, University Hospital of Heidelberg, Heidelberg, GERMANY, ⁴Division of Preventive Oncology, National Center for Tumor Diseases (NCT), German Cancer Research Center (DKFZ), Heidelberg, GERMANY, ⁵Division of Public Health Sciences, Fred Hutchinson Cancer Research Center, Seattle, WA, ⁶Department of Surgery, University of Tennessee Health Science Center, Memphis, TN, ⁷Cancer Epidemiology Program, Division of Population Sciences, H. Lee Moffitt Cancer Center & Research Institute, Tampa, FL, 8Department of Medicine, Samuel Oschin Comprehensive Cancer Institute, Cedars-Sinai Medical Center, Los Angeles, CA, ⁹Department of Surgery, Division of Public Health Sciences, Washington University School of Medicine in St. Louis, St. Louis, MO.

Introduction: Accumulating evidence suggests that chronotype, i.e., circadian topology of an individual indicating morning or evening type, is associated with inflammation. To date, no study has examined the relationship between chronotype and inflammation in colorectal cancer patients. We investigated the associations between chronotype and inflammatory and angiogenesis biomarkers in colorectal cancer patients.

Methods: We used pre-surgery serum samples from n=67 newly diagnosed colorectal cancer patients (stage I-IV) recruited at the ColoCare Study site in Heidelberg, Germany. The ColoCare Study is an ongoing, international, multisite, prospective cohort study in colorectal cancer patients. Inflammatory and angiogenesis biomarkers [c-reactive protein (CRP), interleukin (IL)-6, IL-8, monocyte chemoattractant protein-1 (MCP-1), soluble intercellular adhesion molecule-1 (sICAM-1), soluble vascular cell adhesion molecule-1 (sVCAM-1)] were measured at the Huntsman Cancer Institute, USA using the Meso Scale Discovery platform and were log transformed. Chronotype was assessed prior to surgery with the reduced Morningness-Eveningness Questionnaire (rMEQ; scale 4-25; a higher score indicates more morning-type). Patients were dichotomized, based on the median values for rMEQ, into 2 groups: rMEQ-low (score≤16.0; n=35; indicating more evening-type) or rMEQ-high (score>16.0; n=32; indicating more morning-type).

Results: Using Mann-Whitney U test, we observed that rMEQlow group (i.e., more evening-type) compared to rMEQ-high group (i.e., more morning-type) had approx. two times significantly higher levels of log transformed IL-6 (mean=2.24 vs. 1.30; U=382.0; Z=-2.23; p=0.03), but not for other inflammatory or angiogenesis biomarkers. This association between chronotype and IL-6 was maintained even after adjusting for age, sex, tumor stage, tumor site, and sleep duration using a generalized estimating equations model (adjusted mean difference=1.10; 95% confidence interval=0.33, 1.88; p=0.01; effect size, Cohen's d=0.69). **Conclusion:** These preliminary findings suggest that the evening chronotype is associated with increased IL-6 inflammatory biomarker in colorectal cancer patients. Further research is needed to confirm and understand the mechanistic underpinnings of the observed results.

Support: Funding: NCI U01 CA206110, R01 CA189184, and R01 CA207371.

0045

BIOBEHAVIORAL MARKERS FOR SLEEP/WAKE DISTURBANCE AND FATIGUE IN YOUNG CHILDHOOD BRAIN TUMOR SURVIVORS

Johnson, A. H.¹ Bashore, L.^{1,2} Hines, A.² Aufricht, J.¹ Smith, A. M.³ Pearson, H.²

¹Texas Christian University, Fort Worth, TX, ²Cook Children's Medical Center, Fort Worth, TX, ³Sam Houston State University, The Woodlands, TX.

Introduction: Survivors of childhood and adolescent brain tumors and subsequent treatment may experience many neurological processes involving the forebrain, brainstem, and hypothalamus as well as the symptom cluster of stress, sleep, and fatigue. As a result, the impact of brain tumor treatment (chemotherapy/biotherapy, radiotherapy, and surgery) may have lasting biobehavioral effects. Description of symptoms during early survivorship is not always evident in the literature.

Methods: Convenience sampling and the following inclusion criteria were utilized: brain tumor survivors ages 8–17 years; ≥ 6 months, <6 years from completion of treatment; disease free or stable disease. Participants completed polysomnography (PSG) followed by a multiple sleep latency test (MSLT), and subjective measures of sleep, fatigue, stress, and pubertal status. Collection of salivary biomarkers for stress (cortisol) and sleep (melatonin) was completed the evening of and morning after the PSG.

Results: Analysis of the first 12 participants (5 males; 3 Hispanic/ Latino; average age 14 years; 9–72 months post treatment) revealed mean (minutes) total sleep time (TST) 442, sleep latency (SL) 42 and waking (WASO) 88; sleep efficiency (SE) mean 83%, There were large magnitude correlations between several variables of interest, notably PM Cortisol with fatigue, TST (r=.472; -.453); AM Cortisol with SL (r=.479); AM Melatonin with SE, SL, WASO (r=-.459; .692; .458). Average AM melatonin level (26.6 pg/ dl) was higher than PM (6.66 pg/dl). Seven participants were diagnosed with clinical sleep disorders, including one with narcolepsy and two with hypersomnia.

Conclusion: During early survivorship after pediatric brain tumor treatment, survivors may be at high risk for sleep/wake disturbance (SWD). Morning melatonin and biomarker correlations with sleep and fatigue in this sample warrant further exploration and may be related to first night effect versus circadian rhythm differences or clinical sleep disorder. Recommendations for future practice include developmentally matched protocols and routine screening of biobehavioral markers to assess risk for stress, SWD, and fatigue.

Support: 1. Center for Oncology Education and Research Harris College of Nursing & Health Sciences Texas Christian University 2. Neuro-Oncology Program Hematology/Oncology Center Cook Children's Health Care System 3. Nursing Research and Evidence-Based Practice James A. "Buddy" Davidson Endowed Fund

DIFFUSION TENSOR IMAGING EVIDENCE OF HYPOTHALAMIC INJURY IN TRAUMATIC BRAIN INJURY WARFIGHTERS WITH SLEEP DYSFUNCTION

Werner, K.¹ Gerstenslager, B.² Yeh, P.³ Srikanchana, R.³ Kenney, K.¹ Ollinger, J.³

¹Uniformed Services University of Health Sciences, Bethesda, MD, ²Walter Reed National Military Medical Center, Bethesda, MD, ³National Intrepid Center of Excellence, Walter Reed National Military Medical Center, Bethesda, MD.

Introduction: While sleep disorders occur in 40–70% of chronic traumatic brain injury (TBI) patients, the pathophysiology remains unknown. Increasingly, DTI has been used to evaluate gray matter structures, but no prior studies have evaluated hypothalamic regions in TBI. We hypothesized that TBI patients with poor sleep quality by questionnaire and/or polysomnography (PSG) may have structural injury to hypothalamic sleep circuitry and that this may be detectable by diffusion magnetic resonance imaging (dMRI). We examined diffusion tensor parameters in warfighters using dMRI within the hypothalamus of poor sleepers and compared them to good sleepers.

Methods: A retrospective review of 92 warfighters with blast TBI and loss of consciousness included demographics, structural MRI, dMRI, PSG and Pittsburgh Sleep Quality Index (PSQI) questionnaire. Acquisition of diffusion-weighted and structural data was performed with three Tesla MRI. Using the California Institute of Technology probabilistic high-resolution in vivo atlas as a prior, the hypothalamic nuclei were segmented by applying diffeomorphic registration of T1- and T2-weighted structural images and mapped to dMRI space.

Results: TBI patients within the lowest quartile of hypothalamic fractional anisotropy (FA) measures demonstrated decreased total sleep time (320 +/- 52 minutes vs. 382 +/- 19, p=0.006) on PSG and had more sleep complaints on PSQI (p=0.029) compared to those with the highest quartile of FA measures. There was no difference in BMI, age or AHI among the quartiles. Radial, mean and axial diffusivity quartiles did not carry significant differences in TST or PSQI. Linear models did not show significant correlation between any imaging parameter and sleep quality measures.

Conclusion: Our results reveal microstructural differences in the hypothalami of military TBI patients that may be related to clinical sleep dysfunction. Biomarkers of sleep circuitry damage may further our understanding of sleep dysfunction after TBI. Lack of correlations in linear models may be a reflection of the small sample size or a complex interaction, and removal of outliers did not change our results. Larger longitudinal studies may help clarify the association between hypothalamic and brainstem circuitry structure after TBI and sleep dysfunction.

Support: This work was supported by a grant 130132 from USAMRMC.

0047

THE CHANGE IN MELATONIN RHYTHM DEPENDING ON DEMENTIA SEVERITY IN ALZHEIMER'S DISEASE (AD) PATIENTS

Lee, J.^{1,2} Kim, S.³ Lee, S.⁴ Suh, I.⁵ Jang, J.⁶ Jhoo, J.^{1,2} ¹Department of Psychiatry, Kangwon National University Hospital, Chunchon, KOREA, REPUBLIC OF, ²Department of Psychiatry, Kangwon National University School of Medicine, Chunchon, KOREA, REPUBLIC OF, ³Department of Psychiatry, Doeun Hospital, Jincheon, KOREA, REPUBLIC OF, ⁴Department of Psychiatry, Silverheals Hospital, Namyangju, KOREA, REPUBLIC OF, ⁵Department of Laboratory Medicine, Kangwon National University Hospital, Chunchon, KOREA, REPUBLIC OF, ⁶Department of Neurology, Kangwon National University Hospital, Chunchon, KOREA, REPUBLIC OF. **Introduction:** In Alzheimer's disease (AD), both sleep and circadian dysfunctions are commonly reported and these are associated with neurodegenerative change. Actually, it has been reported that changes in circadian rhythms in AD were apparently discrete from those seen in normal aging. Previous studies reported the delayed phase in the activity or core body temperature rhythms in severe AD patients compared to normal controls. However, it is unknown whether similar changes in melatonin rhythms occur in AD patients who were not severely demented. We aimed to compare melatonin rhythms depending on dementia severity in mild and moderate AD patients.

Methods: We recruited AD patients of mild or moderate degree who had the Pittsburgh Sleep Quality Index (PSQI) score of 5 or greater and/or complained insomnia symptoms more than 3 times a week for a month. The patients were classified according to their Clinical Dementia Rating (CDR) score into 3 groups (CDR=0.5, 1, 2). The dim light melatonin onset (DLMO) was determined from seven hourly saliva samples obtained in the laboratory prior to sleep onset measured by actigraphy. The phase angle between the DLMO and sleep onset (PA-SO), and that between the DLMO and midsleep time (PA-MST) were calculated. Each group included 13, 13 and 6 AD patients with the CDR score of 0.5, 1 and 2, respectively. The DLMO and PA were compared among the 3 groups, and correlation analyses of the DLMO and PA with the MMSE in the Korean version of CERAD Packet (MMSE-KC) scores were done in total patients. Results: There was no significant difference in the DLMO and PA between the 3 groups. The MMSE-KC score was positively correlated with the DLMO and negatively correlated with the PA-MST. Conclusion: There were no changes in melatonin rhythms according to dementia severity in mild and moderate AD patients with sleep complaints. However, our study showed that earlier melatonin phase was associated with more impaired cognitive function.

Support: Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Science, ICT & Future Planning (2017R1A2B4003493)

0048

THE ASSOCIATION BETWEEN REM SLEEP AND RISK OF MORTALITY IN THREE INDEPENDENT COHORTS

Leary, E. B.¹ Watson, K. T.¹ Ancoli-Israel, S.² Redline, S.³ Yaffe, K.⁴ Ravelo, L. A.⁵ Peppard, P. E.⁵ Zou, J.¹ Goodman, S.¹ Mignot, E.¹ Stone, K. L.⁴

¹Stanford University, Palo Alto, CA, ²University of California San Diego, San Diego, CA, ³Brigham and Women's Hospital, Boston, MA, ⁴University of California, San Francisco, San Francisco, CA, ⁵University of Wisconsin-Madison, Madison, WI.

Introduction: Sleep disorders and sleep characteristics have been linked to higher risk of mortality. Despite the emerging evidence of a sleep-mortality association, the relationship between sleep architecture and mortality aren't well understood. We hypothesize that reduced REM is associated with increased mortality risk.

Methods: The Osteoporotic Fractures in Men (MrOS) study is a population-based study of 2,675 older men. Cox regression was used to evaluate the association between %REM and mortality rate. Potential covariates were evaluated using 6-fold cross validation. Sensitivity analyses were performed to rule out alternative explanations. Wisconsin Sleep Cohort (WSC) and Sleep Heart Health Study (SHHS) data were used to replicate the findings.

Results: The MrOS sample mean age was 76.3 years (SD=5.51) and the median follow-up time was 12.1 years. There was a 13% higher rate of mortality for every absolute 5% reduction in REM sleep (HR=1.13, 95%CI, 1.08–1.19) after adjusting for multiple

demographic, sleep, and health covariates. The association persisted for cardiovascular disease-related mortality (CVD) (HR=1.18, 95%CI, 1.09–1.28), cancer-related mortality (HR=1.14, 95%CI, 1.03–1.26), and other mortality (HR=1.19, 95%CI, 1.10–1.28). The WSC included 45.7% women. The mean age of the 1,388 individuals analyzed was 51.5 (SD=8.5); the median follow-up time was 20.8 years. The effect size for 5% reduction in REM on rate of all-cause mortality was similar in this cohort despite the younger age, inclusion of women, and longer follow-up period (HR=1.17, 95%CI, 1.03–1.34). SHHS data is still being analyzed; however the unadjusted model is consistent with the other cohorts.

Conclusion: We found an association between reduced REM and mortality in two, possibly three independent cohorts, which persisted across different causes of death and multiple sensitivity analyses. Mechanistic studies are needed and strategies to preserve REM may influence clinical therapies and reduce mortality risk.

Support: NHLBI provides funding for the MrOS Sleep ancillary study "Outcomes of Sleep Disorders in Older Men" under grant numbers: R01 HL071194, R01 HL070848, R01 HL070847, R01 HL070842, R01 HL070831, R01 HL070837, R01 HL070838, and R01 HL070839. Wisconsin Sleep Cohort was supported by R01HL62252, RR03186, and R01AG14124 from the NIH. Dr. Redline was partially supported by NHLBI R35 HL135818.

0049

ODDS RATIO PRODUCT AS A MEASURE OF SLEEP DEPTH DURING REM SLEEP: EFFECTS ON REM DURATION AND REM SLEEP FRAGMENTATION

Mazzotti, D. R.¹ Younes, M.²

¹Division of Sleep Medicine, Department of Medicine, University of Pennsylvania Perelman School Medicine, Philadelphia, PA, ²Sleep Disorders Centre, Department of Medicine, University of Manitoba, Winnipeg, Canada., Winnipeg, MB, CANADA.

Introduction: The odds ratio product (ORP) is a new highlyvalidated electroencephalogram biomarker of sleep depth. ORP has been validated as such by several studies investigating the effect of sleep disorders, responses to sleep deprivation and traffic noise. ORP during REM sleep varies considerably among individuals. Whether ORP reflects sleep depth also in REM sleep is unknown. We hypothesized that subjects with high REM ORP are more prone to REM sleep fragmentation.

Methods: Using data from the baseline (SHHS1; N=5,537) and follow-up (SHHS2; N=2,595) visits of the Sleep Heart Health Study, we calculated and summarized ORP in 30-second intervals corresponding to manually scored sleep stage epochs. We developed a heuristic to identify REM periods, defined as sequences of REM sleep epochs separated by no more than 10 minutes of other sleep stages or wake epochs. Using general linear models adjusted by age, sex, body mass index, race and ethnicity, we evaluated the relationship between REM ORP and total REM duration, number of awakening episodes per REM period and arousal index during REM sleep.

Results: Higher REM ORP was correlated with shorter total REM duration (ρ_{SHHS1} =-0.12; p < 0.001, ρ_{SHHS2} =-0.07; p < 0.001), more awakening episodes (ρ_{SHHS1} =0.26; p<0.001, ρ_{SHHS2} =0.30; p < 0.001) and higher arousal index (ρ_{SHHS1} =0.18; p < 0.001, ρ_{SHHS2} =0.16; p < < 0.001) during identified REM periods. In

adjusted analyses, one-unit increase in REM ORP was associated, on average, with a 7 minute decrease in total REM duration (β =-7.10; p < 0.001), 1 more awakening episode per REM period (β =1.29; p < 0.001) and an increase of 6 arousals/hour (β =6.16; p < 0.001) during REM sleep periods.

Conclusion: We found that higher REM ORP was associated with shorter REM periods, higher proportion of awake during REM periods and higher REM arousal index. Although small, these differences suggest that ORP is consistent with the concept of sleep depth also during REM sleep.

Support: None

0050

IDENTIFICATION OF A PLASMA METABOLOME-BASED BIOMARKER FOR DIM-LIGHT MELATONIN OFFSET AND ONSET IN HUMANS

Cogswell, D. T.¹ Bisesi, P. J.¹ Markwald, R. R.¹ Cruickshank-Quinn, C.² Quinn, K.² McHill, A. W.¹ Melanson, E. L.² Reisdorph, N.² Wright, K. P.¹ Depner, C. M.¹ ¹University of Colorado at Boulder, Boulder, CO, ²University of Colorado Anschutz Medical Campus, Aurora, CO.

Introduction: Easily measuring individual circadian timing is increasingly important to inform personalized chronotherapy, screen for circadian disorders and circadian misalignment, and advance circadian research. Findings from multiple studies show that transcriptomics is a viable method to estimate dim-light melatonin onset (DLMO), but no published omics-based findings have predicted dim-light melatonin offset (DLMOff), and only one known study has used metabolomics to predict DLMO. Here, we developed and tested a plasma metabolomics-based biomarker of circadian phase using DLMO and DLMOff as phase markers.

Methods: Sixteen (8M/8F) healthy participants aged 22.4 \pm 4.8y (mean \pm SD) completed an in-laboratory study with 3 baseline days (9h sleep opportunity/night), followed by a randomized cross-over protocol with 9h sleep and 5h sleep conditions, each lasting 5 days. Blood was collected every 4h on the final 24h of each condition for untargeted metabolomics analyses. DLMO and DLMOff were determined during the final 24h of each condition. Samples from all conditions were randomly split into training (68%) and test (32%) datasets. DLMO and DLMOff models were developed using partial least squares regression in the training dataset and validated in the test dataset.

Results: When validating with the test dataset, R^2 for the DLMO model was 0.60, median absolute error (MdAE) was 2.2 ± 2.8h (± interquartile range), and 44% of samples had MdAE under 2h. R^2 for the DLMOff model was 0.62, MdAE was 1.8 ± 2.6, and 51% of samples had MdAE under 2h. The DLMOff model predicted baseline samples, under conditions of 9h sleep and controlled food intake, with an R^2 of 0.91 and MdAE 1.1 ± 1.1h.

Conclusion: These findings show promise for metabolomicsbased biomarkers of circadian phase and highlight the need for biomarker efforts to predict multiple circadian phase markers. Additional analyses with an independent validation dataset will help advance these initial findings.

Support: NIH-R01HL085705, NIH-R01HL109706, NIH-R01HL132150, NIH-K01HL145099, NIH-F32DK111161, and NIH-UL1TR000154; and Sleep Research Society Foundation 011-JP-16;

RESPIRATORY CYCLE-RELATED EEG CHANGES (RCREC) PREDICT INCIDENCE AND RECURRENCE OF CARDIOVASCULAR DISEASE

Tsimpanouli, M.¹ Chervin, R. D.¹ Gliske, S. V.¹ ¹University of Michigan, Ann Arbor, MI, ²University of Michigan, Ann Arbor, MI.

Introduction: Sleep-disordered breathing (SDB) is a common sleep disturbance and is associated with both incident and recurrent cardiovascular disease (CVD). Respiratory Cycle-Related EEG Changes (RCREC), an SDB biomarker, are thought to reflect inspiratory microarousals and are based on quantitative analysis of sleep EEG and breathing. The objective of this study was to assess whether RCREC may predict CVD incidence or recurrence in a large cohort of adults. The RCREC for several frequency bands have been previously shown to predict either higher or lower all-cause mortality in the same cohort.

Methods: Data were obtained from the Sleep Heart Health Study (SHHS), a multicenter longitudinal study that included polysomnograms in middle-aged to older adults. Information about CVD events was collected at baseline and for up to 16 years later. The RCREC values at baseline were computed in the delta, theta, alpha, sigma, beta, and gamma frequency bands during scored epochs of sleep. Cox Proportional Hazard models, were used to assess the relation of each RCREC frequency band and incidence or recurrence of CVD. These models were stratified by sex and adjusted for body-mass index, age, race, smoking status, diabetic status, hypertensive status, HDL cholesterol, LDL cholesterol, and the apnea-hypopnea index (AHI).

Results: There were 3,032 adults with sufficient data quality (mean age at baseline $62\pm11(SD)$ years, 58% female). Among 2,500 adults with no reported prior CVD history at baseline, the adjusted odds ratios (95% CI) for delta RCREC 0.948(0.920-0.977), theta RCREC 0.938(0.895-0.984), and alpha RCREC 0.946(0.902-0.993) separately suggested associations with lower CVD incidence, whereas gamma RCREC 1.017(1.001-1.032) predicted a marginal increase. Among 532 adults having prior CVD history at baseline, delta RCREC 0.958(0.927-0.989) and sigma RCREC 0.931(0.895-0.969) separately predicted decreased CVD recurrence. The apneahypopnea index (AHI) was not similarly predictive in any model. Conclusion: The RCREC for several frequency bands, in contrast to AHI, may predict CVD incidence and recurrence. The directionality of the association was surprising and merits further exploration. Support: NIH:NCATS-TL1-TR-002242, BD2K-K01-ES-026839, HL105999

0052

WITHDRAWN

0053

EFFECT OF CHANGES IN INTRACELLULAR ADHESION MOLECULE-1 ON MEASURES OF SLEEPINESS AND 24-HOUR AMBULATORY BLOOD PRESSURE AFTER 4 MONTHS OF CONTINUOUS POSITIVE AIRWAY PRESSURE

Pak, V. M.¹ Maislin, D.² Keenan, B. T.² Townsend, R.³ Dunbar, S. B.¹ Pack, A. I.² Gislason, T.⁴ Kuna, S. T.^{2,5} ¹Emory University, School of Nursing, Atlanta, GA, ²Division of Sleep Medicine, Department of Medicine, University of Pennsylvania, Perelman School of Medicine, Philadelphia, PA, ³University of Pennsylvania, Nephrology and Hypertension, Philadelphia, PA, ⁴University of Iceland, Reykjavik, ICELAND, ⁵Sleep Medicine Section, Crescenz Veterans Affairs Medical Center, Philadelphia, PA.

Introduction: Previous studies have shown that continuous positive airway pressure (CPAP) therapy of adults with obstructive sleep apnea (OSA) reduces circulating levels of intercellular adhesion molecule 1 (ICAM-1). ICAM-1 levels may affect daytime sleepiness and elevated blood pressure associated with OSA. Our goals were to explore associations between changes in ICAM-1 and objective and subjective measures of sleepiness, as well as 24-hour ambulatory blood pressure monitor (ABPM) parameters in adults with OSA following 4 months of CPAP treatment.

Methods: We identified 140 adults with newly diagnosed OSA in the Penn Icelandic Sleep Apnea (PISA) Study, with a mean (±SD) body mass index (BMI) of 31.5±4.2 kg/m² and apnea-hypopnea index (AHI) of 36.8±15.3 events/hour; 83.3% were males. Plasma ICAM-1 levels, 24-hour ABPM, Epworth Sleepiness Scale (ESS), and Psychomotor Vigilance Task (PVT) measures were obtained at baseline and after 4 months of CPAP treatment. Associations between changes in natural log ICAM-1 and both sleepiness and 24-hour mean arterial blood pressure (MAP) were assessed using multivariate regression models, controlling for *a priori* baseline covariates of age, sex, BMI, race, site, smoking status, physical activity, use of antihypertensive medications, AHI and hours/night of CPAP usage.

Results: Overall, there was no significant change in ICAM-1 from baseline to follow-up among all participants after 4 months (0.027 ng/ml, p=0.52). There were no statistically significant associations between the change in ICAM-1 and change in sleepiness measures (all p>0.05) or 24-hour MAP (1.124 mm Hg, p=0.07). A nominal association between increased ICAM-1 and increased daytime MAP after 4 months was observed (1.39 mm Hg, p=0.033), although this result was not significant after correction for multiple comparisons.

Conclusion: Our results do not support changes in ICAM-1 as the biological pathway linking changes in sleepiness or ABPM following CPAP treatment of adults with OSA.

Support: P01-HL094307 (NHLBI, PI: Pack AI)

0054

METABOLITE PROFILES OF OBSTRUCTIVE SLEEP APNEA DISTINGUISHES CASES FROM CONTROLS AND IMPROVE WITH CPAP

Sengupta, A.¹ Lim, D. C.² Keenan, B. T.² Keele, L.² Pack, A.² Weljie, A.²

¹University of Pennsylvania, Philadelphia, PA, ²University of Pennsylvania, Philadelhia, PA.

Introduction: Obstructive sleep apnea (OSA) is a common sleep breathing disorder with significant public health consequences. Despite this, no clinically available objective molecular biomarkers to diagnose, risk stratify and quantify treatment efficiency exist. To this end, high-throughput metabolomics data could serve as a valuable quantitative tool.

Methods: We designed a pilot study to investigate the metabolomic effects of OSA and CPAP treatment. Blood serum samples were collected from OSA patients and healthy controls matched with respect to age (\pm 5 years), BMI (\pm 2.5 kg/m²) and gender (N = 20/ group). Samples from OSA patients were obtained before and after continuous positive airway pressure (CPAP) treatment. Polar metabolites were analyzed using a targeted ultra-performance liquid

chromatography-tandem mass spectrometry (UPLC-MS/MS) metabolomics technique.

Results: Supervised multivariate analysis using serum metabolic values of OSA patients and healthy controls showed a significantly different overall metabolic profile between the two groups (orthogonal partial least squares discriminant analysis [OPLS-DA] Q²=0.25, p=0.04). Acetylornithine, choline, cytidine, dodecenoylcarnitine, methionine sulfoxide and 3-indoxylsulfate were among the most perturbed metabolites. Major metabolic pathways altered in the OSA patients were methionine and phospholipid metabolism, as well as gut microbial co-metabolism. Lysophosphatidylcholine (16:0), a phospholipid metabolite, demonstrated significant linear association with improved oxygen saturation nadir post CPAP treatment (R² = 0.57), suggesting the metabolic features may be used as prognostic clinical biomarkers.

Conclusion: These results suggest that OSA significantly impacts blood metabolites, which could potentially be used to establish OSA biomarkers. Moreover, specific metabolic features are associated with post CPAP improvement, such as phospholipids, suggesting a functional association of these metabolites that may help us understand the heterogeneity of OSA. Overall, these results demonstrate the potential of metabolic profiling to develop quantitative molecular markers of OSA. Further studies are underway to validate these findings and investigate the utility of metabolic profiles to objectively measure CPAP efficacy.

Support: The work was supported by the program project grant P01 HL094307.

0055

PHYSIOLOGICAL BASED PREDICTIVE MODELS OF VIGILANCE

Daley, M. S.¹ Gever, D. H.¹ Chon, K. H.² Posada-Quintero, H.² Bolkhovsky, J. B.¹

¹Naval Submarine Medical Research Laboratory, Groton, CT, ²University of Connecticut, Storrs, CT.

Introduction: The Naval Submarine Medical Research Laboratory (NSMRL) is developing predictive models to examine how non-invasive, non-disruptive physiological monitoring can be used to track performance decrements due to sleep deficiency. Utilizing biometrics extracted from physiological measures to track performance changes would allow for automated tracking of fatigue and alleviate the overhead necessary to monitor individual schedules and sleep patterns.

Methods: NSMRL collaborated with the University of Connecticut to run a sleep deprivation study that deprived 20 participants of sleep for a period of up to 25 hours. During this time, subjects completed multiple tasks, including the Psychomotor Vigilance Test (PVT) every few hours. A non-invasive monitoring system collected physiological data from participants, which includes eye tracking, electrocardiography, electrodermal activity, and facial tracking (e.g., blink metrics, heart rate variability, skin conductance levels, facial action units). Using this multimodal approach, biometrics were extracted and evaluated to determine their predictive power on PVT performance. Multiple linear regression, using predictors selected via sequential forward selection, was used to develop a model of performance at an individual level based on a subset of these metrics chosen using principal component regression.

Results: Thirty-eight biometrics were extracted from the collected data and used to produce a predictive model of PVT performance. Sequential forward selection was used to select 11 primary

biometrics. The criteria for primary metric inclusion in the model was minimization of root mean squared error. The resultant model had a correlation coefficient (r) of 0.71 (p < 0.001) with a root mean squared error (RMSE) of 49.8 ms between the predicted reaction time and true reaction time for each subject.

Conclusion: Non-invasive, non-disruptive monitoring could be used to track individual cognitive performance decrement due to sleep deficiency. This study examined the capability of combining the data from four physiological monitors that can be contained within a wrist worn device and a desk or helmet mounted camera. Utilizing 11 biometrics obtained from these monitors a stepwise regression model was developed that significantly correlates with PVT reaction time at both an individual and group level.

Support: This work was supported by the Military Operational Medicine Research Program.

0056

IDENTIFICATION OF A STABLE HUMAN METABOLOMICS-BASED BIOMARKER OF INSUFFICIENT SLEEP AND ITS ASSOCIATION WITH COGNITIVE PERFORMANCE

Depner, C. M.¹ Reisdorph, N.² Wright, K.¹

¹Sleep and Chronobiology Laboratory, Department of Integrative Physiology, University of Colorado Boulder, Boulder, CO, ²Skaggs School of Pharmacology, University of Colorado Anschutz Medical Campus, Aurora, CO, ³Sleep and Chronobiology Laboratory, Department of Integrative Physiology, University of Colorado Boulder, Boulder, CO.

Introduction: There are numerous potential benefits of developing objective biomarkers of insufficient sleep including providing screening and diagnostic tools, increasing our understanding of poor sleep health, and supporting sleep-based countermeasures. We analyzed plasma metabolites in humans during two separate occasions of experimental sleep restriction to identify stable biomarkers of insufficient sleep, and assessed the association of these biomarkers with cognitive performance.

Methods: 12 healthy adults (6M/6F), aged $24\pm5y$ (mean \pm SD), completed two 18-day protocols separated by 10 days. For each 18-day protocol, participants maintained habitual 9h sleep schedules for two weeks at home, and then completed 4-day laboratory visits with sleep opportunities of: 9h on night 1, 5h on nights 2 and 3, and recovery sleep on night 4. Energy balanced diets were provided 2 days before and throughout laboratory visits. Blood was collected every 2h during scheduled wakefulness on days 1–2 (baseline) and days 3–4 (insufficient sleep), and was analyzed by untargeted liquid chromatography/mass-spectrometry. Sustained attention was assessed every 2 h during scheduled wakefulness with the Psychomotor Vigilance Test (PVT).

Results: After filtering, we detected 6,822 metabolites. Elasticnet regression identified 21 metabolites consistently altered by insufficient sleep in both 18-day protocols. We developed a logistic regression-based biomarker fingerprint of insufficient sleep using 11 of these 21 metabolites with consistent responses to insufficient sleep. This biomarker fingerprint has 74.4% accuracy and 0.822 (0.782–0.858; 95%CI) area under the receiver operator curve. Median reaction time was slower (P<0.05) during insufficient sleep versus baseline. Mediation analyses show our biomarker fingerprint accounts for ~6% of the slower reaction time.

Conclusion: We identified a plasma metabolomics-based biomarker of insufficient sleep with "good" performance. However, this biomarker only accounts for $\sim 6\%$ of reduced PVT performance during insufficient sleep. Our findings suggest that specific

biomarkers for insufficient sleep versus cognitive deficits associated with insufficient sleep may be required.

Support: This work was supported by NIH R01HL132150, NIH K01HL145099, NIH F32DK111161, NIH/NCATS Colorado CTSA Grant UL1TR002535, Sleep Research Society Foundation 011-JP-16, and the University of Colorado Boulder Undergraduate Research Opportunities Grant.

0057

ACTIGRAPHY-BASED CIRCADIAN MEASURES AND CEREBROSPINAL FLUID BIOMARKERS OF NEURODEGENERATION IN ALZHEIMER'S DISEASE WITH MILD COGNITIVE IMPAIRMENT

Mehra, R.¹ Bhambra, R.¹ Bena, J.² Bekris, L.³ Leverenz, J.⁴ Rao, S.⁴ Foldvary-Schaefer, N.¹ Rao, S.⁵ Pillai, J.⁴ ¹Sleep Disorders Center, Cleveland, OH, ²Quantitative Health Sciences, Cleveland, OH, ³Genomics Medicine Institute, Cleveland, OH, ⁴Lou Ruvo Center for Brain Health, Cleveland, OH, ⁵Cole Eye Institute, Cleveland, OH.

Introduction: Although recent data implicates sleep and circadian disruption to neurodegeneration in Alzheimer's Disease (AD), the association of objective circadian biomarkers and neurodegeneration remains understudied. We hypothesize that actigraphy-based circadian measures are associated with cerebrospinal fluid (CSF) biomarkers of neurodegeneration in those mild cognitive impairment due to AD (MCI-AD).

Methods: Eighteen patients with CSF biomarker-confirmed MCI-AD underwent actigraphy monitoring generating the following circadian measures: amplitude, F-ratio and mesor and morning collection of CSF biomarkers of neurodegeneration (A β 42,t-tau,p-tau). Linear models were used to evaluate the association of circadian and CSF measures; logarithmic transformations were performed on neurodegenerative markers for greater normality. Analysis was performed using SAS software. A significance level of 0.05 was assumed for all tests.

Results: Eighteen MCI-AD patients who were 68 ± 6.2 years, 44% female, with median AHI=12 and underwent actigraphy monitoring for 8.2+/-3.2 days were included. There was no significant association of circadian measures and A β 42 nor with mesor and neurodegeneration biomarkers. Amplitude was associated with both p-tau and t-tau, such that each 10 unit increase in amplitude resulted in a predicted increase in p-tau of 8% (95% CI:1%-15%, p=0.018) and an increase of 13% (3%-23%; p=0.01) in t-tau. F-ratio was positively associated with p-tau and t-tau; each 1000 unit increase in F-ratio resulted in a predicted 12% (4%-22%; p=0.007) increase in P-tau and 20%(6%-35%; p=0.005) increase in t-tau. Associations of these circadian measures and CSF levels of p-tau and t-tau remained statistically significant after adjustment for age and sex.

Conclusion: Among patients with symptomatic MCI stages of AD, objective measures of circadian rhythm disruption are associated with CSF-based biomarkers of neurodegeneration even after consideration of age and sex. Future investigation should clarify directionality of this association and potential utility of circadian-based interventions in the mitigation of AD progression. **Support:** N/A

0058

EFFECTS OF ACUTE TOTAL SLEEP DEPRIVATION ON HUMAN PLASMA N-GLYCANS

Chatterton, B. D.¹ Mullington, J.^{1,2} Yang, H.^{1,2} Haack, M.^{1,2} Cummings, R.^{3,2} Lehoux, S. D.^{3,2} ¹Department of Neurology, Beth Israel Deaconess Medical Center, Boston, MA, ²Harvard Medical School, Boston, MA, ³Department of Surgery, Beth Israel Deaconess Medical Center, Boston, MA.

Introduction: There is a need for a novel biomarker that can be used to measure sleep sufficiency as it pertains to fitness for duty. As glycans (polysaccharides) are known to be involved in modifying protein effectiveness, we are exploring these as biomarkers that may be sensitive to differences between sleep deprivation and normal healthy adult sleep duration. We have measured one major class of glycans, called N-glycans, which are covalently linked to asparagine residues of polypeptide chains of membrane-bound and secreted proteins. We compared the plasma N-glycan profiles of participants before and after they participated in a total sleep deprivation protocol.

Methods: 10 healthy participants (6 male, 4 female) aged 30–44 went through 88 hours of total sleep deprivation. Hourly blood draws were taken via forearm catheter throughout the protocol. N-glycan analysis was performed using plasma samples collected at 17:35 prior to the first night of sleep deprivation and at 17:35 following 82.5 hours of continuous wakefulness. N-glycans were first cleaved from peptides and isolated from plasma, and profiles were then measured using Matrix-Assisted Laser Desorption/ Ionization-Time of Flight (MALDI-TOF) mass spectrometry.

Results: 66 N-glycans were observed in our profiles. Of these, the relative abundance of 17 N-glycans were significantly different following sleep deprivation (paired t-test, 13 with p<0.05, 4 with p<0.01). In each case, the relative abundance was lower in the sleep deprivation time point. We found two structures, Hex6HexNAc5NeuAc3 and Hex7HexNAc6NeuAc2, which were also significant in one of our previous chronic sleep restriction protocols.

Conclusion: While we observed that many N-glycans decreased in relative abundance, it is unclear whether these changes represent a shift in glycan synthesis or result from decreased expression of the proteins they are bound to. Our next steps involve exploring the functions of the proteins associated with Hex6HexNAc5NeuAc3 and Hex7HexNAc6NeuAc2, and measuring their expression levels. **Support:** NIH/HL75501; NIH/National Center for Research Resources UL1-RR02758 and M01-RR01032 to the Harvard Clinical and Translational Science Center.

0059

DISRUPTION OF CIRCADIAN CLOCK PROTEINS IN OBSTRUCTIVE SLEEP APNEA PATIENTS

Gabryelska, A. Sochal, M. Bialasiewicz, P. Department of Sleep Medicine and Metabolic Disorders, Medical University of Lodz, Lodz, POLAND.

Introduction: Circadian clocks are endogenous coordinators of 24-hour rhythm of behavioral and molecular processes in living organism. They are composed of set of genes, which function as activators - CLOCK and BMAL1, which through binding to regulatory elements containing E-boxes activate transcription of repressor proteins Period (PER1) and cryptochrome (CRY1). The aim of the study was to assess: CLOCK, BMAL1, PER1 and CRY1 in obstructive sleep apnea (OSA) patients.

Methods: The study included 20 individuals, who underwent PSG and based on apnea-hypopnea index (AHI) were divided into severe OSA group (n=10; AHI30; 90% male) and healthy control (n=10; AHI<5; 70% male). All participants had their peripheral blood collected in the evening (9:00-10:00 pm) before and in the

morning (6:00-7:00 am) after the PSG. CLOCK, BMAL1, CRY1 and PER1 protein concertation measurements were performed using ELISA.

Results: Increased level of following proteins was observed in OSA group: evening CLOCK (p=0.037), morning CLOCK (p=0.019), morning BMAL1 (p=0.0.16), evening PER1 (p=0.004), morning PER1 (p=0.029) and evening CRY1 (p=0.035). Yet, no significant difference was found between morning and evening level of any of the proteins in OSA and control group. Additionally, morning level of activator proteins CLOCK and BMAL1 had positive correlation with AHI (p=0.022, R=0.510 and p=0.010, R=0.560, respectively) and desaturation index (p=0.209, R=0.487 and p=0.009, R=0.570, respectively), while for repressor proteins PER1 and CRY1 significant correlations were found with desaturation index in the evening (p=0.025, R=0.500 and p=0.048, R=0.448, respectively), AHI in REM stage (p=0.009, R=0.569 and p=0.027, R=0.495, respectively) and AHI (for PER1 only p=0.014, R=0.540).

Conclusion: OSA patients have increased level of circadian clock proteins that corelates with severity of the disease. Further research is needed into the disruption of circadian clock should in OSA patients and possible effect of OSA treatment on concentrations of these proteins should be investigated.

Support: The study was financed by Polish National Centre Grant no. 2018/31/N/NZ5/03931.

0060

MORNING LOCUS COERULEUS ACTIVATION DURING THE PVT PREDICTS LATER-DAY SLEEPINESS

LEI, H.¹ QUAN, P.¹ LIU, W.¹ ZHANG, X.¹ CHAI, Y.¹ YANG, F.¹ DINGES, D.² RAO, H.^{1,2}

¹Center for Functional Neuroimaging, Department of Neurology, University of Pennsylvania, Philadelphia, PA, ²Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA.

Introduction: The locus coeruleus (LC) plays a key role in the regulation of arousal and autonomic function. Homeostatic sleep pressure refers to the drive for sleep that increases as a saturating exponential when we stay awake and decreases exponentially when we sleep. The current study used arterial spin labeling (ASL) functional magnetic resonance imaging (fMRI) to investigate the relationship between homeostatic sleep pressure (sleepiness) and LC activity during the psychomotor vigilance test (PVT).

Methods: We analyzed sleepiness and ASL imaging data from N=70 health adults (40 males, age range 21-50 years) who participated in a controlled in-laboratory sleep study. All participants were scanned at rest and during the PVT on the morning between 0700h-1000h after 9 hour time-in-bed (TIB) baseline sleep. LC regions-of-interest (ROI) were defined by standard templates from Keren et al. (2009). Sleepiness was assessed by the Karolinska Sleepiness Scale (KSS) every two hours from 10:30 am to 10:30 pm. Results: Sleepiness scores gradually increased over wakefulness time and reached its peak in the evening at about 10:20pm. PVTinduced CBF changes did not correlate with sleepiness scores on the morning (p > 0.05), but showed significant negative correlations with sleepiness scores on later day when sleep pressure became higher, especially during the night-time (r = -0.41, p < 0.001). Specifically, LC CBF showed significant increases during the PVT scan as compared to the resting scan (p = 0.04) in individuals with less nigh-time sleepiness (KSS < 4), but no differences (p > 0.1)in individuals with greater nigh-time sleepiness (KSS \geq 5). After controlling for age, gender, and total sleep time, PVT-induced

regional CBF difference in the LC still negatively predicted sleepiness ($\beta = -0.325$, p = 0.005).

Conclusion: Our findings showed that individuals with greater LC CBF increases during the PVT were less sleepy during the night, supporting the key role of LC activity in promoting wakefulness and maintaining sleep homeostasis. PVT-induced LC activation may provide a non-invasive bio-marker of homeostatic sleep pressure in healthy adults.

Support: Supported in part by NIH grants R01-HL102119, R01-MH107571, R21-AG051981. CTRC UL1RR024134, and P30-NS045839.

0061

DOES COMBINING M1 M2 REFERENCE INFLUENCE AMPLITUDE OF SLOW WAVES?

Walker, N. A. Roth, H. L. Fan, Z. Vaughn, B. V. UNC at Chapel Hill, Chapel Hill, NC.

Introduction: Slow wave amplitudes are critical to determining Stage N3 sleep yet ECG artifact frequently interferes with accurate amplitude measurement. This artifact may be lessened by using a combined M1-M2 reference however theoretically this may decrease the amplitude due to shorter inter-electrode distance (predicted 27% loss). The AASM Scoring Manual recommends scoring slow wave activity using F4-M1 channel or alternatively F3-M2, but does not recognize a combined reference. This study measures the differences in slow wave amplitude using contralateral versus combine reference.

Methods: 12 polysomnograms were randomly selected for analysis of amplitude of slow wave using contralateral and combined reference channels. Six separate EEG channels (F3-M1, F3-M2, F3-M1+M2, F4-M1, F4-M2, and F4-M1+M2) were used to analyze 25 different slow waves from each polysomnogram. Individual slow waves from Stage N3 sleep were analyzed using the Natus Sleepworks Amplitude Measurement Tool if their peak and trough were free EKG artifact. Averages and standard deviations of the waveforms were calculated for each patient and channel. Differences were normalized by dividing by the amplitude of the original wave using the contralateral reference.

Results: Subjects age ranged from 30–69 yrs, with 6 being females. Mean amplitudes were as follows: F3-M2 was 131.75 μ V, F3-M1+M2 125.84 μ V, F4-M1 130.57 μ V, and F4-M1+M2 128.22 μ V. The overall average difference of F4-M1 to F4-M1+M2 was 0.92% and the average difference of F3-M2 to F3-M1+M2 was 3.52% with the average standard deviation of 8.47%.

Conclusion: This study shows the average loss in amplitude of converting F4-M1 to F4-M1+M2 was less than 1% and 3.5% for F3-M2 to F3-M1+M2. Combining M1M2 reference may be a valuable alternative to reduce EKG artifact.

Support: None

0062

IMPROVED CIRCADIAN DATA ORDERING IN THE PRESENCE OF BIOLOGICAL AND TECHNICAL CONFOUNDS

Hammarlund, J.¹ Anafi, R.²

¹Drexel University School of Biomedical Engineering and Health Systems, Philadelphia, PA, ²University of Pennsylvania, Philadelphia, PA.

Introduction: We recently used unsupervised machine learning to order genome scale data along a circadian cycle. CYCLOPS (Anafi et al PNAS 2017) encodes high dimensional genomic data onto

an ellipse and offers the potential to identify circadian patterns in large data-sets. This approach requires many samples from a wide range of circadian phases. Individual data-sets often lack sufficient samples. Composite expression repositories vastly increase the available data. However, these agglomerated datasets also introduce technical (e.g. processing site) and biological (e.g. age or disease) confounders that may hamper circadian ordering.

Methods: Using the FLUX machine learning library we expanded the CYCLOPS network. We incorporated additional encoding and decoding layers that model the influence of labeled confounding variables. These layers feed into a fully connected autoencoder with a circular bottleneck, encoding the estimated phase of each sample. The expanded network simultaneously estimates the influence of confounding variables along with circadian phase.

We compared the performance of the original and expanded networks using both real and simulated expression data. In a first test, we used time-labeled data from a single-center describing human cortical samples obtained at autopsy. To generate a second, idealized processing center, we introduced gene specific biases in expression along with a bias in sample collection time. In a second test, we combined human lung biopsy data from two medical centers.

Results: The performance of the original CYCLOPS network degraded with the introduction of increasing, non-circadian confounds. The expanded network was able to more accurately assess circadian phase over a wider range of confounding influences.

Conclusion: The addition of labeled confounding variables into the network architecture improves circadian data ordering. The use of the expanded network should facilitate the application of CYCLOPS to multi-center data and expand the data available for circadian analysis. **Support:** This work was supported by the National Cancer Institute (1R01CA227485-01)

AGE RELATED CHANGES IN CENTRAL AUTONOMIC COUPLINGS DURING SLEEP

Chen, P.¹ Naji, M.² Sattari, N.¹ Whitehurst, L. N.³ Mednick, S. C.¹ ¹Department of Cognitive Science, UC Irvine, CA, USA, Irvine, CA, ²School of Medicine, UCSD, CA, USA, San Diego, CA, ³School of Psychiatry, UCSF, CA, USA, San Francisco, CA.

Introduction: Studies show coupling between central nervous system (CNS) and autonomic nervous system (ANS) activity during sleep. We reported on a novel central/autonomic coupling event (ACE) during non-rapid eye movement (NREM) sleep, in which bursts in heart rate (HRBs) coincide with increased slow-wave activity (SWA) 5 seconds prior to the HRB, followed by a surge in vagal high-frequency activity in the RR signal (HFRR) 5 seconds after the HRB. ACEs predicted sleep-related explicit memory improvement. Aging is characterized with impaired sleep and autonomic loss. We, therefore, investigated ACE activity in older adults.

Methods: We compared ACEs during a daytime nap between youngers (18-25yrs, N=49) and olders (60-75yrs, N=32). Subjects took an EEG-monitored, 90-minute nap. We measured SWA and HFRR in a 20-sec window around the HRB peak separately for Stage 2 and slow-wave sleep (SWS). EEG were binned into 5-sec intervals around the HRB: -10, -5, +5, +10 bins. For Stage 2 and SWS, repeated-measure ANOVAs with two factors (age and windows) were performed on SWA and HFRR. Corrections used Greenhouse-Geisser and Bonferroni methods.

Results: For SWA, we found an interaction between age and windows during Stage 2 (p<.001), and SWS (p=.001). SWA during the -5bin was greater in youngers than olders during both Stage 2 and SWS (ps < .001). The ACE profile in youngers showed highest SWA in the -5bin in Stage 2 and SWS (ps < .001) and highest HFRR in the +5bin in Stage 2 (ps < .001) The ACE profile in olders, however, showed no clear pattern for SWA in either sleep stage. Olders showed greater HFRR during the +5bin compared to the -10bin (p=.041) during Stage 2 but no HFRR modulations were found during SWS.

Conclusion: Our results replicated the ACE profile in daytime naps first reported by Naji et al (2018). In youngers, heart rate bursts were coupled with increased SWA and vagal activity. In contrast, olders demonstrated a lack of boost in SWA and HFRR, which provides implications in cognitive aging. Future research is needed to further understand the impact of decreased coupling between ANS/CNS activity on cognitive decline. **Support:**

0064

HEIGHTENED NEURAL RESPONSES TO NEGATIVE WORDS IN SHIFT WORKERS USING THE STROOP TASK

Lee, K.¹ Lee, H.¹ Jeon, J.¹ Jeon, S.² Kim, N.³ Oh, S.⁴ Lee, M.¹ Kim, S.² Lee, Y.¹

¹Department of Psychiatry and Center for Sleep and Chronobiology, Seoul National University, College of Medicine and Hospital, Seoul, KOREA, REPUBLIC OF, ²Sungkyunkwan University College of Medicine, Seoul, KOREA, REPUBLIC OF, ³Gachon University, Incheon, KOREA, REPUBLIC OF, ⁴Department of Psychiatry, Dongguk University Ilsan Hospital, Ilsan, KOREA, REPUBLIC OF.

Introduction: Shift work is known to have a negative impact on a wide range of health problems such as sleep disturbance, cognitive

impairment, and emotional disorders (e.g., anxiety and depression). It is important to understand underlying mechanisms for negative impact of shift work on health problems. This study aimed to investigate psychological and neural mechanisms associated with shift work.

Methods: Thirty six shift workers (28 females, age = 29.9 ± 7.4) and 35 non-shift workers (20 females, age = 30.5 ± 5.5) participated in this study. They were performing the word Stroop task during fMRI scanning. This task included sleep-related words and negative words to investigate neural substrates associated with sleep-related information and emotional information processing. Neutral words were included as control stimuli. The participants also completed questionnaires assessing sleep-related problems such as Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale, and emotion-related problems such as Beck Depression Inventory and Beck Anxiety Inventory. Two-sample t-tests were conducted to find group differences in self-report measures and neural response to sleep-related words and negative words compared to neutral words. Results: Relative to non-shift workers, shift workers showed greater sleep disturbance (i.e., higher PSOI), but they did not show any evidence of emotion-related problems. Shift workers also demonstrated greater neural response to negative words (vs. neutral words) in several prefrontal regions (e.g., dorsal anterior cingulate cortex and dorsolateral prefrontal cortex), anterior insula and caudate compared to non-shift workers. However, shift workers did not show significantly different neural response to sleep-related words (vs. neutral words) compared to non-shift workers.

Conclusion: The result from this study provides supporting evidence that shift work is associated with subjective sleep disturbance. Shift workers' heightened neural response to negative information may reflect their increased sensitivity to negative information, that may contribute to sleep disturbance.

Support: Brain Research Program through the National Research Foundation of Korea funded by the Ministry of Science, ICT & Future Planning (Study No.: 2016M3C7A1904338 and NRF-2018R1D1A1B07049704).

0065

INITIAL FINDINGS ON ASSOCIATIONS BETWEEN SLEEP AND CLINICAL MEASURES WITH NEURAL ACTIVATIONS ACCOMPANYING FEAR CONDITIONING AND EXTINCTION IN TRAUMA-EXPOSED INDIVIDUALS

Seo, J. Oliver, K. I. Daffre, C. Pace-Schott, E. F. Massachusetts General Hospital, Harvard Medical School, Charlestown, MA.

Introduction: We examined associations of sleep and hyperarousal with neural responses to a fear conditioning and extinction protocol in trauma-exposed individuals. We hypothesized, greater hyperarousal, poorer sleep quality and more nightmares would accompany greater activation of the salience network (associated with fear) and lesser activation of the prefrontal cortex (PFC; associated with fear regulation) throughout this protocol.

Methods: Persons exposed to trauma within the past 2 years (N=119, 43 with Post-traumatic stress disorder; PTSD) completed the PTSD Checklist-5 (PCL-5), two weeks of actigraphy and sleep/nightmare diaries, and a 2-day fear-conditioning and extinction protocol during fMRI. Hyperarousal items from PCL-5, sleep quality, and nightmare frequency were used to predict fMRI contrasts representing (1) initial activations to reinforced conditioned stimuli (CS+) during fear conditioning, (2) change in neural activation to CS+s across extinction

SLEEP, Volume 43, Abstract Supplement, 2020

learning, and, (3) after 24 hours (extinction recall), selective activation to an extinguished (CS+E) versus an un-extinguished CS+.

Results: During fear conditioning, hyperarousal was positively correlated with activation to the CS+ in the right lateral PFC, whereas nightmare frequency was negatively correlated with activations in bilateral orbitofrontal cortex (OFC). Across extinction learning, sleep onset latency (SOL) was negatively correlated with increased activation to CS+ in bilateral insular and dorsal and middle anterior cingulate cortices (salience regions). At extinction recall, nightmare frequency was negatively correlated with selective activation to the CS+E in the left insular cortex.

Conclusion: Except that fewer nightmares predicted greater OFC activation during fear conditioning, results did not support hypothesized relationships of hyperarousal, poor sleep and nightmares with increased salience network and decreased PFC activation to fear-related stimuli.

Support: Funding: R01MH109638

0066

GLYCINERGIC POSTSYNAPTIC INHIBITION IS RESPONSIBLE FOR THE SUPPRESSION OF HYPOGLOSSAL MOTONEURON ACTIVITY DURING NATURALLY-OCCURRING REM SLEEP

Tobin, C.^{1,2} Fung, S. J.^{1,2} Xi, M.^{1,2} Chase, M. H.^{1,2,3}

¹WebSciences International, Los Angeles, CA, ²VA Greater Los Angeles Healthcare System, Los Angeles, CA, ³UCLA School of Medicine, Los Angeles, CA.

Introduction: The present study was undertaken to explore the role of glycinergic postsynaptic inhibition and monoaminergic disfacilitation (a withdrawal of excitatory noradrenergic and serotonergic inputs) in the control of hypoglossal motoneuron activity during REM sleep. Accordingly, glycinergic, noradrenergic and serotonergic antagonists were microinjected into the hypoglossal nucleus, and their effects on the hypoglossal nerve activity during REM sleep were examined in chronically-instrumented, unanesthetized cats.

Methods: Adults cats were prepared for monitoring behavioral states of sleep and wakefulness, and for extracellular recordings from hypoglossal nerve. Strychnine (a glycinergic antagonist) and a mixture of prazosin (a noradrenergic antagonist) and methysergide (a serotonergic antagonist) were microinjected, separately, into the hypoglossal nucleus during naturally-occurring states of sleep and wakefulness.

Results: During REM sleep, compared to non-REM sleep, the hypoglossal nerve activity decreased by $17.4\pm1.5\%$ (n=17) in the control recordings (prior to the injection of strychnine). Following the microinjection of strychnine, there was only a mean decrease of $7.2\pm1.2\%$ (n=12) in the nerve activity during REM sleep versus NREM sleep. The strychnine effect was statistically significant compared to control (p<0.001; unpaired t-test), which indicates that strychnine blocks REM sleep-related suppression of hypoglossal nerve activity. In contrast, the microinjection of prazosin and methysergide did not significantly reduce the hypoglossal nerve activity during REM sleep (control: 15.9 ± 2.3 , n=9 vs. prazosin+methysergide: $12.6\pm1.4\%$, n=10, p=0.229, unpaired t-test).

Conclusion: The present results demonstrate that the microapplication of strychnine, but not prazosin and methysergide, into the hypoglossal nucleus significantly reduces the suppression of the hypoglossal nerve activity during naturally-occurring REM sleep. We therefore suggest that glycinergic postsynaptic inhibition is primarily responsible for the suppression of hypoglossal motoneuron activity during REM sleep.

Support: 5R01NS094062

0067

AN ANATOMIC SUBSTRATE FOR GABAERGIC PROCESSES TO SUPPRESS ACTIVE SLEEP AND PROMOTE WAKEFULNESS IN THE NUCLEUS PONTIS ORALIS

Zhang, J.¹ Sampogna, S.¹ Xi, M.^{1,2} Fung, S. J.^{1,2} Tobin, C.^{1,2} Chase, M. H.^{1,2,3}

¹WebSciences International, Los Angeles, CA, ²VA Greater Los Angeles Healthcare System, Los Angeles, CA, ³UCLA School of Medicine, Los Angeles, CA.

Introduction: Our previous electrophysiologic data have provided compelling evidence that GABAergic processes in the nucleus pontis oralis (NPO) play a critical role in the generation and maintenance of wakefulness as well as active (REM) sleep (AS). We therefore hypothesized that one of the neuronal mechanisms of GABA actions in the NPO to promote wakefulness and suppress AS is due to a direct GABAergic inhibition of NPO neurons that generate AS (AS-generator neurons). However, the anatomical substrate for this inhibition is undetermined. Accordingly, the present study was undertaken to examine whether there is any direct interaction between GABAergic neurons and glutamatergic AS-generator neurons in the NPO.

Methods: Adult cats were deeply anesthetized and perfused transcardially. The brainstem containing the NPO was removed, postfixed and cut into 15 μ m coronal sections with a Reichert-Jung cryostat. The sections were incubated with a mixture of a rabbit polyclonal antibodies against glutamine and GABA following the procedure of double fluorescence immunohistochemistry.

Results: There was a large number of neuronal somata labeled by anti-glutamine antibody and terminals labeled by anti-GABA antibody in the NPO. These glutamine-positive neurons were medium to large, multipolar cells (> 20μ m), which resemble glutamatergic, AS-generator neurons that have been previously identified in the NPO. Specifically, majority of glutamatergic neuronal somata were closely apposed by multiple GABAergic terminals, indicating that AS-generator neurons in the NPO receive direct GABAergic inputs.

Conclusion: The present results demonstrate that a direct connection exists between glutamatergic AS-generator neurons and GABAergic processes in the NPO. These data provide the anatomical evidence which supports our hypothesis that the pontine GABAergic control of wakefulness and active sleep is partially mediated via GABAergic processes project to NPO AS-generator neurons that suppress the activity of these cells. **Support:** NS092383

0068

CIRCADIAN ALIGNMENT PREDICTS NEURAL RESPONSE TO MONETARY REWARD IN LATE ADOLESCENT DRINKERS

Hasler, B. P. Soehner, A. M. Ngari, W. Clark, D. B. University of Pittsburgh School of Medicine, Pittsburgh, PA.

Introduction: Abundant evidence from animal models implicates the circadian system in modulating the brain's reward circuitry, but evidence in humans has been more limited. In particular, published evidence has relied on self-report and/or behavioral proxies of circadian misalignment and cross-sectional designs. Here, we employed objective measures and a prospective design to assess

whether circadian alignment predicts the neural response to reward in a sample of late adolescent drinkers.

Methods: Participants included 23 late adolescents (18–22 y/o; 14 females) reporting weekly alcohol use. Participants completed pre-weekend (Thursday) circadian phase assessments via the dim light melatonin onset (DLMO). Sleep-wake timing was assessed via wrist actigraphy (midpoint of sleep on Tuesday and Wednesday prior to DLMO assessment). Circadian alignment was operationalized as the DLMO-midsleep interval. Neural response to reward was assessed via a card-guessing monetary reward fMRI task; analyzed on reward anticipation and reward win relative to neutral conditions. Mean BOLD signal was extracted from two regions-of-interest (striatum and medial prefrontal cortex, mPFC) for analyses in regression models, accounting for sex and scan order (participants also completed scans on Monday in counterbalanced order, not reported here).

Results: Shorter DLMO-midsleep intervals (i.e., greater misalignment) predicted lower striatal response to anticipated reward (beta=0.48,p=0.02) and showed a trend towards predicting lower mPFC response to anticipated reward (beta=0.39,p=0.06). No statistically-significant effects were found for reward win. Notably, a lower striatal response to anticipated reward correlated with more binge-drinking episodes in the past 30 days, but was not associated with alcohol use in the weekend immediately following the scan.

Conclusion: Our findings provide preliminary evidence of proximal associations between objectively-determined circadian alignment and the neural response to anticipated monetary reward. Ongoing work in a larger sample of adolescents aims to replicate this finding and more definitively determine its relevance to adolescent drinking.

Support: This work was supported by the National Institute on Alcohol Abuse and Alcoholism (R21 AA023209; R01 AA025626).

0069

SLEEP IN DROSOPHILA IS REGULATED BY THE CHROMATIN REMODELING FACTOR ISWI

Gong, N. N. Dilley, L. C. Moscato, E. H. Williams, C. E. Kayser, M. S.

University of Pennsylvania, Philadelphia, PA.

Introduction: Sleep is commonly disrupted in patients with neurodevelopmental disorders (NDDs). Despite strong clinical associations between disrupted sleep and other NDD symptoms, we lack an understanding of how these are pathophysiologically related. *Drosophila melanogaster* exhibit essential characteristics of human sleep and have well-defined neural circuits underlying learning and social behaviors. This represents an ideal system to investigate the mechanistic interaction between disrupted sleep and other behavioral dysfunctions in NDDs.

Methods: We performed a reverse genetic RNAi-based screen targeting *Drosophila* homologs of human genes within NDD-associated risk loci. Pan-neuronal knockdown of risk genes was achieved using the Gal4-UAS system.

Results: Pan-neuronal knockdown of ISWI led to dramatic deficits in sleep and circadian arrhythmicity in the adult fly. Across species, ISWI and its homologs are ATP-dependent chromatin remodelers that regulate gene expression important for neural differentiation. We found that depleting ISWI also leads to memory and social deficits. ISWI functions during dissociable temporal windows of pre-adult development and in different circuits to establish different adult behaviors. The sleep phenotype associated with ISWI knockdown mapped to a specific population of cells.

RNA-Seq of developing brains during the window important for sleep deficits revealed significant transcriptional changes in genes associated with nervous system development, suggesting ISWI acts in the development of sleep regulatory circuits. Finally, mutations in the human homologs of ISWI, SMARCA1/5, have been implicated in NDDs. Expressing either SMARCA1/5 in the setting of ISWI knockdown differentially rescued adult deficits.

Conclusion: Identification of ISWI provides a platform for unraveling pleiotropic behavioral effects from an NDD risk gene. Sleep, circadian rhythms, memory, and social behaviors are affected by ISWI knockdown, and map to different developmental periods and circuits. In addition, SMARCA1/5 differentially rescue adult behaviors, suggesting NDD-causing mutations in these genes may affect different behaviors. Current work aims to determine how human mutations in SMARCA1/5 affect behaviors.

Support: This work was supported by NIH K08 NS090461 (MSK) and T32 HL007953 (NNG), Hearst Foundation Fellowship 2018 (NNG), Burroughs Welcome Career Award for Medical Scientists, March of Dimes Basil O'Connor Scholar Award, and Sloan Research Fellowship (MSK).

0070

THE EFFECTS OF ACUTE BLUE WAVELENGTH LIGHT EXPOSURE ON FUNCTIONAL BRAIN CONNECTIVITY AND MOOD

Alkozei, A.¹ Dailey, N. S.¹ Bajaj, S.¹ Vanuk, J. R.¹ Raikes, A. C.¹ Grandner, M. A.² Killgore, W. D.²

¹University of Arizona, Tucson, AZ, ²University of Arizona, University of Arizona, AZ.

Introduction: Blue wavelength light is an effective treatment for delayed sleep phase syndrome, seasonal affective disorder and bipolar depression. The role of blue light in regulating melatonin production has been extensively studied, but other potential neurophysiological effects remain poorly understood. Some studies have suggested that daily blue light exposure may modulate functional brain responses within the amygdala and prefrontal cortex (PFC), potentially explaining blue light's antidepressant effect. In this study we investigated the effects of a single 30-minute session of blue light exposure on functional resting state connectivity between the amygdala and PFC.

Methods: Twenty-nine healthy 18-32 year olds were randomly assigned to either receive 30 minutes of blue (n=17) or non-blue (amber) light (n=12) exposure followed by a 7-minute resting state scan. Pre- and post light exposure, participants completed the Positive and Negative Affect Scale, as a measure of state affect.

Results: Individuals who received blue versus amber light showed greater positive connectivity between the right amygdala and the left dorsolateral prefrontal cortex (DLPFC) (x=-24, y=46, z=18, k=90, volume p-FDR corrected, p<0.001). Increased amygdala-DLFC connectivity correlated with greater decreases in negative mood for the blue (ρ =-.55, p=0.03), but not the amber group. Using Granger Causality, we found that the directionality of information flow between these two areas was bidirectional (p < 0.0025). Conclusion: Blue light exposure appears to facilitate greater information flow between the amygdala and the DLPFC at rest, potentially enhancing cognitive processes that regulate arousal and mood. As blue light exposure has been shown to enhance attention and learning, using blue light exposure during practice of emotional regulation strategies, such as reappraisal, may further increase the beneficial effects of blue light on mood. In order to use blue light exposure in a more targeted manner for sleep and mood disorders, further research into the underlying neurophysiological mechanisms is needed.

Support: This research was supported by a USAMRAA grant to WDSK (W81XWH-14-1-0571) as well as by an Arizona Health Education Centers (AHEC) Research Grant to AA.

0071

A MEDULLARY CIRCUIT CONTROLLING REM SLEEP

Schott, A. Baik, J. Chung, S. Weber, F. University of Pennsylvania, Philadelphia, PA.

Introduction: Rapid eye movement (REM) sleep is a distinct brain state known for its association with vivid dreaming in humans, though it is also crucial for other mental processes such as memory consolidation and emotion regulation. REM sleep is punctuated by phasic neurophysiological events known as pontine (P)-waves, which are thought to contribute to the cognitive functions of REM sleep. However, little is known about the neural circuits regulating these P-waves, or those responsible for initiating REM sleep itself. Here, we show that a yet unstudied population of medullary neurons expressing corticotropin-releasing-hormone (CRH) are important for controlling both the induction of REM sleep and its phasic events.

Methods: To measure the endogenous activity of CRH+ neurons in the dorsomedial medulla (dmM), we injected the calcium indicator GCaMP6 in the dmM of CRH-Cre mice. To optogenetically manipulate dmM CRH+ neuron activity, we delivered either an excitatory (ChR2) or inhibitory (iC++) opsin to the dmM of CRH-Cre mice. To record P-waves, we implanted microelectrodes to record local field potentials in the subcoeruleus region of the pons. Results: Fiber photometry recordings showed that dmM CRH+ neurons are selectively active during REM sleep, and optogenetic stimulation and inhibition of this population is sufficient to promote and reduce REM sleep, respectively. Additionally, dmM CRH+ neuron activity is correlated with P-waves in the pons, and optogenetic activation of dmM CRH+ cells reliably triggers P-waves during REM sleep. Finally, histological examination of fluorescently labeled dmM CRH+ axons revealed strong projections to several pontine areas involved in P-wave generation as well as modulation of the theta rhythm during REM sleep.

Conclusion: Our results suggest that dmM CRH+ neurons are involved in controlling REM sleep initiation as well as phasic events within REM sleep. These neurons thus constitute an important component of the brainstem circuitry regulating REM sleep. **Support:** National Institutes of Health (R01 HL149133)

0072

PRECLINICAL CHARACTERIZATION OF SUVN-G3031, A HISTAMINE H3 RECEPTOR INVERSE AGONIST FOR THE TREATMENT OF NARCOLEPSY

Jayarajan, P. Subramanian, R. Kamuju, V. Muddana, N. Palacharla, R. Mekala, V. Abraham, R. Reballi, V. Achanta, P. Nirogi, R.

Suven Life Sciences, Hyderabad, INDIA.

Introduction: Narcolepsy is a chronic sleep disorder characterized by overwhelming daytime drowsiness, sudden attacks of sleep and sometimes accompanied by cataplexy. Although the orexin deficiency is considered to be the primary cause of this disorder, lot of attention has been focused recently on targeting histaminergic neurotransmission by blockade of histamine H3 receptor (H3R). SUVN-G3031 is one of the potent and selective H3R inverse agonist currently being evaluated in a Phase 2 study as monotherapy for the treatment of narcolepsy with and without cataplexy (ClinicalTrials.gov Identifier: NCT04072380).

Methods: Binding of SUVN-G3031 in human and rat histamine H3R was evaluated in *in vitro* radioligand binding assay and functionality was assessed in GTP γ S assay. Pharmacokinetic properties were evaluated after oral administration in rat and dog. Neurotransmitters like histamine, dopamine and norepinephrine were estimated in rat cortex using microdialysis.

Results: SUVN-G3031 is an inverse agonist at histamine H3R with hKi of 8.7 nM and showed minimal binding against over 70 target sites. SUVN-G3031 exhibited desired pharmacokinetic properties in rat and dog with excellent brain penetration in rats. SUVN-G3031 produced significant increase in histamine, dopamine and norepinephrine levels in cortex. SUVN-G3031 produced no change in the striatal and accumbal dopamine levels in rats, suggesting no propensity to induce abuse liability. SUVN-G3031 blocked R- α -methylhistamine induced water intake and produced dose dependent increase in *tele*-methylhistamine levels in various brain regions and in cerebrospinal fluid of male Wistar rats.

Conclusion: SUVN-G3031 is an inverse agonist at histamine H3 receptor and results from the preclinical studies presented here provide a strong evidence for the potential utility of SUVN-G3031 in the treatment of narcolepsy with and without cataplexy. **Support:** None

0073

LEFT ANTERIOR CINGULATE HYPERAROUSAL DURING SLEEP ANXIETY-INDUCING EMOTIONAL TASKS PERFORMANCE IN PATIENTS WITH INSOMNIA DISORDER

Kang, S.¹ Ma, H.¹ Cho, S.¹ Kang, J.¹ Kim, N.²

¹Department of Psychiatry, Gil Medical Center, Gachon University College of Medicine, Incheon, KOREA, REPUBLIC OF, ²Neuroscience Research Institute, Gachon University, Incheon, KOREA, REPUBLIC OF.

Introduction: Patients with insomnia frequently experience sleep/ insomnia-related anxiety; this anxiety has been associated with hyperarousal. We investigated the underlying brain function changes in patients with insomnia during emotional task performance that induced sleep/insomnia-related anxiety.

Methods: Functional magnetic resonance imaging (fMRI) was performed during emotional task performance in healthy individuals and patients with insomnia who met the diagnostic criteria of insomnia disorder based on the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, and had chronic insomnia for more than 6 months. The participants underwent fMRI scanning during three types of emotional task performance—insomnia-anxiety task, reading sentences that cause insomnia-related anxiety; general-anxiety task, reading sentences that cause anxiety for everyone; and neutral task, reading neutral sentences that do not cause emotional anxiety. The images obtained from fMRI and blood oxygen level-dependent (BOLD) signal changes were compared between patients with insomnia and healthy controls. Interim analysis was performed with the data of 13 patients with insomnia and 9 controls.

Results: The brain activity in the left anterior cingulate was higher during insomnia-anxiety task performance than that during general-anxiety task performance in the insomnia group (voxel-wise uncorrected p < 0.05; cluster size, 100). In the insomnia group, the brain activity during insomnia-anxiety task performance was

not lower in any brain area than that during general-anxiety task performance.

Conclusion: We show that patients with chronic insomnia experience sleep anxiety related with hyperarousal in the left anterior cingulate area. Additional subject recruitment and re-analysis are needed to confirm the findings of this interim analysis.

Support: This research was supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education (NRF-2017R1D1A1B03032431).

0074

INHIBITORY NEURONS IN THE DORSOMEDIAL MEDULLA PROMOTE REM SLEEP

Stucynski, J. Schott, A. Baik, J. Hong, J. Weber, F. Chung, S. University of Pennsylvania, Philadelphia, PA.

Introduction: The neural circuits controlling rapid eye movement (REM) sleep, and in particular the role of the medulla in regulating this brain state, remains an active area of study. Previous electrophysiological recordings in the dorsomedial medulla (DM) and electrical stimulation experiments suggested an important role of this area in the control of REM sleep. However the identity of the involved neurons and their precise role in REM sleep regulation are still unclear.

Methods: The properties of DM GAD2 neurons in mice were investigated through stereotaxic injection of CRE-dependent viruses in conjunction with implantation of electrodes for electroencephalogram (EEG) and electromyogram (EMG) recordings and optic fibers. Experiments included in vivo calcium imaging (fiber photometry) across sleep and wake states, optogenetic stimulation of cell bodies, chemogenetic excitation and suppression (DREADDs), and connectivity mapping using viral tracing and optogenetics.

Results: Imaging the calcium activity of DM GAD2 neurons in vivo indicates that these neurons are most active during REM sleep. Optogenetic stimulation of DM GAD2 neurons reliably triggered transitions into REM sleep from NREM sleep. Consistent with this, chemogenetic activation of DM GAD2 neurons increased the amount of REM sleep while inhibition suppressed its occurrence and enhanced NREM sleep. Anatomical tracing revealed that DM GAD2 neurons project to several areas involved in sleep / wake regulation including the wake-promoting locus coeruleus (LC) and the REM sleep-suppressing ventrolateral periaquaductal gray (vIPAG). Optogenetic activation of axonal projections from DM to LC, and DM to vIPAG was sufficient to induce REM sleep.

Conclusion: These experiments demonstrate that DM inhibitory neurons expressing GAD2 powerfully promote initiation of REM sleep in mice. These findings further characterize the dorsomedial medulla as a critical structure involved in REM sleep regulation and inform future investigations of the REM sleep circuitry. **Support:** R01 HL149133

0075

NEURAL CORRELATES OF COGNITIVE FATIGUE IN PARKINSON DISEASE

Liu, W.¹ Bhavsar, R.¹ Mamikonyan, E.² Yang, F. N.¹ Lei, H.¹ Weintraub, D.² Detre, J. A.¹ Rao, H.^{1,3}

¹Center for Functional Neuroimaging, Department of Neurology, University of Pennsylvania, PHILADELPHIA, PA, ²Department of Psychiatry, University of Pennsylvania, PHILADELPHIA, PA, ³Department of Psychiatry, University of Pennsylvania, Philadelphia, PA. **Introduction:** Parkinson's disease (PD) is a common neurodegenerative disease affecting millions of people world-wide. Fatigue is a prevalent and debilitating non-motor symptom in PD. However, the neural correlates underlying cognitive fatigue are poorly understood. Our previous studies suggested that continuous performance of a simple but mentally demanding psychomotor vigilance task (PVT) induced cognitive fatigue, operationalized as subjective exhaustion and time-on-task performance decline. Here we used arterial spin labeling (ASL) perfusion fMRI to investigate regional cerebral blood flow (CBF) changes in PD patients during cognitive fatigue induced by continuous performance of 20-min PVT.

Methods: Twenty-one PD patients completed a 20-min PVT during the ASL scan and two additional 4-min resting-state ASL scans before and after PVT. Reaction times (RTs) and regional CBF changes throughout the PVT as well as during pre- and post-task resting baselines were measured. Cognitive fatigue was analyzed by dividing the entire PVT performance into five quintiles in addition to the immediate measurement of self-rated fatigue before and after PVT.

Results: PD patients demonstrated significantly increased selfreported fatigue ratings after the task (p < 0.05) and progressively slower RTs across quintiles (p < 0.05). Perfusion data showed that the PVT activates the right middle frontal cortex, right inferior parietal lobe, right insula, bilateral occipital cortex, and right cerebellum (FDR corrected). Moreover, the bilateral middle frontal gyri were less active during the post-task rest compared to the pretask rest.

Conclusion: These results demonstrated that cognitive fatigue has an ongoing effect on brain activity after a period of continuous mental effort and supported the critical role of prefrontal cortex in mediating cognitive fatigue in PD. The findings also suggest the utility of continuous PVT as an appropriate paradigm to induce and examine cognitive fatigue in PD.

Support: Supported in part by Parkinson's Foundation Translational Research Grant and NIH grants R01-MH107571, R21-AG051981, and P30-NS045839.

0076

THE ROLE OF PREOPTIC AREA GABAERGIC AXONAL PROJECTIONS TO TUBEROMAMMILLARY NUCLEUS IN SLEEP HOMEOSTASIS

Maurer, J. Covarrubias, I. Baik, J. Weber, F. Chung, S. Department of Neuroscience, University of Pennsylvania, Philadelphia, PA.

Introduction: Sleep deprivation has profound widespread physiological effects including cognitive impairment, compromised immune system function and increased risk of cardiovascular disease. The preoptic area (POA) of the hypothalamus contains sleepactive GABAergic neurons that respond to sleep homeostasis. We have shown that activation of POA GABAergic axons innervating the tuberomammillary nucleus (TMN, GABAergic^{POA ->TMN}) are critical for sleep regulation but it is unknown if these projections modulate sleep homeostasis.

Methods: To monitor *in vivo* neural activity of GABAergic^{POA} ->TMN projection neurons during sleep deprivation and rebound, fiber photometry was used. GAD2-Cre mice (*n*=6) were injected with AAV-DIO-GCaMP6S into the POA and an optic fiber was implanted into the TMN. An electroencephalogram (EEG) and electromyography (EMG) implant was mounted upon the skull to identify brain states. Calcium activity was measured for six hours

ness and minimize the stress to the animal. **Results:** During baseline sleep recordings, GABAergic^{POA ->TMN} projection neurons are most active during sleep (NREM and REM) which is maintained until wake onset. As sleep pressure increases, GABAergic^{POA ->TMN} projection neurons display gradual increase in neural activity compared to time-matched points during baseline sleep recordings. Once mice were permitted to enter sleep rebound, GABAergic^{POA ->TMN} projection neurons gradually displayed decreased activity as sleep pressure eased.

Conclusion: GABAergic^{POA ->TMN} projection neurons show a strong increase in activity to drive homeostatic sleep need during periods of increased sleep pressure but subside once this pressure is reduced.

Support: This work is supported by NIH grant R01-NS-110865.

0077

OBJECTIVE SLEEP AND NEURAL RESPONSE TO THERMAL PAIN TESTING FOLLOWING COGNITIVE BEHAVIORAL TREATMENT IN PATIENTS WITH COMORBID INSOMNIA AND FIBROMYALGIA: A PILOT STUDY

McCrae, C.¹ Craggs, J.¹ Curtis, A.¹ Staud, R.² Berry, R.² Robinson, M.²

¹University of Missouri, Columbia, MO, ²University of Florida, Gainesville, FL.

Introduction: Fibromyalgia (FM) is characterized by high rates of insomnia and abnormal central pain processing/heightened response to stimuli (i.e., central sensitization). This study examines whether cognitive behavioral treatments (CBTs) that target insomnia and pain improve central pain processing [indicated by decreased response to quantitative sensory testing (QST) using thermal stimuli] in patients with fibromyalgia and insomnia.

Methods: Before and after CBT-I, CBT-P or waitlist, adults (N=32, M_{age} =55.9, SD=12.2) with FM and insomnia completed QST during *f*MRI (Phillips Achieva 3T scanner), 14-daily pain ratings [least(0)-most(100) intense pain imaginable] and 1-night in-home polysomnography (AURA/Grass Technologies). Imaging data were processed using Brain Voyager (Brain Innovation/Netherlands). Random effects ANCOVA identified regions with significant group (3-CBT-I, CBT-P, waitlist) by time (baseline, post-treatment) interactions in brain hemodynamic response to QST. Linear regressions (using residualized change scores) were conducted for each significant region to examine how pain and sleep changes (%Stages 1–3 NREM, %REM) were related to brain response changes.

Results: Eleven regions exhibited significant interactions (ps<.00; large effects; right hemisphere: inferior frontal, superior temporal, mid-occipital, and cingulate gyri, lentiform nucleus; left hemisphere: angular, superior temporal, mid-frontal, inferior occipital, mid-temporal, and inferior frontal gyri). CBT-I decreased brain response to QST in 8 regions and CBT-P in 3 regions (CBT-I effects>CBT-P). Waitlist increased response in 6 regions. Pain ratings, %Stage 2 and %REM sleep were not significant for any region and were dropped from the models. Increased %Stage 1 and/ or %Stage 3 predicted decreased brain response to QST in 8 of the 11 regions (ps<.01), accounting for 19–45% of the variance.

Conclusion: Compared to CBT-P, CBT-I prompted greater improvement in abnormal pain processing in patients with fibromyalgia and insomnia. Increased NREM sleep may underlie these pain processing improvements following treatment. Future research examining the potential role of NREM sleep in central sensitization and pain processing is warranted.

Support: National Institute of Arthritis and Musculoskeletal and Skin Diseases (R01AR055160 and R01AR005160-S1; McCrae, PI). Data collected as part of clinical trial NCT02001077 Sleep and Pain Interventions (SPIN) at the University of Florida (McCrae, PI).

0078

INFLUENCE OF LIGHT ON BRAIN ACTIVITY UPON WAKING FROM SLOW WAVE SLEEP

Flynn-Evans, E. E.¹ Hilditch, C. J.² Chachad, R.² Bansal, K.³ Wong, L. R.² Santamaria, A.⁴ Bathurst, N. G.² Feick, N. H.² Garcia, J.³

¹Fatigue Countermeasures Lab, NASA Ames Research Center, Moffett Field, CA, ²Fatigue Countermeasures Lab, SJSU Research Foundation, Moffett Field, CA, ³US CCDC Army, Moffett Field, CA, ⁴University of South Australia, Adelaide, AUSTRALIA.

Introduction: Waking from sleep is associated with reduced alertness due to sleep inertia. Light acutely improves alertness during sleep deprivation. In this study we assessed the influence of light on brain activity and connectivity after waking from slow wave sleep (SWS).

Methods: Twelve participants kept an actigraphy-confirmed stable sleep schedule with 8.5 hours for five nights and five hours for one night prior to an overnight laboratory visit. Participants completed two three-minute Karolinska Drowsiness Tests (KDT) before going to bed at their habitual bedtime. They were monitored continuously using high-density EEG (32-channel; Brain Products GmbH). Participants were woken twice and exposed to red light (0.01 melanopic-lux; control) or blue-enriched light (63.62 melanopic-lux) for one hour, in a randomized order, following at least five minutes of SWS. EEG artifact were removed algorithmically and the spectral composition of each electrode (i.e., fast fourier transform, FFT) and effective connectivity (i.e., partial directed coherence, PDC) between each electrode were estimated. A graphical analysis was conducted to extract features relevant to the facilitation of efficient communication between electrodes. All data were averaged within frequency bins of interest that correspond to delta (1-3Hz), theta (4-7Hz), alpha (8-12Hz), and beta (13-25Hz) bands and expressed relative to the pre-sleep baseline.

Results: Compared to the pre-sleep baseline, participants exposed to blue-enriched light experienced reduced theta and alpha activity; however, these results were not significantly different from the control. In contrast, the communication of frontal electrodes significantly increased across all frequency bands compared to the control, and this effect was most prominent in the alpha (t(11)=3.80, p=.005) and beta bands (t(11)=3.92, p=.004).

Conclusion: Exposure to blue-enriched light immediately after waking from SWS may accelerate the process of waking and help to improve alertness by facilitating communication between brain regions. Future analyses will explore the temporal persistence and granularity of the communicative properties associated with this response.

Support: Naval Postgraduate School Grant. NASA Airspace Operations and Safety Program, System-Wide Safety Project.

BLUE LIGHT EXPOSURE ENHANCES NEURAL EFFICIENCY OF THE TASK POSITIVE NETWORK DURING A COGNITIVE INTERFERENCE TASK

Killgore, W. D.¹ Dailey, N. S.¹ Raikes, A. C.¹ Vanuk, J. R.¹

Taylor, E.¹ Grandner, M. A.¹ Alkozei, A.¹

¹University of Arizona, Tucson, AZ, ²University of Arizona, Tucson, AZ.

Introduction: Light exposure has powerful effects on the circadian timing of sleep and wake, primarily through the regulation of the secretion of melatonin. However, it is becoming clear that light has additional alerting effects beyond its primary effect on the circadian system. Exposure to light, particularly blue-wavelength light, has been shown to acutely increase brain activation, alertness, and some elementary aspects of cognitive performance such as working memory and emotional anticipation during the day. Whether blue light exposure can have longer-lasting effects on brain activation and performance during more complex cognitive control tasks up to 30-minutes after light cessation is unknown.

Methods: In a sample of 30 healthy adults, we examined the effects of a single 30-minute exposure to either blue (n=14) or amber placebo (n=16) light on subsequent brain activation and performance during the Multi-Source Interference Task (MSIT) measured a half-hour after light cessation using functional magnetic resonance imaging. Mean activation in all regions showing increased task-related activation (i.e., Task Positive Network; TPN) and regions showing decreased activation (i.e., Default Mode Network; DMN) at p<.001 (FWE corrected) was extracted separately for each network in SPM12 and compared between light conditions.

Results: Performance metrics for the MSIT, including accuracy, response time, and cognitive throughput, did not differ between the blue and amber conditions, suggesting that performance was sustained equally between light conditions. However, brain activation within the TPN to the interference condition of the MSIT was significantly lower (p=.024) in the blue relative to the amber condition, with no group differences observed for suppression of the DMN.

Conclusion: Compared to amber, a single exposure to blue light was associated with enhanced neural efficiency a half-hour later as demonstrated by reduced TPN activation to achieve the same level of cognitive performance. Blue light may be an effective method for optimizing neurocognitive performance under some conditions. **Support:** US Army Medical Research and Materiel Command: W81XWH-14-1-0571

0080

DAYTIME SLEEPINESS CORRELATES WITH INCREASED GRAY MATTER VOLUME IN THE RIGHT MIDDLE TEMPORAL GYRUS IN HEALTHY YOUNG INDIVIDUALS

Burns, A. I. Bullock, A. Raikes, A. C. Dailey, N. S. Grandner, M. A. Killgore, W. D.

University of Arizona, Tucson, AZ.

Introduction: Daytime sleepiness has been associated with some neuroimaging metrics, including altered functional connectivity within the default mode network and decreased gray matter volume (GMV) of the medial prefrontal cortex. Most prior studies, however, have focused on patients with sleep disorders or other pathologies. Here we examined the association between GMV and

self-reported daytime sleepiness among a healthy group of young adults who reported no sleep-related problems.

Methods: Forty-five healthy adults (22 female; Mean Age=25.4, SD=5.6), who self-reported no history of sleep-related disorders or major medical conditions, completed the Epworth Sleepiness Scale (ESS), the Repeatable Battery for Neuropsychological Status (RBANS) and underwent high-resolution structural neuroimaging at 3T. Gray matter volumes were processed using standard procedures in SPM12. After controlling for age, sex, and intracranial volume, GMV was regressed against ESS scores.

Results: Greater ESS was associated with larger GMV within a cluster of voxels in the right middle temporal gyrus (MNI coord-inates: 57, -9, -22; k=1344 voxels, p=.003, FWE cluster corrected). After controlling for ESS scores, larger GMV in this region was associated with poorer delayed memory performance (r=-.345, p=.022) and total neurocognitive performance on the RBANS (r=-.303, p=.046).

Conclusion: Greater daytime sleepiness in healthy normal sleepers was associated with greater GMV within a region of the right middle temporal gyrus. Greater volume of this region was also associated with poorer neuropsychological performance. Decreased GMV of this same region has previously been reported in patients with obstructive sleep apnea and insomnia, suggesting that it may be particularly sensitive to sleep disruption or may play a role in the etiology of sleep disorders, even among young individuals who deny any history of sleep-related dysfunction. Longitudinal work should focus on the potential of this region as a biomarker of vulnerability to sleep problems. **Support:**

0081

HABITUAL SLEEP DURATION IS NEGATIVELY CORRELATED WITH EMOTIONAL REACTIVITY WITHIN THE ROSTRAL ANTERIOR CINGULATE CORTEX IN INDIVIDUALS WITH PTSD

King, R. Jecmen, D. Mitchell, J. Ralston, K. Gould, J. Burns, A. Bullock, A. Grandner, M. A. Alkozei, A. Killgore, W. D. University of Arizona, Tucson, AZ.

Introduction: Sleep difficulties, such as insomnia, are highly prevalent in individuals with Post-Traumatic Stress Disorder (PTSD). However, sleep deprivation can also increase emotional reactivity to positive (as well as negative) stimuli. While the effects of sleep loss on emotional perception healthy individuals has been documented, it remains unclear how lack of sleep in individuals with PTSD may affect their emotional reactivity to positive stimuli. We hypothesized that lower habitual sleep duration would be associated with greater functional brain activation changes in response to subliminally presented happy faces in brain areas of the reward network, such as the rostral anterior cingulate cortex (rACC).

Methods: Thirty-nine individuals with DSM-5 confirmed PTSD were administered the Pittsburgh Sleep Quality Index (PSQI) as a measure of their average nightly sleep duration over the past month. Participants then underwent fMRI imagining while viewing subliminal presentations of faces displaying happiness, using a backward masked facial affect paradigm to minimize conscious awareness of the expressed emotion. Brain activation to masked happy expressions was regressed against sleep duration in SPM12. **Results:** There was a negative correlation between habitual sleep duration and activation within the rACC in response to the masked happy faces ($x=14, y=40, z=0; k=102, p_{FWF-corr} = 0.008$).

Conclusion: Individuals with PTSD who average less sleep at night showed greater emotional reactivity, as indexed by greater functional brain activation changes within an area of the reward network, than individuals who obtained more sleep per night. Future research involving actual sleep duration manipulation will be necessary to determine whether this finding reflects the well-known antidepressant effect of sleep deprivation or a form of greater emotional expression error monitoring among traumatized patients when lacking sleep. Regardless, these findings suggest that insufficient sleep could affect unconsciously perceived emotion in faces and potentially affect social and emotional responses among individuals with PTSD.

Support: US Army Medical Research and Materiel Command: W81XWH-14-1-0570

0082

HIPPOCAMPAL GRAY MATTER VOLUME IN HEALTHY ADULT POPULATION IS ASSOCIATED WITH HABITUAL SLEEP DURATION

King, R. Jecmen, D. Alkozei, A. Raikes, A. C. Grandner, M. A. Killgore, W. D.

University of Arizona, Tucson, AZ.

Introduction: The hippocampus is well known for its role in sleep and memory consolidation in adolescents, and has been shown to demonstrate neural plasticity and neuronal regeneration. However, the relationship between sleep and hippocampal gray matter volume in healthy adults remains to be fully characterized. We hypothesized that total sleep time (TST), as measured by actigraphy, would correlate positively with gray matter volume (GMV) in the hippocampus, a key memory region of the brain.

Methods: Forty-five healthy normal sleeping adults between 20–45 years of age wore an actigraph for seven days to quantify habitual sleep duration and underwent magnetic resonance imaging during the actigraphy period. Voxel based morphometry in SPM12 was used to estimate GMV at the whole brain level. A region-of-interest mask was used to constrain data analysis to the left and right hippocampi.

Results: Habitual sleep duration per night correlated positively with gray matter volume within part of the left hippocampus (x=-36,y=-20,z=-18; k=32, pFWE-corr=0.093), controlling for age, sex, total intracranial volume, intelligence scores and mood. No correlation was found between TST and hippocampal GMV in the right hippocampus.

Conclusion: Longer sleep time was associated with greater gray matter volume in the left hippocampus. This finding is consistent with what has been observed in healthy children and extend these findings to healthy normal sleeping adults. While TST and GMV are correlated, the causal association cannot be established here. Further research may explore the effects of sleep extension on GMV and how these volume differences associate with various aspects of cognition, particularly memory. It should be noted that this study only included healthy adults with sleep durations between 6–9 hours per night. Future studies would benefit from including adults with a greater variance in their sleep patterns to better understand the relationship between sleep and hippocampal volume, and its potential effects on memory performance.

Support: Defense Advanced Research Projects Agency Young Faculty Award: DARPA-12-12-11-YFA11-FP-029

SLOW WAVE ACTIVITY DURING SLEEP IS ASSOCIATED WITH LONGITUDINAL CHANGE IN OVERNIGHT MEMORY RECALL

Buck, C.¹ Parker Fong, K.² Linkovski, O.² Kawai, M.² O'Hara, R.² ¹Stanford School of Medicine, Palo Alto, CA, ²Stanford University School of Medicine, Palo Alto, CA.

Introduction: Duration of slow wave sleep (SWS) declines with age and may not be the most sensitive biomarker for memory in older adults. Analyzing the spectral power of slow wave activity (SWA) (0.5–4 Hz) may provide a more sensitive measure to capture the impact of sleep on memory. We investigated the association of SWA at baseline with the change of overnight memory recall over one year in older adults.

Methods: Participants were 42 community-dwelling healthy older adults (22 women and 20 men). We performed a polysomnography (PSG) and list-learning memory tests at baseline (T1) and after a one-year follow-up (T2). The procedure includes, 1) the participants memorized a 16 word list in the evening, 2) after a 5 minute delay, participants wrote down as many words as they could remember (evening recall), 3) overnight PSG was then performed, and 4) the following morning, participants wrote down as many words from the original list from the night before (overnight memory recall). This procedure was repeated at T2. Mixed modeling was utilized to analyze the association between baseline SWA and trajectory of overnight memory recall.

Results: For the SWA component, higher relative power of slow oscillation (0.5-1 Hz) during the first ultradian cycle at baseline was correlated with a greater decline in overnight memory recall after 1 year (t = -2.198, p = .034), which covaried for age and gender. There was no correlation with evening recall. Relative power of delta (1-4 Hz) range activity did not show an association with evening and overnight memory recall.

Conclusion: Higher relative power of slow oscillation at baseline predicts a greater decline of overnight memory recall. This may indicate a differential effect among the frequency ranges of SWA on longitudinal change in memory in older adults.

Support: This work was supported by National Institute of Health grants MH 070886, AG 18784 and AG17824 and the Office of Academic Affiliations, Advanced Fellowship Program in Mental Illness Research and Treatment, Department of Veterans Affairs.

0084

SLEEP CONSOLIDATES INCIDENTALLY ENCODED INFORMATION AFTER DEEP BUT NOT SHALLOW ENCODING

Wernette, E. M. Fenn, K. M. Michigan State University, East Lansing, MI.

Introduction: Slow wave sleep (SWS) strengthens declarative memory for information studied for a later test. However, research on the effect of sleep on information that is not intentionally remembered is scare. Previous research from our lab suggests sleep consolidates some, but not all, information that has been encoded incidentally, meaning that it has been acted on but not intentionally remembered. It remains unclear what determines which information benefits from sleep-dependent consolidation processes and what aspects of sleep are related to these mnemonic benefits. In two experiments, we test the hypothesis that sleep consolidates strong but not weak memory traces following incidental encoding, and

assess the relationship between memory performance and objective sleep characteristics.

Methods: In Experiment 1, participants rated words one (weak traces) or three times (strong traces) in a deep or shallow incidental encoding task. Participants either rated words on a scale from 'concrete' to 'abstract' (deep) or counted the vowels in the words (shallow). Following a 12-hour period containing sleep or wake-fulness, participants took a surprise memory test. In Experiment 2, participants rated words one or three times in the deep encoding task, received an 8-hour sleep opportunity with polysomnography, and took the surprise memory test.

Results: In Experiment 1, participants remembered words better after sleep than wake regardless of whether words were encoded one or three times, but only after deep encoding. Sleep did not consolidate information following shallow encoding. Experiment 2 is ongoing, but we predict that the amount of SWS will correlate positively with memory.

Conclusion: Results thus far suggest sleep may have consolidated information based on the strength of memory traces. Because deep encoding results in stronger memory traces than shallow encoding, this work is broadly consistent with theories of memory consolidation that predict sleep is more beneficial for strong memory traces than weak, such as the synaptic downscaling hypothesis. **Support:** N/A

0085

VULNERABILITY TO SLEEP RESTRICTION IS ASSOCIATED WITH DECREASED WORKING MEMORY PERFORMANCE

Mathew, G. M.¹ Strayer, S. M.¹ Ness, K.² Buxton, O. M.^{1,3,4} Chang, A.^{1,5}

¹Department of Biobehavioral Health, College of Health and Human Development, Pennsylvania State University, University Park, PA, ²Department of Medicine, Division of Metabolism, Endocrinology, and Nutrition, University of Washington, Seattle, WA, ³Division of Sleep Medicine, Harvard Medical School, Boston, MA, ⁴Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, Boston, MA, ⁵College of Nursing, Pennsylvania State University, University Park, PA.

Introduction: We investigated whether individuals with more lapses on the psychomotor vigilance task (PVT) after sleep restriction (SR) demonstrated poorer working memory compared to those with fewer PVT lapses.

Methods: Fifteen healthy men (22.3±2.8 years) participated in a 10-night inpatient protocol with three nights of 10-hour baseline time in bed (TIB), five nights of SR (5-hour TIB), then two recovery (10-hour TIB) nights. Participants completed the Visual Object Learning Task (VOLT) and Fractal 2-Back (F2B; visual n-back) measuring working memory and the PVT (Joggle Research® battery) approximately every two hours during wake. During the VOLT, participants indicated whether presented images had been shown previously. Outcomes included number of misses and false alarms. During the F2B, participants tapped the screen when an image appeared that had been shown 2 images previously. Outcomes included sensitivity and specificity. Median split of mean PVT lapses after the last night of SR was used to categorize participants into "vulnerable" (n=8) versus "resistant" (n=7) groups. Outcomes were analyzed in mixed models with the predictor day*vulnerability, excluding the first three baseline days to preclude practice effects.

Results: There was a significant interaction between day and attentional vulnerability for VOLT misses (p<.001); misses increased linearly across days in vulnerable (b=.18, p<.001) but not resistant (p=.956) participants. There was no interaction between day and vulnerability for VOLT false alarms, which did not change across days. There was a significant interaction between day and attentional vulnerability for F2B sensitivity (p=.002); sensitivity increased linearly across days in resistant (b=.02, p<.001) but not in vulnerable (p=.273) participants. There was no interaction between day and vulnerability for F2B specificity, which did not change across days.

Conclusion: Performance on the VOLT decreased in vulnerable participants only; performance on the F2B improved in resistant participants likely due to practice effects not seen in vulnerable participants. Findings indicate vulnerability to attentional lapses after SR is a marker of vulnerability to working memory decrements.

Support: This study was funded by grant UL1TR000127 from the Clinical and Translational Science Institute (Chang PI) and the College of Health and Human Development at the Pennsylvania State University.

0086

DAYTIME NAPPING AND MEMORY CONSOLIDATION OF NOVEL WORD LEARNING IN CHILDREN AND ADULTS

van Rijn, E. Walker, S. A. Knowland, V. C. Cairney, S. A.

Gouws, A. D. Gaskell, M. Henderson, L.

Department of Psychology, University of York, York, UNITED KINGDOM.

Introduction: Memory for novel words benefits from sleep, particularly non-rapid eye movement (NREM) sleep and its features, such as sleep spindles and slow oscillations. This is consistent with systems consolidation models, in which sleep supports transfer from hippocampal to neocortical memory networks. Larger amounts of slow wave sleep in children has been proposed to account for enhanced consolidation effects, but such studies have typically focused on nocturnal sleep. We examined whether daytime naps benefit word retention in adults and children aged 10–12 years, and whether this relationship in children is affected by differences in white matter pathway microstructure. We hypothesized that the link between memory consolidation and structural brain connectivity will be mediated by the degree of sleep spindles during the nap.

Methods: Adults (N = 31; mean age = 20.91, SD = 1.55) and children (N = 38; mean age = 11.95, SD = 0.88) learned spoken novel words, followed by a 90-minute nap opportunity monitored with polysomnography. Memory for the words was tested pre- and postnap. Children's structural brain connectivity was measured using diffusion tensor imaging (DTI).

Results: Word memory was preserved following sleep in adults, while an adult wake control condition showed deterioration. Similarly, in children memory performance was stable over the nap, with wake control data currently being collected. Analyses relating behavioral changes over the nap to NREM sleep features and structural brain connectivity will be presented.

Conclusion: In line with sleep-dependent memory consolidation models, daytime naps protect novel words from forgetting in adults and children. Examining potential relationships between nap-based consolidation and structural integrity has important theoretical implications, given the increase in brain connectivity in language areas during childhood, as well as white matter alterations in developmental populations. **Support:** This research was supported by the UK Economic and Social Research Council, grant no. ES/N009924/1.

0087

CHILDHOOD NAPS PROMOTE SHORT-TERM DESTABILIZATION BUT LONG-TERM CONSOLIDATION OF EMOTIONAL MEMORIES

Hanron, O. Mason, G. Holmes, J. F. Spencer, R. M. University of Massachusetts Amherst, Amherst, MA.

Introduction: Early childhood naps have been shown to support emotional memory consolidation, but this benefit only emerges the following day. It is unknown whether consolidation occurs during the nap itself, or if napping only prepares memories for overnight consolidation. In this study, we used a forced-choice recognition task to determine whether naps protect emotional memories against post-nap interference, which would indicate the occurrence of consolidation.

Methods: Preschool children (33–67 months; N=63) viewed neutral faces paired with negative or neutral descriptions. Following a nap or an equal interval awake (within-subjects, order counterbalanced, ~1 week apart), half of these participants (N=33) were presented with an interfering set of faces and descriptions, while the other half (N=30) did not receive interference. For all participants, recognition of the original faces was probed after encoding, after the nap or wake interval, and the next morning.

Results: To assess the influence of napping on changes in emotional memory, 2 (stimulus valence: negative vs. neutral) x 2 (condition: nap vs. wake) repeated-measures ANOVAs were performed. Recall of negative and neutral items did not immediately differ between the nap and wake conditions for the participants who received no interference. 24 hours later, these children trended towards recalling negative and neutral items better if they had napped the previous day (condition main effect: F(1,29)=3.539, p=0.070). In contrast, participants who received interference recalled fewer negative items than neutral items immediately following a nap (p=0.034), while this difference did not emerge following an interval awake.

Conclusion: Our results suggest that naps initially destabilize emotional memories rather than protecting them against interference. However, this initial destabilization may reflect the partial processing of memories during naps, perhaps allowing for enhanced long-term consolidation. Overall, our findings provide important insight into the mechanism of nap-dependent emotional processing.

Support: Supported by NIH R01 HL111695 and an Honors Research Grant from Commonwealth Honors College

0088

GRAVITY DREAMS FOLLOWING A VIRTUAL REALITY FLIGHT SIMULATION

*Picard-Deland, C.*¹ *Pastor, M.*² *Solomonova, E.*³ *Paquette, T.*² *Nielsen, T.*²

¹Université de Montréal, Neuroscience Department, Montreal, QC, CANADA, ²Dream & Nightmare Laboratory; Center for Advanced Research in Sleep Medicine, Montreal, QC, CANADA, ³McGill University; Department of Psychiatry, Montreal, QC, CANADA.

Introduction: Flying is a prevalent but infrequent experience in dreams. Despite a broad interest in such unique dream experiences, there is still no experimental procedure for reliably inducing them. Our study aimed 1) to induce flying dreams in the laboratory using

virtual reality (VR), 2) to examine phenomenological correlates of flying dreams, such as lucidity and emotions and 3) to investigate the dynamics of dreamed gravity imagery in relation to participant state and trait factors.

Methods: A total of 137 healthy participants (24.01 ± 4.03 y.o.; 85 F; 52 M) took part in a custom-built immersive VR task in which they learn how to 'fly' as precisely and quickly as possible, engaging vestibular, motor and visuo-spatial systems. Dreams were collected a) from home dream journals for 5 days before and 10 days after the laboratory VR task and b) after a 90-min morning nap in laboratory. Dream reports were scored by 2 independent judges for flying and other gravity-related imagery. Linear mixed models statistics were used to compare dreams from this cohort with a separate control cohort (N=52) that followed a similar protocol in the same lab but did not undertake a virtual flying task.

Results: The VR task successfully increased the likelihood of experiencing flying in dreams from both the laboratory nap (7.1%) and the following night (10.6%) compared to baseline (1.3%) and the control cohort on those days (Lab: 2.4%; following night: 0%). In contrast, the occurrence of other gravity imagery showed no differences. Flying dreams were altered qualitatively, exhibiting higher levels of lucid-control and emotional intensity after VR exposure. Moreover, various factors such as sex, prior dream experiences and sensory immersion in VR differentially modulated flying dream induction.

Conclusion: Our findings provide both quantitative and qualitative insights into flying dreams that may facilitate understanding of these typical dream experiences and future developments in dream flight-induction technologies.

Support: Natural Sciences and Engineering Research Council of Canada

0089

SLEEP AND HIPPOCAMPAL DEVELOPMENT IN EARLY CHILDHOOD

Allard, T. L.¹ Lokhandwala, S.² Spencer, R. M.² Riggins, T.¹ ¹University of Maryland, College Park, MD, ²University of Massachusetts Amherst, Amherst, MA.

Introduction: With sleep, memories are consolidated, leaving them less susceptible to interference. This process is believed to reflect transfer of memories from the hippocampus to the cortex. Research has established that naps benefit memory in typically napping children. This nap-benefit has been associated with sleep spindles during nREM2 sleep. Although research has separately related memory development to the hippocampus and to sleep, the association between hippocampal development and sleep physiology is not understood. The purpose of this investigation is to assess relations between sleep physiology and the hippocampus in early childhood.

Methods: Subjects are part of an ongoing longitudinal investigation. Preliminary analyses included 24 participants (Mage= 3.96 years, 14 females). Children participated in three consecutive visits, one week apart. During the first two visits, they completed a visuospatial memory task before and after a wake or nap period (order counterbalanced). Sleep physiology was assessed via polysomnography and hippocampal volumes were obtained via Freesurfer v5.1 using T1-weighted scans (.9 mm³).

Results: Preliminary results showed that total hippocampal volume was positively related to minutes spent in nREM2 sleep when controlling for age and gender (B=14.7, p=0.03). Further analysis showed that this relation held for left but not right hippocampus (B=10.1, p=0.01). Results also indicate a positive relation

between sleep spindle count and left but not right hippocampal volume when controlling for age and gender (B=16.1, p=0.02). **Conclusion:** Results show that greater time spent in nREM2 and

greater sleep spindles across nREM2 are both related to a larger hippocampus in early childhood. These findings demonstrate an association between sleep physiology and the hippocampus during an important period of memory development, early childhood. Future analyses will assess differences in hippocampal volume between typical nappers and non-nappers at the second wave of data collection. **Support:** Support was provided by NIH (HD094758) and NSF (BCS 1749280).

0090

STAGE-SPECIFIC SLEEP DISRUPTION AND ITS EFFECT ON SPATIAL NAVIGATIONAL MEMORY

Parekh, A. A.¹ Kam, K.² Mullins, A.² Fakhoury, A.² Castillo, B.² Roberts, Z.² Fleysher, L.² Rapoport, D. M.² Ayappa, I.² Varga, A.² ¹Icahn School of Medicine at Mount Sinai, New York, NY, ²Icahn School of Medicine at Mount Sinai, NEW YORK, NY.

Introduction: The mechanisms by which sleep disruption impact memory may depend on sleep stage, as rapid eye movement (REM) and slow wave sleep (SWS) differ in several significant ways, including degree of neuronal synchrony and frequency of cortical local field potential oscillations. Here we sought to examine the relationship between stage-specific disruption of sleep and its effect on spatial navigational memory.

Methods: 9 healthy adult subjects participated in this study which involved 3 in-lab polysomnograms (normal, REM-disruption, and SWS-disruption) accompanied by pre- and post-sleep functional neuroimaging of brain during a spatial navigational memory task. Graded auditory stimuli consisting of 0.5 second bursts of high-frequency tones (300-3000Hz) were used to disrupt sleep (REM/SWS) in real time. Primary metrics to ascertain the effect of these auditory tones on sleep were time in sleep stage (REM/SWS) as a % of total sleep time (TST), bout length. The primary metric for spatial navigational memory was %change in overnight completion time on a first-person-experience 3D maze task.

Results: Sleep macrostructure was normal during the normal night (TST:379.9 \pm 56.6 min; SWS:19.5 \pm 7.6%; REM:19.4 \pm 5.3%; mean \pm std). Stage-specific disruption of sleep was achieved using auditory tones during a) SWS-disruption condition (TST:388.9 \pm 47.4 mins; SWS:6.6 \pm 4.8%; REM:18.7 \pm 5.2%) and b) REM-disruption condition (TST:365.3 \pm 69.8 mins; SWS:17.1 \pm 7.7%; REM:12.1 \pm 6.6%). SWS-disruption reduced mean bout length of SWS as compared to no disruption (1.3 \pm 0.8 mins vs. 10.3 \pm 8.2 mins; p<0.01) and REM-disruption reduced mean bout length of REM as compared to no disruption (2.2 \pm 1.7 vs. 10.6 \pm 5.2 mins; p<0.01). When sleep was not disrupted, subjects achieved overnight improvements in performance (25.3 \pm 17%) which remained unchanged during REM-disruption (18.8 \pm 29.6%, p=0.5) and during SWS-disruption (38.8 \pm 24.4%; p=0.2). Morning psychomotor vigilance was also unaffected by condition.

Conclusion: Stage specific disruption of sleep can be achieved using graded auditory tones. While performance on a virtual 3D maze remain unchanged with stage specific sleep disruption, lower sample size may have limited our ability to detect the change. Activation patterns from functional neuroimaging that were acquired during the spatial navigation task may elucidate the interaction between stage-specific sleep disruption and performance.

Support: NIH R21AG059179

SLEEP AS A MEDIATOR OF THE RELATIONSHIP BETWEEN EXERCISE AND SELF-REPORTED COGNITIVE FUNCTION

Chappel-Farley, M. G.¹ Nan, B.² Grill, J. D.³ Mander, B. A.³ Yassa, M. A.¹ Benca, R. M.³

¹Department of Neurobiology & Behavior, University of California Irvine, Irvine, CA, ²Department of Statistics, University of California Irvine, Irvine, CA, ³Department of Psychiatry & Human Behavior, University of California Irvine, Irvine, CA, ⁴Department of Neurobiology & Behavior, University of California Irvine, Irvine, CA.

Introduction: Insufficient sleep and sedentary behavior are associated with cognitive decline. How sleep and physical activity interact to influence cognition is not fully understood. This study sought to examine whether self-reported sleep and exercise impact subjective cognitive complaints across adulthood.

Methods: Self-report questionnaire data from 2,744 adults (μ =56.18 yrs/old; 63.8% F; μ =16.36 yrs/edu) in the University of California Irvine Consent-to-Contact Registry were analyzed. Multiple regression models, analysis of covariance, and ordinary least squares path analysis were conducted to investigate relationships among the Cognitive Function Instrument (CFI), Medical Outcome Study Sleep Scale (MOS-SS) Subscales, and exercise frequency. All models adjusted for education, sex, age, BMI, medical comorbidities, depression, cancer diagnosis, and antidepressant usage. Individuals taking medications known to affect sleep or with a diagnosis major psychiatric illness were excluded from analyses.

Results: All MOS-SS Subscales significantly predicted CFI score (all p<0.001). Individuals who exercise >3times/week had significantly lower scores on the CFI (p<0.05), Sleep Problems Index I & II (both p<0.05), Somnolence Subscale (p<0.01), and higher scores on the Sleep Adequacy Subscale (p=0.001). Mediation analyses revealed that all subscales, aside from the Sleep Disturbance Subscale, mediated the relationship between exercise frequency and CFI Score (Bootstrapped CI's did not include zero).

Conclusion: More self-reported sleep disturbance and greater daytime sleepiness are associated with more subjective cognitive complaints. Individuals who exercise more frequently report lower daytime sleepiness and higher quality sleep. The effects of exercise frequency on cognitive complaints appear to be mediated by the impact of exercise on sleep. These results suggest that sleep health may be a crucial consideration when evaluating outcomes of exercise-based therapies aimed at delaying the onset of cognitive impairment.

Support: The C2C registry was made possible by a donation from HCP, Inc. and is supported by NIA AG016573 and NCATS UL1 TR001414.

0092

A DAYTIME NAP RESTORES HIPPOCAMPAL FUNCTION AND IMPROVES DECLARATIVE LEARNING

Ong, J.¹ Lau, T.¹ Lee, X.¹ van Rijn, E.² Chee, M. W.¹ ¹National University of Singapore, Singapore, SINGAPORE, ²Duke-NUS Medical School, Singapore, SINGAPORE.

Introduction: Daytime naps have been shown to improve learning outcomes. One theory underlying enhanced encoding following a nap is via the synaptic downscaling of neurons potentiated during

wake - a process facilitated by slow oscillations. In this study, we sought to investigate neural mechanisms underlying enhanced encoding following a nap compared to a waking period using a combination of PSG and fMRI methods.

Methods: 40 healthy undergraduates (M=23.3y, SD=2.96y; 10 males) who slept normally the previous night encoded word pair lists across 2 runs in an MRI scanner at 1PM and 4.30PM. In between encoding sessions, participants either stayed awake and watched a documentary (Wake group; N=20), or napped for 90-min while undergoing polysomnography (Nap group; N=20). Approximately 40min after each encoding session, memory of these word lists were assessed in a cued-recall fashion. Performance in each session was measured by percentage of correct responses.

Results: There were no baseline differences in encoding performance. However, a Session x Group interaction effect (p<0.001) was observed whereby performance significantly improved only in the Nap group in the second encoding session (Nap: $20\pm19\%$ vs. Wake:- $1\pm13\%$). Concurrent to this, fMRI analyses revealed a Session x Run x Group interaction effect in the hippocampus (p=0.002) whereby hippocampal activation during encoding of the word lists increased only in the Nap group. In addition, although there was no association between degree of performance improvement in the nap group with duration of sleep or the various sleep stages (N1,N2,N3,REM), spindle count (12-15Hz) in the Nap group correlated significantly with both performance improvement (r_s =0.46) and increase in hippocampal activation (r_s =0.46).

Conclusion: These results confirm the benefit of a nap on encoding processes. Hippocampal activation also increased following the nap, which could indicate renewed hippocampal capacity to store new information. While we hypothesized that slow wave sleep would aid in this transfer, we instead found a relationship between spindle count and both degree of performance improvement and increase in hippocampal activation. The interplay between NREM sleep oscillations, hippocampal downscaling and encoding performance could be more complex than originally thought.

Support: National Medical Research Council, Singapore (NMRC/ STaR/015/2013) and the Far East Organization.

0093

TOPOGRAPHICAL ANALYSIS OF SLEEP SPINDLES AND THEIR COORDINATION WITH SLOW OSCILLATIONS

Malerba, P.¹ Whitehurst, L. N.² Mednick, S. C.³ ¹The Research Institute at Nationwide Children's Hospital, Columbus, OH, ²University of California San Francisco, San Francisco, CA, ³University of California Irvine, Irvine, CA.

Introduction: Brain oscillations found during sleep are hypothesized to mediate sleep-dependent memory consolidation, by coordinating cortical-subcortical activity and enabling synaptic plasticity. In particular, sleep spindles (10-16Hz) density and coordination with slow oscillations (SOs, 0.5–1.5 Hz) have been shown to correlate with memory performance post-sleep. In this study, we characterize how spindles are organized on the electrode manifold, and their relation to SO topography.

Methods: We conducted a sleep-memory study where subjects learned word-pair associations in the morning and were tested in the evening and the next morning. Polysomnography was collected during the night. We detected sleep spindles at each electrode independently and study their basic biophysical properties (density, amplitude, frequency, duration) in light sleep (stage 2, S2) and deep sleep (slow wave sleep, SWS) separately, including their

co-occurrence with SOs. We categorize spindles that are co-detected across electrodes within a short time window and study how properties change across groups.

Results: We find a gradual increase in average spindle frequency in the frontal-occipital axis, but no bimodality in frequency distribution. Furthermore, spindles paired to SOs (a minority in both stages) are shorter than non-paired spindles. We find that coordination between spindles and SO troughs is frequency but not amplitude selective; and differs between the two sleep stages. In S2, slow spindles precede SOs in frontal electrodes and fast spindles follow SOs in centro-posterior electrodes. Our clusters of spindle topography include a Frontal and a Posterior cluster. These clusters mirror the commonly considered slow-frontal and fast-posterior spindles, but contain less than half of all S2 spindles. Clustering identifies sub-types of spindle-SO complexes whose density is linked to memory performance.

Conclusion: Our study shows that specific sub-types of sleep oscillations, defined by their topography, support the coordination between spindles and SOs which could mediate cortical-subcortical dialogue during sleep.

Support: This work was supported by NIH grant (R01 AG046646) to Sara C. Mednick

0094

THE EFFECT OF ZOLPIDEM ON SLEEP-DEPENDENT DECLARATIVE MEMORY CONSOLIDATION

Zhang, J. Mednick, S.

University of California Irvine, Irvine, CA.

Introduction: Sleep plays a critical role in memory consolidation. At the same time, about 20% of population in the U.S. suffer from sleep disorders or deprivation, and about 16% of them reported using sleep aids (CDC, 2015). However, the effect of sleep aids on sleep-dependent memory consolidation remains unclear. Previous studies have observed an improvement in sleep-dependent declarative memory consolidation with zolpidem over a daytime nap (Mednick et al., 2013). The current study investigates the effect of zolpidem on declarative memory consolidation over a night of sleep and over 24 hours.

Methods: This study employed a double-blind, placebo-controlled, within-subject design, in which every subject (N=26, 12 females) experienced both zolpidem and placebo. All subjects were healthy, college-aged adults without sleep disorder. A 32-channel electroencephalogram cap was used to record brain activity during sleep. Word paired-associates task was used to evaluate memory performance. Participants reported to the laboratory in the evening, performed word paired-associates task (test1), then ingested either zolpidem or placebo before sleep. They were tested on the task in the following morning (test2) as well as in the following evening (test3). Paired-sample t-tests for retrieval difference scores between placebo and zolpidem conditions were conducted.

Results: Participants showed similar baseline performance on the word paired-associates task (test 1, p=0.45). Zolpidem condition showed higher memory retention compared to placebo 24hr after drug ingestion (test3-test1, $t_{2_5}=2.09$, p<0.05). The improvement in performance for zolpidem condition occurred across the following day (test3-test2, $t_{2_5}=2.22$, p<0.05), as no difference was observed between conditions after sleep (test2-test1, $t_{2_5}=0.34$, p=0.74)

Conclusion: Consistent with previous studies, participants showed better memory performance after taking zolpidem compared to placebo. However, the current study showed that the improvement

in memory occurred across a day of wakefulness after nighttime drug ingestion, while other studies observed improvements shortly after sleep, indicating a potential delayed benefit of zolpidem on memory consolidation.

Support: This work was supported by the Office of Naval Research grant N00014-14-1-0513

0095

SLEEP BOOSTS SCHEMA-RELATED MEMORY CONSOLIDATION AND INFERENCE

Golkashani, H. A.¹ Chee, M. W.²

¹Yong Loo Lin School of Medicine, National University of Singapore, Singapore, SINGAPORE, ²Center for Sleep and Cognition, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, SINGAPORE.

Introduction: A schema is a previously learned framework of information that helps the learning and retention of new, but related material. We examined how sleep, compared to staying awake following the acquisition of a schema, and thereafter novel material, affects the consolidation of *new* memoranda that were either embedded or not into the learned schema. We also tested if sleep affects the inference of hierarchy within these memoranda.

Methods: 54 adolescents (mean age 16.6 years; 26 males) learned the age hierarchy of 7 galaxies by viewing adjacent pairs, one at a time e.g. A-B, D-E, and making transitive inferences (If B>C and C>D then B>D). Once this schema was learned to criterion, participants learned two *new* sets of galaxies: one set comprised 5 galaxies from the schema and 4 new, intercalated galaxies; the other contained 9 unfamiliar galaxies (schema & no-schema conditions). Memory for galaxy ordering was tested immediately after learning and again after 12-hours. One group(n=25) was kept awake in the day, while the other group (n=29) slept overnight for 8-h. Memory was tested for galaxies that were directly adjacent e.g. A-B, as well as for 'inference pairs' that were two apart: e.g.:(B-D, C-E).

Results: Change in memory following the respective 12-hour intervals was analyzed using a mixed ANOVA with schema (schema, no-schema) and pair-type (adjacent, inference) as within-subject factors and sleep group (sleep/awake) as the between-subjects factor. There were significant main effects of sleep and pair type as well as a significant interaction, where schema-related memory was better preserved in the sleep group. This group also had higher performance for inference pairs embedded within the original schema. **Conclusion:** Sleep benefitted the consolidation of new memoranda embedded in a schema. This benefit was stronger for non-adjacent inference pairs suggesting that sleep boosts insight into non-explicitly declared, hierarchically organized information.

Support: Supported by NMRC/STaR/0015/2013 and NRF2016-SOL002-001

0096

FUTURE-RELEVANT INFORMATION IS ENHANCED AFTER SLEEP DESPITE EMOTIONAL SALIENCE OF STIMULI

Whitehurst, L. N.¹ Sattari, N.² Mednick, S. C.² ¹University of California, San Francisco, San Francisco, CA, ²University of California, Irvine, Irvine, CA.

Introduction: A substantial literature supports that sleep biases memory consolidation and retrieval for emotionally-salient stimuli. A smaller, yet growing, literature supports that information deemed relevant to future events may also be favored by sleep consolidation processes. However, it is unclear whether both emotion and predictability would act together to bias sleep-dependent memory formation. Here, we utilized a directed forgetting paradigm with negative and neutral word pairs to examine this open question.

Methods: Sixty young and healthy adults were exposed to word pair stimuli before a polysomnographically-recorded nap. Before the nap, participants were instructed to remember half of the word pairs presented for a later test, and for the other half of word pairs, participants were told to forget them as there would not be a test on them later. Additionally, during encoding, half of the subjects in the sample were exposed to negative valenced, high arousal word pairs while the other half were exposed to neutral, low arousal word pairs. After the nap, subjects were tested on all word pairs presented, regardless of previous instruction. This also included a set of novel word pairs to which participants had not been previously exposed.

Results: A series of 2x2 mixed measures ANOVAs revealed that individuals had better accuracy (p<.001) and fewer misses (p<.001) for the words they were told to remember compared to those they were instructed to forget. Additionally, participants had relatively few false alarms to novel stimuli, 7.14 times, on average (SD=6.10), out of a possible 50 word pairs. Importantly, contrary to predictions, post-nap performance was not dependent on the word pair valence (interaction p's>.70). No correlations between sleep stages and memory emerged.

Conclusion: These findings suggest that predictability of future events may be a relevant catalyst for sleep-related memory processing.

Support: This work was supported by an Office of Naval Research Young Investigators Award to Sara Mednick and a National Institutes of Mental Health Training grant to Lauren Whitehurst T32MH019391.

0097

EFFECTS OF EXPERIMENTAL SLEEP DISRUPTION ON MORNING COGNITIVE PERFORMANCE AND ALERTNESS

Chinoy, E. D.^{1,2} Hirsch, D. A.¹ Cuellar, J. A.^{1,2} Snider, M. N.^{1,3} Dunn, T. L.^{1,2} Brookfield, J. S.^{1,2} Markwald, R. R.¹ ¹Naval Health Research Center, San Diego, CA, ²Leidos Inc., San Diego, CA, ³Innovative Employee Solutions, San Diego, CA.

Introduction: While sleep duration is known to affect next-day cognitive performance and alertness, largely in a dose-response manner, the effects of disrupted sleep (where one is awoken multiple times overnight, common in military settings) are much less understood. Therefore, we examined the effects of experimentally disrupted sleep on morning cognitive performance and alertness.

Methods: We tested 34 healthy participants (12 men, 22 women, 28.1 ± 3.9 years; mean \pm SD) who slept for 8-hours time-in-bed on three consecutive nights with polysomnography in a controlled sleep lab. The final two nights were randomized and counterbalanced between an undisrupted and a disrupted sleep condition. On the disrupted sleep night, participants were awoken by auditory tones for a 5–10 min period every hour. The following morning, participants completed a cognitive test battery that included Karolinska Sleepiness Scale (KSS), 10-min psychomotor vigilance task (PVT), addition calculations (ADD), go/no-go (GNG), task switching (TS), and working memory (WM). Mixed effects models were used to test factors: condition (undisrupted vs. disrupted), condition-order, and their interaction.

Results: Significant (p<0.05) effects of condition (i.e., disrupted sleep caused worse performance) were found for PVT reaction

time (RT), GNG RT, TS RT, WM percent correct, and KSS alertness ratings. Condition was not significant for number or percent correct on ADD, GNG, and TS. Condition-order was significant for TS percent correct, and significant interactions were found for ADD number correct and TS RT.

Conclusion: One night of sleep disruption caused significant negative effects on morning subjective alertness and on several, but not all, cognitive performance domains tested, including RT and WM. Condition-order and interaction effects were also found, indicating that some performance outcomes were impacted by possible learning effects over the study. Sleep disruption factors should be taken into account, especially in operational settings like the military where environmental factors (e.g., noise) disrupt sleeping conditions. **Support:** Office of Naval Research, Code 34

0098

THE ROLE OF SLEEP IN NOVEL CATEGORY LEARNING IN INFANCY

Sucevic, J. Plunkett, K.

University of Oxford, Oxford, UNITED KINGDOM.

Introduction: A growing body of research suggests that sleep has an important role in consolidating newly learnt information and generalising this knowledge to novel instances (e.g. Djonlagic et al., 2009). The effects of sleep on early categorisation abilities haven't been investigated so far and thus the aim of this study is to examine the role of sleep in formation of category representations. Two experiments were conducted. Experiment 1 set out to determine 4-months-old infants' ability to form novel visual categories when category formation test immediately followed learning. Experiment 2 investigated the impact of sleep on category consolidation by testing category formation after a 2 hours delay.

Methods: Both experiments employed eye-tracking familiarisationnovelty-preference paradigm. In Experiment 1, infants were familiarised with a set of exemplars from a novel category. Immediately after the familiarisation phase, infants' category formation was tested and their looking references were used as an index of category learning. Experiment 2 tested consolidation of the newly acquired categories by introducing a delay between the familiarisation and the test phase. Half of the participants took a nap during the delay, whereas the other half remained awake. Polysomnography was recorded using a standard PSG protocol. Sleep stages were scored according to the AASM guidelines, and sleep spindles were detected using an adapted algorithm for children.

Results: Results of the Experiment 1 revealed that infants were able to learn novel visual categories when category formation was tested immediately after learning suggesting therefore that infants are able to extract relevant information and generalise to novel instances. Preliminary results of Experiment 2 (data analysis ongoing) suggested that infants who napped performed better than infants who stayed awake.

Conclusion: The present study suggests that infants are able to rapidly form novel categories, and that consolidation of this knowledge is shaped by the activity following learning.

Support: N/A

0099

THE EFFECTS OF SLEEP ON THE CONSOLIDATION OF FEARFUL MEMORIES

Arsic, M.¹ Heiss, L.¹ Chambers, A. M.¹

¹North Central College, Naperville, IL, ²North Central College, Naperville, IL.

Introduction: Previous research has found that emotionally intense stimuli are better remembered than neutral stimuli, especially after a period of sleep. However, few studies have examined memory for experienced emotional events, especially fearful ones. The purpose of the current study was to investigate the impact of sleep on memory consolidation using a fearful emotion induction task.

Methods: Thirty-three young adults (18.94±1.06 years; 64% female) were randomly assigned to either a fearful or neutral emotion induction condition. Participants were induced into their assigned emotion by visualizing each of eight emotion-congruent scenarios while corresponding music played in the background. Emotional state was measured using the Affect Grid before and after the emotion induction procedure. Twelve hours later, spanning either a day of wakefulness (wake group) or night of sleep (sleep group), participants were asked to recall the previously presented scenarios.

Results: A 2 x 2 ANOVA examined differences in the number of scenarios recalled between the conditions. A significant main effect of sleep was found, F(1,29)=8.41, p=.007, η_p^2 =.23, reflecting better recall in the sleep (3.21±1.78) vs. the wake group (1.79±1.72). There was also a main effect of emotion, F(1,29)=22.17, p<.001, η_p^2 =.43, reflecting better recall in the fear (3.58±1.54) vs. the neutral condition (1.29±1.44). However, there was no interaction. Results were similar for the number of details recalled between the conditions. The sleep group (12.74±9.09) recalled more details than the wake group (5.50±5.81), F(1,29)=8.05, p=.008, η_p^2 =.22. More details were also recalled in the fear condition (13.16±8.73) than the neutral condition (4.93±5.77), F(1,29)=10.54, p=.003, η_p^2 =.27. There was again no interaction.

Conclusion: Results demonstrate that both sleep and fearful emotion facilitate memory consolidation. This work both supports and extends existing research by examining emotional memory consolidation through the manipulation of experienced events, which may more closely approximate real world learning than previous methods. **Support:** N/A

0100

EFFECT OF NAPS ON PRESCHOOLERS' CONSOLIDATION OF AN EMOTIONAL STORYBOOK

Holmes, J. F. Deighan, M. K. Miranda, N. W. Mason, G. M. Spencer, R. M.

University of Massachusetts - Amherst, Amherst, MA.

Introduction: Naps are known to benefit emotional memory consolidation in preschoolers, though improvement is not evident until the following day. The mechanisms by which naps aid emotional memory, and how they differ from those facilitating more neutral declarative memory consolidation, are currently unknown. In this study, we used an emotional storybook task to assess change in memory for emotionally salient vs. neutral events across a nap and overnight sleep. PSG was included to explore sleep physiology correlates.

Methods: Preschool children (n = 9; M_{age} = 43.2 months) were read a novel storybook featuring negative and neutral events. Memory of story events was probed through sets of multiple-choice questions and assessed at three time points: immediately following the story, following a nap or equivalent wake period (within-subject; counterbalanced; separated by ~1 week), and 24h post-encoding. PSG was recorded during the nap period and both subsequent overnight sleep bouts.

Results: Memory performance across time points was assessed via change scores. Recall of story events did not differ between conditions from immediate to post-nap/wake assessment. When probed the following morning, children better remembered events when a nap took place the day prior (F(1,7) = 8.848, p=.021). This delayed

nap benefit correlated with time spent in NREM2 during the nap (r=.91, p=.017). No differences were found between recall of negative vs. neutral events at any time point or between conditions.

Conclusion: Our results show a delayed benefit of napping on recall of a storybook, though at present no preference for emotional events is seen. Time spent in NREM2 during the nap was strongly associated with our finding, likely reflecting the declarative memory benefits conferred from this stage. Further analyses will include overnight sleep physiology to explore differential enhancement between event types, and possible interactions with nap microstructure. **Support:** This work was supported by NIH R01 HL111695.

0101

EPISODIC FUTURE THINKING TRIGGERS AGE-RELATED DIFFERENCES IN SPINDLES AND SLOW OSCILLATIONS

Diaz, J.¹ Fillmore, P.¹ Gao, C.¹ Scullin, M. K.¹ ¹Baylor University, Waco, TX, ²Baylor University, Waco, TX.

Introduction: In young adults, sleep spindles are theorized to represent memory consolidation. Spindle density may be especially prominent when young adults encode information that has future relevance. Older adults, on the other hand, show reduced capacity for future thinking and deficits in sleep-dependent memory consolidation. To advance these literatures, we investigated whether the process of mentally simulating the future (versus remembering the past) was associated with subsequent alterations to sleep microarchitecture in young and older adults.

Methods: 64 healthy adults aged 18–84 completed a polysomnography adaptation night followed by two in-laboratory experimental nights. On both nights, participants completed the Modified Future Crovitz Test (MFCT) in which they mentally simulated only future events or remembered only past events (night order counterbalanced). To quantify the extent of future/ past thinking, we conducted linguistics analyses on tense (future/ past) using LIWC 2015 software.

Results: On the future-thinking night, young adults with greater future-tense MFCT scores showed significantly greater spindle density across frontal, midline, and central sites (r=.42 to r=.51), even when controlling for age, gender, and total word count (all ps < .01). The opposite was true for middle-to-older aged adults; greater future-tense MFCT scores were associated with less spindle density across midline and central sites after controlling for age, gender, and word count (r=..44 to r=..46, ps<.05). However, while spindle density decreased, frontal slow oscillations increased in older adults with greater future-tense MFCT scores (r=.39, p<.05). On the past-thinking night, spindle density and slow oscillations were unrelated to past-tense or future-tense MFCT scores for either age group.

Conclusion: Age-related deficits in memory consolidation may be due to impaired tagging of information as having future relevance, or impaired physiological responses during sleep to wake-based tagging. Addressing encoding—spindle interactions may inform why cognitive functioning declines in some adults more than others. **Support:** Sleep Research Society Foundation

0102

THE RELATIONSHIP BETWEEN OVERNIGHT CONSOLIDATION AND NEXT-DAY LEARNING

Guttesen, A. V. Appleby, G. Madden, E. Gaskell, M. Cairney, S. A. Department of Psychology, University of York, York, UNITED KINGDOM. **Introduction:** Contemporary models of sleep-associated consolidation posit that overnight memory processing paves the way for next-day learning in hippocampus. However, the extent to which new hippocampal learning is dependent on overnight consolidation has yet to be investigated. In this study, we compared the impacts of sleep and sleep deprivation on the consolidation and encoding of hippocampus-dependent memories and, importantly, examined whether individual differences in consolidation were associated with individual differences in encoding.

Methods: Thirty healthy adults (17 females, mean age 20.1 ± 1.65) were trained on a spatial memory task in the evening before a night of sleep or total sleep deprivation (repeated measures). Participants completed a spatial memory test the following morning, and then encoded a novel set of word-image pairs. Two days later (after recovery sleep), memory for the word-image pairs was tested. We predicted that participants' spatial memory recall would predict performance on the word-image test, suggesting that overnight consolidation lays the groundwork for new encoding in hippocampus.

Results: Sleep (vs. sleep deprivation) improved spatial memory accuracy the following morning (t(29)=3.93, p<.001, d=0.72) and word-image recall two days later (t(29)=12.19, p<.001, d=2.23), suggesting that sleep facilitated the consolidation and encoding of hippocampus-dependent memories, respectively. However, the benefit of sleep for spatial memory recall was not significantly correlated with the benefit of sleep for word-image encoding (t(28)=0.01, p=0.971), suggesting that hippocampal encoding was not contingent on foregoing overnight consolidation processes.

Conclusion: In support of previous findings, overnight sleep improved consolidation and next-day encoding, as compared to an equivalent period of sleep deprivation. However, the present results did not reveal any relationship between an individual's sleep-associated consolidation and their next-day learning.

Support: Department of Psychology, University of York scholarship to A.áV.G. Medical Research Council Career Development Award (MR/P020208/1) to S.A.C.

0103

NOVEL PREBIOTIC ENHANCES SLEEP-WAKE CYCLE AND MEMORY CONSOLIDATION IN 5XFAD MICE

Shimomura, K.

Northwestern University, Chicago, IL.

Introduction: Prior studies have shown that the gut microbiomes of Alzheimer's Disease (AD) patients differ from unaffected individuals. Sleep and circadian rhythm disturbances are common in AD and often precede dementia symptoms. Gut microbiome alterations have also been observed in models of circadian disruption. Therefore, we hypothesized that altering the gut microbiome could improve sleep/circadian rhythms and cognition in an AD mouse model.

Methods: Mice were given a dietary polysaccharide, Modified Resistant Maltodextrin (MRM), as a 1% solution in the drinking water beginning at 2 months of age. 5xFAD and wild-type (WT) littermates were tested. Sleep-wake was recorded by EEG/EMG, memory consolidation was tested by the Object-Location Memory test, and beta amyloid deposition in the brain was assayed (dot blot). Composition of the gut microbiota was determined from amplicon sequencing of the 16s ribosomal RNA gene from fecal DNA.

Results: MRM treatment reduced dark (active)-phase sleep and the phase scattering of REM sleep in 5xFAD mice, indices of circadian consolidation. 6-month-old 5xFAD mice given plain water exhibited no 24hr retention of object location memory. However, MRM-treated 5xFAD mice demonstrated 24-hour memory, even at 12 months of age. Both improved memory and increased consolidation of sleep were also observed in WT mice. Two unclassified species of bacteria from the family Tannerellaceae were significantly increased in MRM-treated 5xFAD and WT mice. At 12 months of age, synaptic and neuronal loss become prominent in this AD model. However, the level of beta amyloid deposition in the brain was not significantly different between MRM and water control groups.

Conclusion: MRM treatment altered the gut microbiome, improved circadian timing of sleep and memory retention, but did not impact beta amyloid deposition in 5xFAD mice. Because these effects were also present in WT mice, MRM-induced microbiome changes may affect sleep and cognition independently from beta amyloid.

Support: Northwestern University Feinberg School of Medicine Center for Circadian and Sleep Medicine

0104

THE EFFECT OF TIME, SLEEP, AND WAKE ON MOTOR MEMORY CONSOLIDATION: A PARTIAL REPLICATION OF WALKER, ET AL. (2002)

Tucker, M. Wani, I.

U. of S. Carolina School of Medicine, Greenville, SC.

Introduction: Findings from Walker, et al (2002) 'Practice with Sleep Makes Perfect: Sleep-Dependent Motor Skill Learning' demonstrate that performance on a widely used motor memory task (motor sequence task (MST)) benefits from a 12hr period of sleep (and not wake) even if the sleep period does not occur for approximately 12hrs after task acquisition, suggesting that sleep is crucial for motor memory consolidation. Using a larger sample, we attempted to replicate this finding, which is derived from Groups B & D from Walker et al (2002).

Methods: Participants (64 medical students: Age 21.2 ± 0.8 ; N=33 females) were trained on the MST in the morning (10am; N=40) or evening (10pm; N=24) and then returned 12 and 24hrs later to be retested. The MST is a simple typing task that requires participants, at training, to type a 5-digit sequence (e.g., 4-1-3-2-4) as fast and accurately as possible over a series of 12 30-second trials with a 30-second break between each trial. At each retest, participants performed three 30-second trials.

Results: With 75% of the data collected we have found that when sleep follows training in the evening (first 12hr interval), the number of correctly typed sequences increased by 19.1% (cf. 20.5% in Walker (2002)). After a subsequent day of wake (second 12hr interval) performance increased by an additional 7.3% (cf. 2.0%). However, when a day of wake spanned the first 12hrs following training, performance increased by 14.5% (cf. 3.9%) followed by another 14.5% increase over the subsequent night (cf. 14.4%). Performance differences between sleep and wake participants were nonsignificant over the first 12hrs (p=0.38) and second 12hrs (p=0.49).

Conclusion: With most of data collection complete, our findings only partially replicate those of Walker et al (2002), and may draw into question the robustness of sleep for the processing motor memory.

Support: None

WORKING MEMORY IMPAIRMENT DUE TO CHRONIC SLEEP RESTRICTION, DOSE RESPONSE TO RECOVERY AND RE-EXPOSURE

Kaizi-Lutu, M. Jones, C. Mange, A. Basner, M. Dinges, D. F. Perelman School of Medicine University of Pennsylvania, Philadelphia, PA.

Introduction: Chronic sleep restriction negatively effects working memory. Recovery sleep following sleep restriction partially restores working memory performance. This study examines the impact of chronic sleep restriction and subsequent recovery sleep dose on the N-Back Task (N-Back), a valid measure of working memory.

Methods: N=223 participants (29.9 \pm 6.9 years; 48.4% female), completed two baseline nights of 8h time in bed (TIB), followed by five nights of 4h TIB, and were then randomized to a sleep dose of 0, 2, 4, 6, 8, 10, or 12 h TIB. A subset of participants (n=73) were re-exposed to another five nights of 4h TIB. Participants completed the three versions of the N-Back (i.e. 1-Back, 2-Back, and B-back) every two hours during wakefulness and daily averages were computed. Mixed effects and linear regression models were used to assess the impact of sleep restriction and the sleep dose response on percent correct on the N-Back corrected for baseline. **Results:** N=219 participants had valid working memory data. The

2-Back (β =-4.5%; P<0.0001) and the 3-Back (β =-12.5%; P<0.0001) were more difficult than the 1-Back. Working memory performance declined across days of sleep restriction for all N-Backs: 1-Back (β =-1.10%; P<0.0001), 2-Back (β =-0.99%; P<0.0001), and 3-Back (β =-1.10%; P<0.0001). The sleep dose analysis revealed a positive association with N-Back performance for all N-Back versions, 1-Back (β =0.99%; P=0.0002), 2-Back (β =1.46%; P<0.0001), and 3-Back (β =1.43%; P<0.0001). Re-exposure to only one night of 4h TIB following recovery sleep resulted in performance decrements equal to performance prior to recovery sleep for all N-Back versions (Ps>0.41).

Conclusion: These data indicate that working memory is adversely impacted by sleep restriction, and that sufficient recovery sleep, possibly across consecutive days, is necessary to maintain optimal working memory performance.

Support: Funded by National Institute of Health NIH R01NR004281 and National Space and Biomedical Research Institute NSRBI NCC 5–98

0106

THE EFFECTS OF CAFFEINE ON BLOOD PRESSURE AND COGNITIVE PERFORMANCE IN HYPOXIC CONDITIONS ON THE SLOPES OF MT. EVEREST

Sparks, K.¹ Wehling, R. R.¹ Acharya, S.² Musliu, T.¹ Baniya, S.² Hackett, P. H.³ Ozuru, Y.¹ Jung, C. M.¹

¹University of Alaska Anchorage, Anchorage, AK, ²Mountain Medicine Society of Nepal, Kathmandu, NEPAL, ³University of Colorado Denver, Denver, CO, ⁴University of Alaska Anchorage, Anchorage, AK.

Introduction: 140 million people live above 2,400m worldwide. High altitude (HA) exposure can lead to sleep disruption, impaired cognitive performance, acute mountain sickness (AMS), elevated blood pressure (BP) and an increase in cardiovascular events in healthy people. Because caffeine can also increase BP, caffeine might need to be avoided at HA. Caffeine is the most widely used drug in the world but has yet to be studied extensively in hypoxic conditions. Therefore, the aim of the current study was to examine the effects of caffeine on cardiovascular variables and cognitive function at HA.

Methods: We conducted a non-randomized, single-blind, mixed model design at 4,300m on Mt. Everest. Thirty-three trekkers (nine females), aged 29.5 ± 10.4 (mean \pm SD), ingested the study drug (placebo or 200 mg of caffeine) 1.5 hours after awakening. To control for withdrawal effects of caffeine, participants that self-reported consuming less than 47 mg of caffeine per day received the placebo while those that consumed more than that received caffeine. Cognitive function was tested using the Stroop task before and after the pill administration. BP was measured by a trained clinician using auscultatory method prior to and 30, 60 and 90 min after the pill administration.

Results: Caffeine improved cognitive performance when compared to the pretreatment measurement but was worse in the caffeine group prior to the pill administration when compared to the pretreatment placebo group. Additionally, caffeine did not have any major effect on BP when compared to pretreatment measures or the placebo group.

Conclusion: Caffeine does not seem to have an additive effect on increasing BP with HA. Additionally, because cognitive performance was worse in the chronic caffeine users prior to the pill administration, caffeine users might be more dependent on caffeine to perform optimally at HA. Based off of these data, caffeine seems to be a safe and beneficial drug at HA.

Support: NIH BUILD EXITO, University of Alaska Faculty Development Grant

0107

NEUROCOGNITIVE FUNCTIONING IN INDIVIDUALS WITH COMORBID INSOMNIA AND SLEEP APNEA: BASELINE FUNCTIONING AND IMPACT OF TREATMENT

Turner, A. D.¹ Ong, J.² Tu, A.² Crawford, M.³

¹New York University, New York, NY, ²Northwestern University, Chicago, IL, ³University of Strathclyde, Glasgow, UNITED KINGDOM.

Introduction: Neurocognitive impairments are common in individuals with insomnia and sleep apnea separately, however, the evidence for impairment in comorbid insomnia and sleep apnea (COMISA) is much less clear. We aim to determine a neurocognitive profile for individuals with COMISA, and evaluate the benefits of treatment on neurocognitive performance.

Methods: Participants with COMISA (n=45; 51.1% female; mean age= 52.07 ± 13.29) were from a two-phase, 3-arm randomized clinical trial combining Cognitive Behavioral Therapy for insomnia (CBT-I) with Continuous Positive Airway Pressure (CPAP) administered sequentially or concurrently compared to CPAP alone. The unique groups from this factorial design were used to establish baseline impairment and the effects of treatment on neurocognitive functioning.

Results: Individuals with COMISA had a slower reaction time (365.66 ± 157.39) and more lapses (9.70 ± 16.16) on the Psychomotor Vigilance Task than has been reported in the literature for insomnia and OSA separately. Our COMISA participants had mean scores indicating mild impairment for their age, based on normative data, on digit span $(9.71\pm2.47$ forward; 3.95 ± 2.63 backward), verbal paired associates $(27.71\pm11.54$ immediate; 8.69 ± 3.16 delayed; 37.17 ± 2.97 recognition), Digit Symbol Substitution (68.83 ± 12.33) and the Stroop $(35.46\pm7.26$ color/word; -3.19 ± 7.5 interference). The only significant treatment outcome was in the acute effect

SLEEP, Volume 43, Abstract Supplement, 2020

after CBT-I on digit span forward (1.7 fewer digits remembered after CBT-I vs. 0.2 fewer after no treatment, $t_{(32)}$ =-2.1, p=0.047). This effect was no longer significant after controlling for multiple comparisons.

Conclusion: This is the first study to report baseline neurocognitive impairments and effects after treatment for individuals with COMISA. Baseline impairments appear to be more significant than reported in the literature for conditions individually and published norms. Results indicate a worsening in neurocognitive functioning after CBT-I, however the numbers within the groups are small, and this effect disappears after controlling for multiple comparisons. Future studies are necessary to provide a conclusive answer regarding the effects of treatment on neurocognition.

Support: NIH/NHLBI (R01 HL114529-03S1) Ong 1/7/2015 - 6/30/2017 *Project Title: Multidisciplinary Treatment for Obstructive Sleep Apnea and Insomnia* - Research Supplement to Promote Diversity in Health-Related Research

0108

BRIEF PERIODS OF SLEEP AND QUIET REST EQUIVALENTLY BENEFIT MEMORY CONSOLIDATION

Baker, K. C. Wang, S. Y. Culbreth, J. L. Morris, S. C. Arora, M. J. Tracy, O. J. Liu, T. J. Collins, M. B. Wamsley, E. J. Department of Psychology, Furman University, Greenville, SC.

Introduction: Past research has demonstrated that sleep benefits the consolidation of memories. However, more recent studies have suggested that quiet rest could have similar benefits for memory. Here, we examined the effect of a brief period of sleep, quiet rest, or active wakefulness on declarative and procedural memory. We hypothesized that sleep and quiet rest would equally benefit memory, compared to a period of active wakefulness.

Methods: After completing a declarative (Icelandic-English word pairs) and procedural memory task (the Motor Sequence Task (MST)), participants began a 30-min retention period with PSG monitoring, in which they either slept (n=24), quietly rested with their eyes closed (n=22), or completed a distractor task (n=28). Following the retention period, participants were tested on the same memory tasks they completed earlier.

Results: Percent improvement on the MST from the end of training to the end of the test session differed by condition, F(2, 73)=4.21, p=.019. Sleep and quiet rest led to nearly identical improvement (p=.95), with improvement in both of these conditions being significantly greater than in active wake (sleep vs. active wake: p=.01; quiet rest vs. active wake: p=.02). Similarly, retention of the Icelandic-English word pairs differed by condition (F(2, 73)=5.68, p=.005), with sleep and quiet rest demonstrating nearly identical memory change over time (p=.81), and retention in both of these conditions being significantly higher than in active wake (sleep vs. active wake: p=.007; quiet rest vs. active wake: p=.004).

Conclusion: These data suggest that sleep and quiet rest can exert an equivalent effect on memory consolidation for both declarative and procedural memory, at least across very brief retention durations. Therefore, neurobiology specific to sleep might not be necessary to induce offline improvement in memory across short intervals.

Support: This research was supported by National Institutes of Health Award R15MH107891.

0109

SLOW WAVE SLEEP TIME AND ITS OSCILLATORY FEATURES SHOW OPPOSITE ASSOCIATIONS WITH EMOTIONAL MEMORY CONSOLIDATION FOLLOWING STRESS

Denis, D.¹ Kim, S. Y.¹ Kark, S. M.² Daley, R. T.³ Alger, S. E.⁴ Kensinger, E. A.³ Payne, J. D.¹

¹University of Notre Dame, South Bend, IN, ²Center For The Neurobiology Of Learning And Memory, Irvine, CA, ³Boston College, Boston, MA, ⁴Walter Reed Army Institute of Research, Silver Spring, MD.

Introduction: Sleep and stress can both enhance emotional memory consolidation. During slow wave sleep (SWS), oscillatory features such as slow oscillations (SO), sleep spindles (SS), and critically, their coupling, are believed to facilitate consolidation. How they relate to emotional memory consolidation is less clear, and how stress interacts with these oscillations is unknown.

Methods: In this study, participants either underwent a psychosocial stressor (the Trier Social Stress Task; n = 32) or a control task (n=32). Next, they encoded 150 neutral, negative, and positive images while undergoing fMRI. Participants then spent the night in the lab with polysomnographic recording. The next day they were given a surprise recognition test.

Results: There was better memory for emotional compared to neutral items in the stress group. Within this group, % of time spent in SWS positively correlated with emotional memory consolidation (r=.37, p=.039). However, SO-SS coupling during SWS was negatively correlated with emotional memory consolidation in the stress group (r=-.47, p=.007). This was driven by participants who showed a high cortisol response following the stressor (cortisol * coupling interaction p=.03) Results were similar when negative and positive items were analyzed separately. No correlations with neutral item memory were found.

Conclusion: Sleep stage time and sleep oscillatory activity exert different effects on emotional memory following stress, and that SO-SS coupling does not always promote episodic memory consolidation. SO-SS coupling can impair emotional memories when encoded during periods of elevated stress, and accompanying neuromodulators such as cortisol are high.

Support: National Science Foundation, Grant/Award Number: BXS-1539361

0110

FLUCTUATIONS ACROSS THE MENSTRUAL CYCLE IN CARDIAC AUTONOMIC ACTIVITY DURING SLEEP AND WAKE MAY AFFECT MEMORY CONSOLIDATION

Sattari, N. Simon, K. Mednick, S. UC Irvine, Irvine, CA.

Introduction: Prior studies have shown that benefits of sleep for memory consolidation may be influenced by menstrual phase. Menstrual phase also impact autonomic regulation during sleep, and autonomic activity has been recently shown to play a role in sleep-dependent memory consolidation.

Methods: We investigated the interaction of menstrual cycle and autonomic activity measured by heart rate-variability (HRV) on sleep-dependent memory consolidation among 18-healthy females. Using a within-subjects design, we investigated episodic memory improvement with a nap paradigm during two phases of women's menstrual cycle: 1) perimenses: -5 to +5 days from menses-onset,

and 2) non-perimenses: window outside of perimenses. Subjects completed the memory test before (Test1) and after (Test2) a 90-minute polysomnographically (PSG)-recorded nap. We recorded sleep and HRV during 5-minutes of wake, and during the nap. Next, we compared sleep, HRV (RMSSD and HFnu), and memory performance between the two menstrual phases.

Results: Sleep architecture did not differ between perimenses and non-perimenses. Women performed similarly on the memory task at Test 1 (all *ps*>.061), but at Test 2, non-perimenses showed better performance (p = 0.02). Autonomically, perimenses had higher parasympathetic activity during wake (RMSSD-p = 0.04) and REM-sleep (HFnu-p = 0.04), compared with non-perimenses. Using bivariate correlations, we found positive associations between wake-HFnu and memory improvement (p = .02) during perimenses. In contrast, non-perimenses' memory improvement was negatively correlated with wake-RMSSD (p < .001). In perimenses, memory improvement was also positively associated with REM-HFnu (p = .04). No associations were found between autonomic sleep activity and memory in non-perimenses phase.

Conclusion: Our findings indicate a role for autonomic activity in memory improvement in both sleep and wake that is modulated by the menstrual cycle. HRV measures of parasympathetic activity were higher during perimenses phase in wake and REMsleep. Interestingly, the HRV measures showed opposing relations with memory improvement based on the phase of the menstrual cycle. In sum, women's cardiac autonomic activity fluctuates by menstrual phase and it is possible that these fluctuations affect the magnitude and direction of sleep-related memory consolidation. **Support:** Sattari et al., 2017; Genzel et al., 2012; de Zambotti et al.,

2013; Whitehurst et al., 2016.

0111

EMOTIONAL MEMORY-ASSOCIATED VOXEL-EXTENT REACTIVATION DURING EPISODIC MEMORY RETRIEVAL VARIES AS A FUNCTION OF POST-LEARNING SLEEP

Bottary, R. M.¹ Kark, S. M.² Daley, R. T.¹ Payne, J. D.³ Kensinger, E. A.¹

¹Boston College, Chestnut Hill, MA, ²University of California Irvine, Irvine, CA, ³University of Notre Dame, Notre Dame, IN.

Introduction: Slow wave sleep (SWS) and rapid eye-movement (REM) sleep enhance neutral and emotional memory consolidation, respectively. Emotional episodic memory retrieval is also enhanced when encoding-specific functional brain patterns are reactivated at retrieval, especially in ventral visual stream and frontal brain regions as well as amygdala. Here we investigate how sleep impacts the association between memory-dependent brain pattern reactivation and episodic memory retrieval.

Methods: Healthy adults (N = 22; 11F, 11M; age: 19–29 years) were scanned during an incidental encoding task and a surprise recognition memory task 24h later. Overnight sleep was monitored with polysomnography. During encoding, participants viewed line drawings of negative, neutral, and positive images, each followed by their full-colored photo. At recognition, participants distinguished new from encoded line drawings. Brain reactivation was measured at the single-subject level as the percentage of voxels activated at encoding that were also activated during successful recognition (reactivation%); this metric was calculated independently in wholebrain and 3 ROI-based maps (inferior temporal lobe (ITL), medial prefrontal cortex, and amygdala). Multiple linear regression was

performed to predict memory performance from functional brain reactivation and sleep physiology.

Results: In whole-brain analyses, the association between negative memory performance and reactivation% decreased with greater REM sleep amount. This interaction approached significance for positive, but was not significant for neutral, memory performance. Additionally, the association between neutral, but not emotional, memory performance and reactivation% decreased with greater amounts of SWS sleep. In ROI-based analyses, positive, but not negative or neutral, memory performance was independently predicted by REM sleep amount and ITL reactivation%. No effects of SWS amount were observed in ROI-based analyses.

Conclusion: Greater amounts of sleep decreased the association between brain reactivation and memory performance. Sufficient sleep may change cortical representations of episodic memories, resulting in less reliance on encoding-related reactivation during memory retrieval.

Support: NSF Grant BCS 1539361

0112

LUCID DREAMING ASSOCIATED WITH POSITIVE WAKING MOOD

Carr, M.¹ Stocks, A.² Mallett, R.³ Konkoly, K.⁴ Freegard, M.² Hicks, A.² Crawford, M.² Pigeon, W.¹ Schredl, M.⁵ Bradshaw, C.² ¹University of Rochester, Rochester, NY, ²Swansea University, Swansea, UNITED KINGDOM, ³University of Texas at Austin, Austin, TX, ⁴Northwestern University, Evanston, IL, ⁵Central Institute of Mental Health, Mannehim, GERMANY.

Introduction: Lucid dreaming (being aware that one is dreaming) is typically a positive experience that may enhance positive mood even after waking. There is concern, however, that lucid dreaming may interfere with sleep quality. In the current experiment, participants practiced common lucid dream induction techniques over the course of a week, and kept a daily sleep and dream diary. The study objective was to assess relationships between dream lucidity and subjective sleep quality, dream emotional content, and subsequent waking mood.

Methods: There were 32 participants aged 19–33 in this open label, single arm study (mean= 22.63 ± 3.48 ; 6 males, 24 females). All participants completed a sleep and dream diary for 7 days that included scaled items (1–7 scale) concerning subjective sleep quality, negative and positive emotional intensity of a dream (if recalled). Participants also completed a 19-item lucidity questionnaire, and the Positive and Negative Affect Schedule. Average scores for the week were computed for all measures and Pearson's correlations conducted between lucidity and all other measures. Participants with no dream recall (n=5) were excluded. Within-subjects analyses were undertaken by selecting each participant's highest and lowest lucidity night (n=22; 5 participants with only minimum lucidity excluded).

Results: Positive correlations were found between lucidity and dream positive emotion (r=.490, n=27, p=.009) and positive waking mood (r=.638, n=27, p<.001); there were no other significant correlations (all p>.1). Higher lucidity was associated with more positive dream content (t(21)= -3.214, p=.004) and positive waking mood (t(25)=-4.568, p<.001); no other significant differences were observed.

Conclusion: These data indicate that lucidity is associated with positive dreams and waking mood, with no detriment to self-reported sleep quality. The findings provide preliminary support of lucid dreaming as an intervention to improve wellbeing and mood in the short term.

Support: N/A

0113

EVALUATING CLOSED-LOOP AUDITORY STIMULATION DURING SLEEP AS AN INTERVENTION TO IMPROVE MEMORY CONSOLIDATION DEFICITS IN SCHIZOPHRENIA

Baxter, B.¹ Kwok, K.² Talbot, C.² Zhu, L.² Mylonas, D.¹ Stickgold, R.³ Manoach, D.¹

¹Massachusetts General Hospital, Harvard Medical School, Boston, MA, ²Massachusetts General Hospital, Boston, MA, ³Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA.

Introduction: Converging evidence supports the hypothesis that reduced sleep spindles and spindle-slow oscillation (SO) coordination contribute to cognitive deficits in schizophrenia. Closed-loop auditory stimulation in healthy adults increases sleep spindles and improves declarative memory consolidation. Here we investigated whether closed-loop auditory stimulation also improves sleep-dependent procedural memory consolidation as a first step towards an intervention in schizophrenia.

Methods: Thirteen healthy adults participated in two nap sessions (stimulation or detection only) with polysomnography in a counterbalanced order. Participants were trained on the finger tapping Motor Sequence Task (MST), which measures sleep-dependent motor procedural memory consolidation, prior to napping and were tested after awakening. We detected the negative peak of SOs during non-REM sleep and, in the stimulation condition, delivered 50ms of pink noise during the SO up-state.

Results: Auditory stimulation increased SOs and spindles during the SO up-state in a frontocentral cluster of electrodes 800-1200ms after stimulation compared to detection only (p<0.05). Stimulation also showed promise for improving memory consolidation (33% increase in MST overnap improvement from detection-only) but this did not reach significance in this small sample and data collection is ongoing.

Conclusion: Auditory stimulation evoked coordinated spindle-SO events that mediate memory consolidation, but more subjects are needed to evaluate whether it also improves memory. If it does, we will test the effects of stimulation on sleep-dependent memory deficits in patients with schizophrenia. Closed-loop auditory stimulation shows promise as a safe, scalable intervention for cognitive deficits that can be implemented at home with commercially available devices.

Support: R01 MH67720 (DSM & RS), NIH-NHLBI 5T32HL007901-17 (BB), K24MH099421 (DSM), and Simons Foundation (DSM).

0114

MORNING STIMULANT ADMINISTRATION REDUCES SLEEP AND OVERNIGHT WORKING MEMORY IMPROVEMENT

Tselha, T.¹ Whitehurst, L. N.² Tina, V.³ Benjamin, Y. D.¹ Mednick, S. C.¹ ¹UC Irvine, Irvine, CA, ²UCSF, San Francisco, CA, ³UC Riverside, Riverside, CA.

Introduction: The goal of cognitive enhancement is to improve mental functions using interventions including cognitive training, brain stimulation and pharmacology. Indeed, psychostimulants, commonly used for cognitive enhancement purposes, while preventing sleep, have been shown to increase working memory (WM) and attention. WM is widely believed play a core role in cognitive ability, and has been shown to correlate with broad measure of cognitive ability and fluid intelligence. Sleep, however, is also important for cognitive function; thus, understanding the interaction between stimulants, sleep and cognition may inform current approaches to cognitive enhancement.

Methods: We used a double-blind, placebo controlled, repeatedmeasure design to investigate the effect of morning administration (9am) of stimulant, dextroamphetamine (DEX, 20 mg), on withinday and overnight WM performance, and sleep in 46 (22 female) healthy young adults. We tested WM using an operation span task (OSPAN) as it engages and captures both the memory retention and online processing capacity of WM. WM was tested at 75 minutes post drug, 12 h post drug, and 24 h post drug over a night of sleep.

Results: Compared with placebo, DEX showed no changes to WM performance at 75min or 12-hr post-drug. After sleep, DEX performed worse than PBO and the overnight improvement in performance in the PBO condition was absent in the DEX condition. Moreover, sleep quality was negatively affected by DEX administration.

Conclusion: In summary, we found no cognitive boost from psychostimulants across a day of wake and a blockade of overnight WM increases with the stimulant, compared to PBO. Given the growing non-medical use of stimulants in young adults, these findings have important implications for assessing their benefit for cognitive enhancement.

Support: Office of Naval Research N00014-14-1-0513 (S.M.) and DoD Young Investigator Prize (S.M.)

0115

THE EFFECT OF ZOLPIDEM ON SLEEP DEPENDENT EMOTIONAL MEMORY CONSOLIDATION

Simon, K. C.¹ Whitehurst, L.² Zhang, J.¹ Mednick, S.¹

¹Cognitive Science Department, University of California, Irvine, Irvine, CA, ²Department of Psychiatry, University of California, San Francisco, San Francisco, CA.

Introduction: Psychopharmacological treatment is widely promoted for insomnia treatment. Zolpidem (ZOL) is a GABA A agonist that depresses the central nervous that has demonstrated unexpected benefits, specifically increased sleep-dependent verbal memory via increased phase-amplitude coupling between slow oscillations and sleep spindles (Niknazar et al., 2015). Here we investigated if ZOL improved sleep-dependent emotional memory consolidation.

Methods: Using a within-subjects, cross-over design, we counterbalanced the administration of zolpidem (ZOL) or placebo (PBO) to 37 subjects in a double-blind study. Prior to drug or placebo administration, subjects rated their subjective physiological arousal of negative and neutral pictures. Subjects were tested on their recognition of the pictures twice, before (PM) and after (AM) a night of sleep. All subjects were monitored polysomnographically across the night.

Results: We analyzed emotional picture recognition using 2-by-2 ANOVA (emotion x drug condition). We found a main effect of emotional picture PM performance, such that negative pictures were remembered better than neutral pictures. There was a significant main effect of sleep on AM false alarm rate, with greater false alarms for negative than neutral pictures, a significant interaction between drug and emotion on AM dprime, and a significant

interaction for the difference between AM and PM performance and drug condition. Specifically, we found memory maintenance for both emotional picture types in ZOL but negative picture memory decline in PBO. Across the night, ZOL showed greater memory performance than PBO if subjects had greater N2 SWA and N2 sigma activity (12-15Hz).

Conclusion: Zolpidem benefits sleep-dependent emotional memory consolidation by decreasing overnight forgetting. Further, it appears that spindle activity may play a key role in ZOL's memory effect. **Support:** NIH AG046646

0116

OPPOSING EFFECTS OF SLEEP ON THE MISINFORMATION EFFECT: SLEEP PROMOTES AND PREVENTS MEMORY DISTORTION

Day, A. J. Fenn, K. M.

Michigan State University, Lansing, MI.

Introduction: The effect of sleep on false memory is equivocal. In the Deese-Roediger-McDermott illusory memory paradigm, some work shows that sleep increases false recall whereas other work shows that sleep decreases false recognition. Given these ambiguous findings, we sought to investigate the effect of sleep on false memory using the misinformation paradigm.

Methods: Participants watched a short film depicting a home burglary, received misinformation about the film, and were tested on their memory for the film. The recognition test was given after a 12-hour retention interval that included either sleep or wake. We manipulated the time at which participants received misinformation. Half were given misinformation after encoding (before sleep or wake) and the other half were given misinformation after the retention interval (after sleep or wake).

Results: There was a main effect of condition on correct recognition; participants in the sleep group showed higher correct recognition than those in the wake group. On false memory, there was a main effect of timing of misinformation and an interaction between condition and timing of misinformation. That is, the effect of sleep on false memory depended on when misinformation was administered. If misinformation was given after the retention interval, false memory tended to be lower after sleep than wake whereas if misinformation was given before the retention interval, false memory tended to be higher after sleep than wake.

Conclusion: Sleep can both protect against and facilitate memory distortion depending on when misinformation is encountered. These results inform our understanding of consolidation processes. When consolidation acts on true memory alone, it strengthens that memory making it resistant to distortion. Conversely, when misinformation is presented before consolidation, sleep may integrate misinformation into memory for the true event, increasing distortion. This work has important theoretical implications for memory consolidation and important applied implications for interrogation practices. **Support:** N/A

0117

CIRCADIAN- AND WAKE-DEPENDENT EFFECTS ON RECALL FOR FACE-NAME PAIRS

Yuan, R. K.^{1,2} Münch, M. Y.^{1,2,3} Cain, S. W.^{1,2,4} Ronda, J. M.^{1,2} Czeisler, C. A.^{1,2} Duffy, J. F.^{1,2}

¹Brigham and Women's Hospital, Boston, MA, ²Harvard Medical School, Boston, MA, ³Massey University, Wellington, NEW ZEALAND, ⁴Monash University, Clayton, AUSTRALIA. **Introduction:** The ability to remember the face and name of a person we have recently met is a critical skill often impacted by cognitive impairment and Alzheimer's disease. We used a forced desynchrony protocol to explore whether recall of recently-learned face-name pairs is affected by time awake and/or circadian phase in healthy adults.

Methods: 13 healthy, cognitively normal adults (20-70yrs; 7F) participated in a 39-day inpatient protocol with 3 baseline days (10h time-in-bed/24h) and a 3-week forced desynchrony (FD) segment, where they lived on a 28-h day with sleep restriction (6.5h timein-bed/28h, equivalent to 5.6h/24h). Core body temperature was collected throughout to estimate circadian period and phase. The face-name test was administered every 4h, beginning 3h after wake. Each test included a learning session with 6 novel face-name pairs. Recall was tested 2h later, when each face was presented twice in random order, once with a correct and once with an incorrect name. Participants were asked to respond whether each face-name pair was correct. Data were averaged across 4-h circadian phase or time awake bins and normalized as a percentage of each participant's baseline performance.

Results: Face-name recall varied by time awake (p<0.05), with performance deteriorating ~12% over the course of 12h of wake-fulness. Face-name recall also varied by circadian phase (p<0.05), with a ~10% difference in recall performance from the peak at circadian phase 240° (corresponding to the early biological evening) to the nadir at circadian phase 60° (corresponding to the early biological morning).

Conclusion: Both duration of prior wake and biological time of day impact the ability to correctly recall face-name pairs. Under normal entrained conditions, opposing circadian- and wake- dependent effects on memory for face-name associations may interact to produce stable performance across the day.

Support: Study supported by P01 AG009975 and conducted at Brigham and Women's Hospital Center for Clinical Investigation, part of Harvard Clinical and Translational Science Center supported by UL1 TR001102. Authors supported by T32HL007901 and F32HL143893 (RKY); fellowships from the Novartis Foundation, the W.&T. La-Roche Foundation, and Jazz Pharmaceuticals (MYM); a fellowship from the Natural Sciences and Engineering Research Council of Canada (SWC).

0118

IMPROVED WORKING MEMORY IS RELATED TO NON-REM DELTA ACTIVITY IN CONTROL BUT NOT PTSD PARTICIPANTS

LaGoy, A. D.¹ Kaskie, R.² Connaboy, C.¹ Laxinarayan, S.^{3,4} Reifman, J.⁵ Germain, A.¹ Ferrarelli, F.¹

¹University of Pittsburgh, Pittsburgh, PA, ²University of Pittsburgh Medical Center, Pittsburgh, PA, ³Department of Defense Biotechnology High Performance Computing Software Applications Institute, Fort Detrick, MD, ⁴Henry M. Jackson Foundation for Advancement of Military Medicine, Bethesda, MD, ⁵Department of Defense Biotechnology High Performance Computing Software Applications Institute, Fort Detrick, PA.

Introduction: Individuals with post-traumatic stress disorder (PTSD) experience altered sleep and daytime function, including deficits in working memory (WM), the ability to store and manipulate information over short timeframes. As sleep contributes to WM, understanding how sleep parameters influence changes in WM may provide insight into potential intervention targets to improve or restore daytime function. Here, we investigated the

relationship between sleep and WM improvement in Veterans with and without PTSD.

Methods: Forty-eight post-911 Veterans without PTSD (Control) and 37 with PTSD (PTSD) completed a 48-hour lab stay during which WM was assessed using a n-back task. Nighttime polysomnography, using high-density (64-channels) electroencephalography, quantified time spent in non-REM and REM sleep and log-transformed spectral activity for non-REM sleep in delta (.5-4Hz), theta (4-8Hz), alpha (8-12Hz), sigma (12-16Hz), and beta (16-32Hz) bands. Pearson's correlations were used to assess associations between baseline sleep and baseline WM on 1-back trials. Within control and PTSD groups, independent samples t-tests were used to compare changes in sleep across nights between improvers and non-improvers categorized by 1-back accuracy changes across davs.

Results: Proportions of improvers and non-improvers were similar between groups ($\chi^2 = .023$, p = .880). Within either group, baseline sleep did not relate to baseline WM and changes in time spent in each sleep stage did not differ between improvers and nonimprovers. Within the control group, improvers (n = 15; 3.67 \pm 3.56) had a greater increase (t = -2.826, p = .007) in delta activity than non-improvers (n = 33; .77 \pm 3.20), but this relationship was not observed in the PTSD group (11 improvers, 26 non-improvers). Conclusion: Increased delta activity related to improved WM in the Control but not PTSD group. This suggests individuals with PTSD do not improve WM through non-REM sleep but that it may be a useful intervention target.

Support: USAMRMC MOMRP PT-130572 (PI: Reifman)

0119

THE ROLE OF AGING AND WORKING MEMORY IN EMOTIONAL-LONG TERM MEMORY FORMATION

Sattari, N.¹ Whitehurst, L.² Vinces, K.¹ Mednick, S.¹

¹UC Irvine, Irvine, CA, ²UCSF Weill Institute for Neurosciences, San Fransisco, CA.

Introduction: It is widely accepted that "offline" processes during sleep contributes to memory. Working Memory (WM) capacity, which reflects "online" memory processing, is an important factor influencing cognitive functioning, which declines with age. In younger individuals, a positive association is reported between WM-capacity and declarative memory improvement.

Methods: We examined the relation between WM and long-term memory consolidation, among younger [N=105, 18-25yr] and older adults (N=119, 60-85yr). Subjects completed an OSPAN WM task, encoded a Word-Paired Association (WPA) task in the morning (Test1), and were tested on the WPA in the afternoon (Test2) after a 90-minute polysomnographically-recorded nap or wake. Half of the subjects were exposed to negatively valenced word-pairs (EWPA) while the other half were exposed to neutral word-pairs (NWPA). Subjects rated valence of the word-pairs at Test1 and Test2. We compared the four groups (young-EWPA, young-NWPA, old-EWPA and old-NWPA) on WM and WPA in both wake and sleep. Results: In both wake and sleep, in the WPA, ageXword-condition interaction was found (p=.004). Post-hoc analysis revealed that in wake, younger-EWPA had higher performance (p=.03) than younger-NWPA, however, older-EWPA had lower performance (p=.03) than older-NWPA. Additionally, we found an ageXwordcondition interaction whereby youngers showed no change in ratings, while older adults rated word-pairs more positively both in wake (p=.03) and sleep (p=.002) at Test 2. Youngers had higher WM performance (p=.007), also their WM performance was

positively associated with WPA both for Neutral (p=.03) and Emotional (p=.01). WM and WPA among older adults was not related. In younger-EWPA, Stage2-sleep-minutes was positively associated to WPA improvement (p=.03) where this association was negative among older-EWPA (p=.02). In older-NWPA, Stage2sleep-minutes was positively associated with WPA (p=.004).

Conclusion: Our findings indicate an association between WM and emotionally-salient memory formation that is modulated by age. Older adults, but not younger, showed the emotional bias previously reported. WM was higher in younger adults related to memory improvement. Stage2-sleep was related to memory improvement in both groups, but in opposite directions. In sum, the role of sleep in memory consolidation changes with aging and WM may play a role in this process.

Support: Fenn et al..2012

0120

ASSOCIATION BETWEEN SLEEP DURATION AND DAYTIME MEMORY AND COGNITION DEPENDS ON **SLEEP QUALITY: DATA FROM THE 2017 ISRAEL SOCIAL SURVEY**

Wills, C. C.¹ Rosenberg, E. A.² Perlis, M. L.³ Parthasarathy, S.¹ Chakravorty, S.³ Grandner, M. A.¹

¹University of Arizona, Tucson, AZ, ²Israel Ministry of Health, Jerusalem, ISRAEL, ³University of Pennsylvania, Philadelphia, PA.

Introduction: This study examines the relationship between sleep duration, sleep disturbance, and cognitive problems in a representative sample of the Israeli population.

Methods: 7,230 Israelis responded to an Israeli Bureau of Statistics population-based survey of households from the year 2017. All variables were self-reported. Outcome of interest was difficulty with memory/concentration (none, mild, or severe). Predictors included previous month sleep duration (<=5hrs, 6hrs, 7hrs [reference], 8hrs, or >=9hrs) and sleep disturbance (none [reference], mild [1/week], moderate [2-3/week], or severe [>3/week]). Covariates included age, sex, ethnic group, and financial status. Multinomial logistic regressions evaluated the relationships between variables, and posthoc testing identified relationships within specific subgroups.

Results: 72.9% denied cognitive problems, 22.2% reported mild problems, and 4.9% severe problems. In adjusted analyses, Sleep ≤ 5 hrs and $\geq=9$ hrs were associated with mild (RRR=1.39, p<0.0005), (RRR=1.46, p=0.004) and severe (RRR=2.75, p<0.0005), (RRR=3.24, p<0.0005) cognitive problems, respectively. Mild, moderate, and severe sleep difficulties were associated with mild cognitive problems (RRR=2.09, p<0.0005), (RRR=2.22, p<0.0005), (RRR=2.44, p<0.0005), and severe cognitive problems (RRR=1.77, p=0.001), (RRR=3.04, p<0.0005), (RRR=4.22, p<0.0005), respectively. There was an interaction between sleep duration and sleep difficulties (p<0.05). Among those denying sleep difficulties, only >=9hrs of sleep was associated with cognitive problems. Among those with mild, moderate, and severe sleep difficulties, both short and long sleep were associated with cognitive problems.

Conclusion: In an Israeli population sample, both sleep duration and quality were associated with cognitive problems. Among those with sleep difficulties, short and long sleep duration were associated with cognitive problems, but among those denying sleep difficulties, only long sleep was associated with cognitive problems. These results suggest that the impact of sleep loss on real-world cognition may also rely on the presence of poor sleep quality. Support: Dr. Grandner is supported by R01MD011600

EFFECT OF TOTAL SLEEP DEPRIVATION ON WORD RECOGNITION OF PREVIOUSLY STUDIED WORDS WITH DIFFERENT EMOTIONAL VALENCE

Hudson, A. N.^{1,2} Whitney, P.^{1,3} Hinson, J. M.^{1,3} Hansen, D. A.^{1,2} Van Dongen, H.^{1,2} Honn, K. A.^{1,2}

¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ³Department of Psychology, Washington State University, Pullman, WA.

Introduction: Stimuli with an emotional valence tend to produce better recognition from memory than neutral stimuli. Sleep loss is believed to increase reactivity to negative stimuli, as compared to positive stimuli, which may comparatively enhance subsequent recognition from memory for negative stimuli. We investigated the impact of total sleep deprivation (TSD) on recognition accuracy for words with different emotional valence using the Affective Item Source Memory Task (AISM).

Methods: N=14 adults (ages 21–39; 7 females) completed a 4-day in-laboratory study with 9h baseline sleep (22:00-07:00), 39h acute TSD, and 9h recovery sleep. The AISM was administered at 16:30 during baseline and after 34h TSD. During a 5min study phase, participants heard a list, twice, of 20 positive, 20 negative, and 20 neutral words spoken with a male or female voice. During an immediately subsequent 8min recognition phase, participants heard 120 words (50% new) and judged whether each word had been presented in the study list (item memory). For words judged to have been presented previously, participants indicated whether those were presented by a female or male speaker (source memory).

Results: Mixed-effects ANOVA showed effects of session (p<0.001) and valence (p<0.001) on item memory. At baseline, accuracy was greatest for neutral and positive words compared to negative words. During TSD, accuracy declined for all three valences, and no interaction of session by valence was detected. No effects of TSD or valence on source memory were observed.

Conclusion: Sleep deprivation reduced item memory for words of all valence types. However, there was no comparatively greater impact on item or source memory for negative words nor any differential effect of TSD for different valences. Whether our results would hold with longer time intervals between task phases or an intervening sleep period remains to be determined.

Support: Jazz Pharmaceuticals

0122

DOES ADDITIONAL TRAINING ON AN IMPLICIT MOTOR SEQUENCE LEARNING TASK FOR OLDER ADULTS IMPROVE SLEEP-DEPENDENT MEMORY CONSOLIDATION?

Rodheim, K. Spencer, R. University of Massachusetts, Amherst, MA.

Introduction: Previous studies show sleep dependent consolidation (SDC) for motor sequence learning with regular training in younger adults, whereas, in older adults, this sleep benefit is missing. If hippocampal engagement underlies age-related differences in SDC, then enhanced training should result in significant SDC in older adults. Thus, this study aims to look at younger vs. older adults with regular training and older adults with regular training vs. overtraining to determine if older adults show evidence of SDC. Alternatively, regardless of initial learning, older adults

may fail to exhibit SDC because the sleep mechanisms supporting consolidation are impaired.

Methods: Seven younger adults (M=22 years) and seven older adults (M=68.5 years) completed the train condition, while ten older adults (M=67.6 years) completed the overtrain condition. In the encoding phase, participants either completed 5 blocks (train) or 10 blocks (overtrain). Between immediate and delayed recall, participants either slept with Polysomnography (PSG) in the lab or remained awake, and subsequently, completed the alternate condition one week later. Actigraphy was collected for 14 days and PSG (32-electrode EasyCap) was recorded for overnight sleep.

Results: Older adults significantly improved their skill learning from immediate to delayed recall, in both the train (p=0.005) and overtrain (p=0.013) conditions, regardless of sleep or wake. Younger adults did not improve their skill learning in the train condition (p>0.05). However, there was a trending main effect, with younger adults performing the task better compared to older adults in the train condition, at both time points (p=0.061). No other main effects or interactions were significant.

Conclusion: These results suggest the alternative hypothesis that, regardless of initial learning, older adults fail to exhibit SDC. This result is of interest as the age-related differences in sleep, such as sleep spindle characteristics may play a role. Future analysis will include more participants and further exploration into the PSG-recorded sleep architecture and actigraphy-recorded measures of habitual sleep.

Support: This work was funded by NIH R01 AG040133 (PI: Spencer)

0123

SLEEP DURATION AND COGNITIVE PERFORMANCE ON THE STROOP COLOR-WORD TASK AND SIMPLE REACTION TIME TASK

Mullins, K. M.¹ Reynolds, A. M.²

¹The University of Virginia's College at Wise, Wise, VA, ²The University of Virginia's College at Wise, Wise, VA.

Introduction: Studies examining sleep factors and cognition suggest that sleep impacts cognitive performance in college students. The focus of the current study was to examine normal sleep patterns in college-aged students and how their sleep affected their cognitive performance.

Methods: Participants were 51 undergraduate students (18 males), average age M=20.25 (SD=1.78) years, who wore actigraph watches to measure their sleep. After one week, participants completed the Multidimensional Assessment of Fatigue (MAF) to assess fatigue and performed a series of cognitive tasks on the computer, including the Stroop Color-Word test. Participants responded to the color of the word presented on the screen instead of the word itself. Stimuli where the color and word did not match were considered incongruent stimuli. Participants also performed a simple reaction time task, where they reacted to an "X" stimulus on the screen.

Results: Mean sleep efficiency was 82.55% (SD=5.70), mean sleep duration was 6.59 hours (SD=79.19 minutes), and the mean MAF score was 21.17 (SD= 7.64). A Pearson correlation indicated a significant negative association between sleep duration and Stroop congruent errors r(49) = -.467, p = .001. Furthermore, a Pearson correlation indicated a significant positive association between sleep duration and incongruent reaction time, r(49)=.290, p= .039 and a significant positive association between sleep duration and simple reaction time, r(49)=.277, p= .049. MAF scores

were positively correlated with simple reaction times, r(49)=.376, p=.008. Sleep efficiency was not correlated with any of the cognitive measures.

Conclusion: As expected, participants' sleep was short and inefficient. Results were expected in that participants made fewer errors with increased sleep, but, unexpectedly, reaction times also increased with more sleep. Fatigue may have played a role in this relationship. It is important to continue this research in order to learn more about sleep factors and cognitive function in college students.

Support: None

0124

A PREFRONTAL-AMYGDALA NETWORK MODEL OF THE CELLULAR AND CIRCUIT-LEVEL MECHANISMS OF EMOTIONAL MEMORY CONSOLIDATION DURING THE AWAKE STATE AND REM SLEEP

Rho, Y. Vijayan, S. Virginia Tech, Blacksburg, VA.

Introduction: Rapid eye movement (REM) sleep has been implicated in the consolidation of emotional memories. Our recent work found a candidate system for REM-related memory consolidation. We showed that during REM sleep, the frontal cortices are dominated by theta (4–8 Hz) oscillations and bursts of beta (15–35 Hz) activity. Studies suggest that rhythmic interactions between the frontal cortices and limbic structures, in particular the amygdala, play a critical role in the consolidation of emotional memories. However, the mechanisms responsible for memory consolidation during these rhythmic interactions during REM sleep remain unknown.

Methods: We used biophysically based neural models to build a large-scale network model of the prefrontal cortex (PFC) and amygdala (AMY) and incorporated synaptic plasticity mechanisms, such as spike-timing dependent plasticity (STDP), into the connections between these two regions. Norepinephrine (NE) and serotonin (SE) levels were manipulated to mimic the different physiological conditions during the awake state and REM sleep.

Results: We were able to reproduce the oscillatory dynamics observed in experimental studies and identify cell-type specific synaptic changes caused by STDP. During the awake state, PFC connections to all cell types of the AMY become strengthened when PFC neurons provide theta frequency inputs, with the connections strengthening to a greater extent when inputs are in burst mode rather than single spike mode. When the PFC provides beta inputs, we see the exact opposite relationship: synaptic strengths become weaker when inputs are in burst mode rather than single spike mode. During REM sleep conditions, the connections to all principal cell types of the AMY become strengthened, with synaptic connections to some subtypes of pyramidal cells becoming stronger than others. Surprisingly, however, the synaptic connections to the interneurons become weaker in response to theta frequency inputs. **Conclusion:** Using our large-scale network model, we show how the levels of the neurotransmitters NE and SE during the awake state and REM sleep affect oscillatory dynamics and in turn influence the strengthening or weakening of connections related to emotional memories.

Support: United States Army Research Office, Award number ARO W911NF-17-1-0300

0125

SLEEP DEPRIVATION IMPAIRS THE ABILITY TO OVERCOME PRE-EXISTING FRAMING BIAS

Honn, K. A.^{1,2} Whitney, P.^{1,3} Hinson, J. M.^{1,3} Nusbaum, A. T.³ Van Dongen, H.^{1,2}

¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ³Department of Psychology, Washington State University, Pullman, WA.

Introduction: When presented with a choice between sure gains or losses versus gambles, people tend to select sure gains over gambles, but gambles over sure losses. This pre-existing framing bias is embedded in the Framed Gambling Task (FGT), in which subjects choose between a sure option (gain or loss) and a gamble (card from one of two decks). For optimal performance, subjects need to recognize that one deck ('good deck') results in better average outcomes than the other deck ('bad deck') and select the gamble or sure option depending on the deck (good/bad) rather than the frame (sure loss/gain). A speeded version of the FGT, with 2s response deadlines to induce time pressure, was used in a laboratory total sleep deprivation (TSD) study to determine the impact of sleep loss on the ability to overcome pre-existing framing bias.

Methods: Eight-six subjects (ages 21-38; 47 females) were randomized (2:1 ratio) to a TSD condition (n=56) or control condition (n=30). They completed the speeded FGT at 11:00 on the baseline day (session 1), and again the following day (session 2) after 27h of wakefulness (TSD group) or 3h of wakefulness (control group). Performance accuracy was defined in terms of optimal task performance, i.e., gambling when the good deck was presented and not gambling when the bad deck was presented. Each test bout had 72 trials across three trial blocks.

Results: Accuracy improved across trial blocks ($F_{1,84}$ =20.44, p<0.001). In session 2, the TSD group showed lower accuracy (condition by session interaction: $F_{2,84}$ =4.15, p=0.045) and less improvement across trial blocks (condition by session by trial block interaction: $F_{2,168}$ =3.97, p=0.021) than the control group. Even under TSD, the frequency of response timeouts (RT>2s) was low (<3.5% of trials).

Conclusion: Sleep deprivation degraded FGT performance under time pressure, indicating reduced ability to overcome pre-existing framing bias.

Support: PRMRP award W81XWH-16-1-0319

LOCAL SLOW WAVE SLEEP AND POST-STROKE BRAIN REPAIR

Landsness, E. C.¹ Brier, L. M.¹ Hua, R. X.¹ Chen, K.¹ Rosenthal, Z. P.¹ Culver, J. P.¹ Lee, J.¹ ¹Washington University St. Louis, Saint Louis, MO, ²Washington University St. Louis, Saint Louis, MO.

Introduction: Recent evidence suggests that slow wave sleep (SWS) is important for synaptic plasticity and brain repair following stroke. Previous studies described a progressive increase in whole cortex and contralesional regional delta power during sleep after stroke, suggesting a global increase in SWS. However, these studies did not distinguish between the effects of global vs. local SWS. We hypothesized that local changes in SWS delta power would parallel changes in the functional remapping and circuit repair.

Methods: To study SWS in living mice we used Thy-1-GCaMP6f mice (n=12), serially imaged (baseline, 24 hours, weeks 1, 4,) during sleep following photothrombotic stroke of the left forepaw somatosensory cortex (S1FP). An optical fluorescence imaging system (OFI) was used to image whole-cortex neuronal activity. The evolution of local delta activity was compared across three ROIs: 1) infarct, 2) perilesional remapped, and 3) perilesional non-remapped left.

Results: The photothrombotic infarct encompassed the left S1FP stimulus map, resulting in significant attenuation of S1FP evoked responses at week 1; however, a small region of activation was retained in posterior left S1FP (peri-lesional remapped). The infarct region demonstrated a decrease in delta power during sleep; however, the perilesional region of future remapping exhibited a rebound in focal delta power at 1 week after an initial decline at 24 hours. In the perilesional non-remapped area delta power decreased, but did not increase until week 4. We also observed an early wide-spread increase in delta power at 24 hours and week 1 that decreased on week 4.

Conclusion: With the high spatial resolution of OFI, we find that SWS is disrupted throughout the brain following focal ischemia. These data suggest that local SWS selectively increases in the region of remapping prior to repair of that circuit and that local SWS may play a role in brain repair following stroke.

Support: AASM Foundation #183-PA-18, #201-BS-18

0127

RELATIONSHIP BETWEEN SLEEP METRICS WITH FREE-LIVING GLUCOSE CONCENTRATIONS AND GLYCEMIC VARIABILITY IN NON-DIABETIC ADULTS

Sparks, J. R. Kishman, E. E. Wang, X.

Department of Exercise Science-University of South Carolina, Columbia, SC.

Introduction: Insufficient sleep and poor sleep quality have been associated with impaired glucose metabolism at fasting and under experimental conditions. Continuous glucose monitoring (CGM) measures glucose concentrations over an extended, free-living period that can be used to assess glycemic health. Relationships between CGM-assessed glucose concentrations and glycemic variability, an emerging glycemic health marker, with sleep metrics have yet to be elucidated. The purpose of this study was to examine the relationships between sleep metrics with glucose concentrations and glycemic variability in non-diabetic adults.

Methods: Twenty-four non-diabetic adults (age= 46.0 ± 5.8 years; BMI= 32.2 ± 5.7 kg/m²) completed actigraphy, sleep diary, and CGM over 7 consecutive days. Time-in-bed (TIB), total sleep time (TST), wake duration after sleep onset, and sleep efficiency [(TST÷TIB)×100%] were determined using actigraphy assisted with sleep diary input. Nightly variability of each sleep metric was calculated as standard deviation (SD) across all nights. Glucose concentrations at waking in the morning, and 1, 2, and 3 hours prior to waking, and diurnal, nocturnal, and 24-hour means were determined. Intra-day glycemic variability, including mean amplitude of glycemic excursions and continuous overlapping of net glycemic action of 1, 2, and 4 hours, and inter-day glycemic variability, mean of daily differences, were calculated. Pearson product correlations between sleep metrics with glucose concentrations and glycemic variability were performed.

Results: Average TIB and TST were 462.6 ± 61.7 minutes and 403.3 ± 59.7 minutes, respectively. TIB negatively correlated with glucose concentrations at 2 and 3 hours prior to waking (r=-0.42, p=0.04, and r=-0.42, p=0.04, respectively). Nightly variability in sleep efficiency positively correlated with waking, and 1, 2, and 3 hours prior to waking glucose concentrations ($0.44 \le x \le 0.48$, p ≤ 0.03 for all). No sleep metrics correlated with glycemic variability measures (p ≥ 0.10 for all).

Conclusion: Findings suggest a longer amount of sleep opportunity and more consistent sleep efficiency relate to better glucose metabolism in non-diabetic adults.

Support: American Heart Association 14BGIA20380706 and University of South Carolina Support to Promote Advancement of Research and Creativity Grant #11530-17-43917.

0128

MILD UPPER AIRWAY OBSTRUCTION LEADS TO INCREASED ENERGY INTAKE AND GROWTH RETARDATION THAT PERSISTS AFTER THE OBSTRUCTION REMOVAL

Rotenberg, A.¹ Assadi, M.¹ Agam, N.¹ Segev, Y.¹ Tarasiuk, A.² ¹Ben-Gurion University of the Negev., Beer-Sheva, ISRAEL, ²Ben-Gurion University of the Negev, Beer-Sheva, ISRAEL.

Introduction: Whereas pediatric obstructive sleep apnea may cause insufficient body weight gain and growth retardation, in some studies, metabolic syndrome and obesity were observed. Interestingly, treatment by adenotonsillectomy can lead to accelerated weight gain by an unclear mechanism. Here, we explored the effects of moderate upper airway obstruction (AO) and mild AO (mAO) and its removal (OR) on ventilation, resting energy expenditure (REE), food intake and growth during the diurnal cycle, from weaning to adulthood.

Methods: The trachea of 22-day-old rats was surgically narrowed to generate AO, mAO, and OR was performed after two weeks on mAO animals. Minute ventilation was recorded by whole body plethysmography and diurnal food intake, and REE was explored with metabolic cages 12 weeks post surgery.

Results: Following tracheal narrowing, inspiratory swings in esophageal pressure increased by 177% (p<0.01) and 36% (p<0.01) in AO and mAO rats, respectively, and was similar to the controls in the OR group. REE (Kcal/h/kg) was 3.7±0.1, 5.7±0.12 (p<0.01), 4.1±0.08 (p<0.01), and 3.6±0.15 in the control, AO, mAO, and OR groups, respectively. Increased EE in the AO and mAO groups was associated with up-regulation of ventilation by 133% and 56%, respectively (p<0.01). In all groups, energy intake (EE) was higher during a 12 h active period compared to a sleep period (p<0.01). EE during the lights on of AO and mAO animals increased by 136% and 126%, respectively, and was similar to the control in the OR group. Active

VII. Physiology

period EE increased by 19% in both obstructed groups (p<0.01). Active period EE was 16.7% higher in the OR group despite the normalization of ventilation and tracheal diameter to the control value. Increased REE was associated with hindrance of bone elongation (p<0.01), and OR partially improved growth.

Conclusion: The need to maintain respiratory homeostasis during upper airway obstruction was associated with a persistent increase in energy intake. Surgical intervention may not be sufficient to correct the energy intake elevation, and endocrine regulation of feeding and growth may have greater impacts post intervention.

Support: This study was supported by the Israel Science Foundation grant no. 164/2018

0129

ASSOCIATION OF SLEEP QUALITY WITH SERUM LIPIDS IN OBESE ADULTS

Mehta, B. Ankita, A. Raghav, P. Chambial, S. Dutt, N. AIIMS, Jodhpur, INDIA.

Introduction: Sleep disturbances have been associated with metabolic dysregulation and have known to contribute to weight gain, obesity, type II diabetes, and cardiovascular disease risk. Obesity due to sleep fragmentation is mediated by multiple pathways like upregulation of orexin neurons and changes in appetite-regulating hormones like Leptin, Ghrelin, which affect food intake and hedonic feeding. Conversely, body mass index (BMI) is associated with alterations in sleep and with high circulating lipids and incidence of coronary heart disease. We hypothesized that poor sleep quality is associated with an adverse serum lipid profile.

Methods: In this cross-sectional study, till date, 27 obese adult participants were recruited after informed consent. The obesity criterion was taken as BMI \geq 25 kg/m². Anthropometric parameters, waist circumference, neck circumference, hip circumference, and BP were measured. Sleep quality was assessed by the "Pittsburgh Sleep Quality Index" (PSQI) questionnaire. A score of 5 or more was considered to be adverse sleep. The fasting blood sugar and lipid profile of each participant was determined.

Results: The average age, BMI and waist circumference of the subjects were 48.96 \pm 13.9 years, 32.41 \pm 6.18 kg/m² and 107.4 \pm 12.18 cm respectively. The Spearman correlation test revealed a significant correlation between the PSQI scores and triglyceride levels of the participants (p=0.033, r = 0.420). The correlation with BMI (p=0.33, r=0.192), fasting blood sugar (p=0.26, r=0.241), HDL (p=0.27, r = -0.221) and waist circumference (p=0.69, r = -0.082) were not found to be statistically significant.

Conclusion: We conclude that high triglyceride levels are associated with poor quality of sleep in adults. Although other biochemical parameters did not show a significant correlation, a greater sample size may give us a clear insight into it.

Support: The study is an intramural project supported by AIIMS, Jodhpur.

0130

BEYOND TRADITIONAL RISK FACTORS: SLEEP METRICS ARE ASSOCIATED WITH ARTERIAL STIFFNESS IN HEALTHY YOUNG ADULTS

Katulka, E. K.¹ Patterson, F.² Berube, F. R.¹ D'Agata, M. N.¹ Farquhar, W. B.¹ Edwards, D. G.¹ Witman, M. A.¹

¹Department of Kinesiology and Applied Physiology, University of Delaware, Newark, DE, ²Department of Behavioral Health and Nutrition, University of Delaware, Newark, DE.

Introduction: Studies in middle-age and older populations have found sleep metrics (i.e., sleep duration) to be an emergent determinant of cardiovascular (CV) health. The extent to which sleep habits relate to CV health in young adults is not yet known. Arterial stiffness is considered an indicator of subclinical atherosclerosis and is predictive of future CV events. Therefore, we aimed to evaluate the associations between traditional CV factors, sleep metrics, and arterial stiffness in young adults.

Methods: 51 healthy young adults $(20.4\pm1 \text{ years}, 20\text{M}/31\text{F})$ wore wrist accelerometers 24h/day for 14 consecutive days to estimate free-living sleep metrics. Carotid-femoral pulse wave velocity (PWV), a measure of arterial stiffness, was performed the morning immediately following completion of sleep monitoring. The American Heart Association's "Life's Simple 7" score (LS7) was calculated for each participant, reflecting an index of CV health when considering seven traditional risk factors (blood pressure, body mass index, cholesterol, fasting glucose, smoking, physical activity, diet). Linear regression modeling was used to evaluate the association between LS7 and PWV with and without consideration of various sleep metrics.

Results: In a regression model of PWV that included sex and LS7, the association between LS7 and PWV remained a trend ($R^2=0.11$, $\beta=-0.28$, p=0.06). However, individually added sleep metrics such as sleep efficiency ($\Delta R^2=0.07$, $\beta=-0.29$, p=0.05), sleep onset latency ($\Delta R^2=0.09$, $\beta=0.32$, p=0.03), and sleep duration standard deviation ($\Delta R^2=0.14$, $\beta=0.38$, p<0.01) were significantly associated with PWV and improved the model, accounting for an additional 7–14% of variability in PWV.

Conclusion: Various sleep metrics, independent of LS7 scores, were significant determinants of arterial stiffness in this sample of apparently healthy young adults. This suggests that sleep may contribute to CV health in early adulthood and that maintaining healthy sleep habits could assist in reducing the risk of future disease development.

Support: Provided in part by NIH P20GM113125.

0131

DECREASED HABITUAL SLEEP EFFICIENCY IS ASSOCIATED WITH INCREASED INSULIN RESISTANCE IN HEALTHY ADULT MEN

Kelly, M. R.¹ O'Byrne, N.² Iranmanesh, A.³ Martin, J. L.¹ Liu, P. Y.²

¹VA Greater Los Angeles Healthcare System, Los Angeles, CA, ²Harbor UCLA Medical Center and Lundquist Institute, Torrance, CA, ³Salem Veterans Affairs Medical Center, Salem, VA.

Introduction: Partial sleep deprivation is associated with increased insulin resistance (IR), a metabolic disease risk marker. Little is known about habitual sleep patterns and IR in the absence of acute sleep restriction. We anticipated greater change in habitual sleep over one month would be associated with increased IR.

Methods: 24 males (age=33.6±6.4 years; BMI=25.7±2.5kg/m²) completed baseline (T1) and follow-up (T2; ≥4 weeks post-T1) study procedures: actigraphy (one week) followed by polysomnography (PSG; one 10h sleep opportunity) and a next morning oral glucose tolerance test (OGTT; homeostatic model assessment insulin resistance [HOMA-IR], β-cell function [HOMA-β], and Matsuda Index). Weekly average actigraphy total sleep time (aTST; 291-511min) and sleep efficiency (aSE; 72–93%) were computed at T1 and T2, as well as across the 1, 2, and 3 days prior to PSG/OGTT. Pearson and Spearman correlations assessed the change (T1-T2) in actigraphy (aSE∆, aTST∆, PSG∆) or PSG sleep (PSG-TST∆,

PSG-SE Δ , sleep stages) versus change in metabolic risk (HOMA-IR Δ , HOMA- $\beta\Delta$, Matsuda Δ).

Results: There were significant correlations between HOMA-IR Δ and aSE Δ [r(22)=-0.42, p=0.01; r_s =-0.45, p=0.03], PSG TST Δ [r(22)=0.50, p=0.012; r_s =0.41, p=.045], and PSG-SE Δ [r(22)=0.49, p=0.015; r_s =0.43, p=.037]. No significant associations emerged between change in metabolic risk versus aTST Δ one week prior to PSG/OGTT, aSE Δ or aTST Δ across 1–3 days prior to PSG/OGTT, or PSG sleep stages.

Conclusion: Within-subject T1-T2 decrease in habitual sleep quality, but not TST, was associated with increased IR. T1-T2 PSG TST and SE were associated with following day IR. At home sleep 1–3 days beforehand were not correlated with IR. Although preceding night sleep quality and TST are associated with IR, habitual sleep quality, rather than TST, may be a more important determinant of metabolic risk in community dwelling middle-aged men. **Support:** This work was supported by NIH/NHLBI R01HL124211, NIH/NHLBI K24HL138632, NIH National Center for Advancing Translational Sciences (NCATS) UCLA CTSI Grant UL1TR001881 (PI: Liu); and NIH/NHLBI K24HL143055 (PI: Martin). Dr. Kelly is supported by the VA Office of Academic Affiliations through the Advanced Fellowship Programs in Geriatrics.

0132

SEX DIFFERENCES IN EVENING FOOD INTAKE AND ASSOCIATED WEIGHT GAIN DURING INSUFFICIENT SLEEP

Withrow, D.¹ Depner, C. M.¹ Boland, E. M.¹ Birks, B. R.¹ Melanson, E. L.^{2,3} Higgins, J.⁴ Eckel, R. H.² Perreault, L.² Bergman, B. C.² Wright Jr., K. P.¹

¹Sleep and Chronobiology Laboratory, Department of Integrative Physiology, University of Colorado, Boulder, CO, ²Division of Endocrinology, Metabolism, and Diabetes, University of Colorado Anschutz Medical Campus, Aurora, CO, ³Division of Geriatric Medicine, University of Colorado Anschutz Medical Campus, Aurora, CO, ⁴Department of Pediatrics, University of Colorado Anschutz Medical Campus, Aurora, CO.

Introduction: Timing of food intake has emerged as a novel risk factor for weight gain and obesity. Higher evening food intake, especially during insufficient sleep, is associated with weight gain. We aimed to explore initial changes in evening food intake and the respiratory quotient (RQ) during insufficient sleep and subsequent weight gain. We also explored sex differences.

Methods: 28 healthy adults (14F) aged 26.3 ± 4.5 y completed a 14–16 daylong laboratory protocol. In their home environment participants maintained one week of ~9h/night sleep schedules and consumed energy balanced diets for 3 days prior to completing the laboratory protocol. The laboratory protocol consisted of 3 baseline days of 9h/night scheduled sleep with energy balanced diets followed by 10 days of 5h/night scheduled sleep with ad-libitum food intake, with (n=14) and without (n=14) weekend recovery sleep. RQ was assessed on days 3 and 5 in a whole room calorimeter. Evening (dinner and after-dinner snacks) energy intake and body weight were assessed daily.

Results: A significant sex by condition effect was observed for evening food intake such that men and women were similar at baseline, but men ate more than women during insufficient sleep, when controlling for body mass (p<0.05). A significant sex by condition

effect was also observed for RQ with women showing similar RQ during baseline and insufficient sleep and men showing a higher RQ during insufficient sleep versus baseline (p<0.05). Linear regression with food intake and RQ as predictors of weight gain showed that increased evening food intake, but not RQ, on the second day of sleep restriction was associated with weight gain in men, but not women, at the end of the study eight days later (p<0.05).

Conclusion: Findings suggest that rapid changes in evening food intake during insufficient sleep contributes to subsequent weight gain during sustained insufficient sleep, especially in men.

Support: NIH HL109706, DK111161, TR001082, DK048520, Sleep Research Society Foundation grant 011-JP-16 and Office of Naval Research MURI (N00014-15-1-2809).

0133

NLRP3 INFLAMMASOMES MODULATE BRAIN VASOHEMODYNAMIC RESPONSES TO SLEEP LOSS

Zielinski, M. R.¹ Atochin, D. N.² Desrosiers, G.³ ¹Harvard Medical School and Boston VA Healthcare System, West Roxbury, MA, ²Cardiovascular Research Center, Massachusetts General Hospital, Charlestown, MA, ³VA Boston Healthcare System, West Roxbury, MA.

Introduction: Increased cerebral blood flow (CBF) is positively associated with non-rapid eye movement (NREM) sleep electroencephalogram (EEG) delta power, also known as slow-wave activity (SWA). The pro-inflammatory somnogenic cytokine interleukin-1 beta (IL-1 β) can induce vasodilation and increase CBF. Nucleotide leucine-rich protein complex-3 (NLRP3) inflammasomes, which activate IL-1 β , are increased in the cortex after sleep deprivation and increase SWA. We aimed to determine the relationship of NLRP3 inflammasomes on sleep loss-induced alterations in vasohemodynamics and SWA.

Methods: NLRP3 knock-out (KO) and wild-type (WT) mice underwent ad libitum sleep, 6 hours of sleep deprivation, or were given 10 ng of IL-1 β or the vehicle intracerebroventricularly. SWA and CBF, blood velocity, and blood volume were determined concurrently during sleep/wake states using polysomnography and laser doppler flowmetry. Regional brain changes in CBF were determined using transponders, spectrophotometry, and fluorescent microspheres.

Results: SWA and CBF were significantly increased during the first 6 hours after sleep deprivation in WT but not NLRP3 KO mice. SWA and CBF significantly increased in the first 6 hours after IL-1ß in both NLRP3 KO and WT mice. Additionally, alterations in cerebral blood velocity and volume demonstrated state specific changes that varied significantly during the transitions between states. SWA and CBF were significantly positively correlated during both ad libitum sleep and sleep after sleep deprivation in WT mice, although this relationship was not observed in NLRP3 KO mice. We also found significant phase-amplitude frequency coupling between SWA and CBF. Cortical changes CBF were significantly enhanced after sleep deprivation and IL-1ß administration in WT mice, although were attenuated in the hypothalamus. NLRP3 KO mice showed these same regional effects in CBF after IL-1 β but not sleep deprivation.

Conclusion: Our findings indicate that NLRP3 inflammasomes are involved in neurovascular coupling involving SWA.

Support: Department of Veterans Affairs IBX002823A (MRZ)

SELF-REPORTED SLEEP IS ASSOCIATED WITH CENTRAL, BUT NOT PERIPHERAL BLOOD PRESSURE VALUES IN HEALTHY CHILDREN

Berube, F. R.¹ Katulka, E. K.¹ D'Agata, M. N.¹ Patterson, F.² Ives, S. J.³ Farquhar, W. B.¹ Witman, M. A.¹

¹Department of Kinesiology and Applied Physiology, University of Delaware, Newark, DE, ²Department of Behavioral Health and Nutrition, University of Delaware, Newark, DE, ³Skidmore College, Saratoga Springs, NY.

Introduction: Shortened and poor quality sleep have emerged as nontraditional risk factors for the development of high blood pressure (BP) in adults, but it is unclear if these relations exist in younger children. Self-report and objective sleep measurements are both clinically relevant and may inform interventions to improve sleep in this population, but do not always coincide with one another. The purpose of this study was to evaluate both self-reported and objective sleep metrics and their associations with central and peripheral BP values in younger children.

Methods: Participants included 21 healthy 7-12-year-old children (10±0.5 yrs, 10M/11F). Self-reported sleep was evaluated using the Children's Sleep Health Questionnaire and a total sleep score was generated, where a higher score indicates worse sleep (a score >41 indicates a pediatric sleep disorder). Objective sleep was recorded for 7 consecutive days and nights outside of the laboratory via wrist accelerometry and reported as sleep duration (SD) and sleep efficiency (SE). Following sleep monitoring, peripheral BP was measured and using pulse wave analysis (PWA) central BP was estimated, both of which were averaged over 3 trials. Pearson's *r* correlations were used to assess relations between self-reported sleep score, objective sleep metrics, and BP values. Significance was set at p<0.05.

Results: Self-reported sleep score averaged 40±1 points, objective SD averaged 7.9±0.1 hours/night, and SE averaged 82±2%. Sleep score was significantly associated with central systolic and diastolic BP (r = .485, p = 0.03, and r = .517, p = 0.02, respectively), but not peripheral BP values. Objective SD and SE were not significantly associated with central or peripheral BP values.

Conclusion: In this sample, self-reported sleep score, but not objective sleep metrics, was associated with higher central BP values in healthy children age 7–12.

Support: Provided in part by P20GM113125.

0135

SELF-REPORTED SLEEP AND GUT MICROBIOME COMPOSITION AND DIVERSITY: ASSOCIATIONS IN WELL-FUNCTIONING OLDER ADULTS

Holingue, C.¹ Mueller, N. T.² Tanaka, T.³ Differding, M. K.²
Chia, C. W.³ Wu, M. N.⁴ Schrack, J. A.² Simonsick, E. M.³ Spira, A. P.⁵
¹Department of Neuropsychology, Kennedy Krieger Institute,
Baltimore, MD, ²Department of Epidemiology, Johns Hopkins
Bloomberg School of Public Health, Baltimore, MD, ³National Institute
on Aging, Bethesda, MD, ⁴Department of Neurology, Johns Hopkins
School of Medicine, Baltimore, MD, ⁵Department of Mental Health,
Johns Hopkins Bloomberg School of Public Health, Baltimore, MD.

Introduction: The gut microbiome is believed to play an important role in health and disease, yet little is known about the link between sleep and the gut microbiome in humans. We investigated

the association of self-reported sleep with gut microbiome composition and diversity in a cohort of well-functioning older adults. **Methods:** We studied 791 participants (mean age = 71.5 ± 12.0 years, 55% women) in the Baltimore Longitudinal Study of Aging with self-report sleep measures and whole-genome DNA sequencing of stool samples. Predictors (modeled as continuous variables) included insomnia symptoms from the Women's Health Initiative Insomnia Rating Scale (WHIIRS), sleep duration (<5, 5–6, 6–7, >7 hours), and frequency of excessive daytime sleepiness (EDS). We tested their association with gut microbiome diversity (Shannon index) and relative abundance of individual taxa using Kendall Tau Correlation. Next, we assessed whether these sleep variables were associated with overall microbiome structure (Bray-Curtis), adjusting for age, sex, race, education, BMI, depressive symptoms, and number of comorbidities.

Results: Sleep duration was associated with overall microbiome composition (p<0.01), with longer sleep duration associated with lower biodiversity of microbes in the gut (p<0.05). In phylum-level analyses, higher WHIIRS total (i.e., more severe insomnia) was associated with lower relative abundance of Actinobacteria, while more frequent EDS was associated with lower relative abundance of Fusobacteria. More frequent trouble falling asleep, staying asleep, early waking, poorer sleep quality and higher WHIIRS total were associated with lower abundance of Synergistetes (all p<0.05).

Conclusion: In well-functioning older adults, self-reported sleep duration, symptoms of insomnia, and EDS were associated with microbiome diversity and composition. The phylum Synergistetes, which has been associated with protective humoral immune response in prior literature, may be an important correlate of insomnia symptoms in older adults. Future investigations are needed to examine the gut microbiome as a driver or mediator of sleep-health associations. **Support:** This study was supported in part by National Institute on Aging (NIA) grant R01AG050507, the NIA Intramural Research Program (IRP), and Research and Development Contract HHSN-260-2004-00012C.

0136

BRIGHT LIGHT COULD BE AN ALTERNATIVE TO THE CAFFEINE FOR IMPROVING DRIVING PERFORMANCE IN CHRONICALLY SLEEP-DEPRIVED YOUNG DRIVERS Shekari Soleimanloo, S.¹ Garcia-Hansen, V.² White, M.³ Smith, S. S.¹ ¹Institute for Social Science Research, The University of Queensland, QLD, Brisbane, AUSTRALIA, ²School of Design, Queensland University of Technology, QLD, Brisbane, AUSTRALIA, ³School of Psychology and Counselling, Queensland University of Technology, QLD, Brisbane, AUSTRALIA.

Introduction: Young drivers are over-involved in sleepiness-related crashes. Daytime and nighttime exposure to light might shift the human circadian phase and alertness. The alerting effects of bright light were compared with those of caffeine in young drivers. Methods: In a within-subjects study, 30 chronically sleep-deprived non-professional drivers (aged 18-24 years) completed two simulated daytime driving sessions per day across three consecutive days. Participants completed the first drive under a Baseline condition (non-caffeinated gum, 555 nm light, 0.3 μ W/cm²), and the second drive under the randomized conditions of Light (500 nm, 230 µW/ cm²), Caffeine (100 mg caffeinated gum) or the combination of Light and Caffeine. Using mixed-effects models, the alerting effects of these conditions on objective sleepiness (ECG beat-to-beat intervals), driving performance (lateral lane variability) and subjective sleepiness (scores on the Karolinska sleepiness scale; KSS) were examined.

Results: Compared to the Baseline condition, lateral lane variability decreased under the Light (P=0.011), Caffeine (P=0.0001), and the combination of Light and Caffeine (P=0.046). Lateral lane variability was lower under Caffeine when compared with the Light (P=0.009) or the combination of Light and Caffeine (P=0.0001). Average beat-to-beat intervals increased from the Baseline condition to the Light (P=0.017), Caffeine (P=0. 0.0001), and the combination of Light and Caffeine conditions (P=0.0001). All three conditions significantly reduced subjective sleepiness compared to the Baseline condition (KSS=4-5 vs KSS=6, P= 0.0001).

Conclusion: Bright light, either alone or combined with caffeine, improves driving performance and subjective sleepiness during daytime drives. Light might better improve objective sleepiness and other sleepiness indicators during nighttime when drivers are sleepier and have an increased sensitivity to the light. Further research would clarify how the circadian effects are aligned with the alerting effects of the light. Bright light, as an alternative to or combined with caffeine, could reduce sleep-related crashes on the road. Support: NA

0137

SELF-REPORTED SLEEP QUALITY IS ASSOCIATED WITH CENTRAL HEMODYNAMICS IN HEALTHY **INDIVIDUALS**

Culver, M. N.¹ Flatt, A. A.² Grosicki, G. J.²

¹Georgia Southern University, Savannah, GA, ²Georgia Southern University-Armstrong Campus, Savannah, GA.

Introduction: Insufficient sleep is associated with arterial stiffness and elevated cardiovascular disease risk. Central hemodynamics are influenced by arterial stiffness, yet independently predict cardiovascular risk. Relationships between sleep characteristics and central hemodynamic parameters are largely unexplored. We aimed to characterize the relationship between self-reported sleep quality and central hemodynamics in healthy individuals. To explore the hypothesis that impairments in glucose metabolism, resulting from lack of sleep, may underlie relationships between sleep and central hemodynamic variables, we also examined associations between self-reported sleep quality and fasting blood glucose values.

Methods: Thirty-one healthy subjects (14 females /17 males; 20-69 years) that were free from metabolic or cardiovascular disease, and that did not take sleep medication were included in the study. Relationships between self-reported sleep quality, obtained using the Pittsburgh Sleep Quality Index (PSQI), with central hemodynamic profiles(systolic and diastolic blood pressures, pulse and augmentation pressures, augmentation index) estimated from oscillometric pulse wave analysis, and fasting blood glucose values were assessed.

Results: Central pulse pressure was significantly elevated (P<0.05) in poor (PSQIscore >5) compared to normal (PSQI score 0-5) self-reported sleepers. Linear regression models, adjusted for age, gender, and body mass index, demonstrated PSQI score to be an independent predictor (P<0.05) of both central pulse (β =0.469) and augmentation (β =0.364) pressures. Global PSQI scores were not related to fasting blood glucose values (r=0.045; P>0.05).

Conclusion: Significant relationships between central pulse and augmentationpressures and self-reported sleep quality highlight the importance of considering sleep when examining lifestyle contributors to central hemodynamics.

Support: No funding.

0138

BEHAVIORALLY ASSESSED SLEEP DURATION AND **OXIDATIVE STRESS**

Decker, A. Cribbet, M. University of Alabama, Tuscaloosa, AL.

Introduction: Sleep may promote health by acting as an antioxidant, thereby increasing the body's resistance to oxidative stress. Lipid peroxidation, a marker of oxidative stress, is one of the key early events in the development of atherosclerosis, the pathologic condition that underlies cardiovascular disease (CVD). Short sleep duration is prevalent in the general population and associated with CVD risk. However, the mechanisms linking short sleep to CVD are not well understood.

Methods: To test the hypothesis that short sleep duration would be associated with higher levels of oxidative stress, we conducted secondary data analysis on a diverse sample of participants (N= 81; M₂₀₀ = 30.1(SD=10.9); 57% Male; 27% African American) from Pittsburgh, Pennsylvania. Participants wore actigraphs on their nondominant wrists for 7 days to collect rest and activity data, which were used to derive average sleep duration. Participants provided urine samples and self-reported demographic data, along with daily mood, alcohol use, and physical activity. Oxidative stress was quantified as urinary concentrations of 15-F2t-isoprostane (15-F2-IsoP). Levels of 15-F2-IsoP were measured with a competitive enzyme linkedimmunosorbent assay (ELISA) in duplicate. Urinary 15-F2-IsoP was adjusted by urinary creatinine to create an index of oxidative stress that took into account a measure of antioxidant defenses.

Results: Sleep duration was inversely correlated with oxidative stress (b = -.24, p = .03). In multiple regression analyses that controlled for age, sex, race, body mass index, alcohol use, physical activity, and depressed mood, sleep duration remained significantly negatively associated with oxidative stress, $\beta = -.16$, t = -2.33, p = .02, R²= .20.

Conclusion: These findings are important because they suggest that a relationship exists between short sleep and increased oxidative damage to lipids. Increasing our understanding of this relationship could motivate new CVD prevention strategies that target the inhibition of oxidative stress through sleep interventions.

Support: This study was supported by a grant from the National Center for Complementary and Integrative Health (AT006694); a grant from the National Institute of Allergy and Infectious Diseases (R01 AI066367); grants from the National Institutes of Health (UL1 RR024153 and UL1 RT000005); and by the John D. and Catherine T. MacArthur Foundation.

0139

ASSOCIATIONS OF ACTIGRAPHIC SLEEP PARAMETERS WITH MAXIMAL OXYGEN CONSUMPTION AND RESTING METABOLISM IN WELL-FUNCTIONING OLDER ADULTS

Alfini, A. J.¹ Wanigatunga, A. A.² Schrack, J. A.² Wanigatunga, S.¹ Li, J.³ Rojo-Wissar, D. M.¹ Mehta, R.¹ Okoye, S.⁴ Zipunnikov, V.⁵ Simonsick, E. M.⁶ Spira, A. P.¹

¹Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ²Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ³Johns Hopkins University School of Nursing, Baltimore, MD, ⁴Department of Health Policy and Management, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ⁵Department of Biostatistics, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ⁶Division of Geriatric Medicine and Gerontology, Department of Medicine, Johns Hopkins School of Medicine, Baltimore, MD.

Introduction: Both poor sleep and poor cardiorespiratory fitness are common in older age and associated with negative health outcomes. Additionally, among older adults, higher resting metabolic rate (RMR) has been associated with increased morbidity and mortality risk. To evaluate whether, and in what ways, sleep may affect these relationships, we investigated the association of actigraphic sleep indices with cardiorespiratory fitness and RMR in older adults.

Methods: We studied 393 community-dwelling participants in the Baltimore Longitudinal Study of Aging (mean age 73.5±10.3 years, 52% women) who completed 6.7±0.9 nights of wrist actigraphy, RMR testing, and a maximal graded exercise test. Primary predictors included mean actigraphic total sleep time (TST, minutes), sleep efficiency (SE, %), wake after sleep onset (WASO, minutes), and average wake bout length (WBL, minutes). Cardiorespiratory fitness, as measured by maximal oxygen consumption (V O_{2MAX}; ml/kg/min), and RMR (kcal/day) were the primary outcomes.

Results: After adjustment for age, sex, race, body mass index, comorbidity index, and depressive symptoms, longer WBL was associated with lower V O_{2MAX} (β =-0.12, 95% confidence interval (CI)=-0.20, -0.04), greater WASO was associated with lower V O_{2MAX} (β =-0.09, 95% CI=-0.17, -0.01), and greater SE was associated with higher V O_{2MAX} (β =0.12, 95% CI=0.03, 0.20). In addition, longer TST was associated with lower RMR (β =-0.10, 95% CI=-0.19, -0.01) and longer WBL was linked to higher RMR (β =0.12, 95% CI=-0.12, 95% CI=-0.04, 0.21).

Conclusion: In well-functioning older adults, indices of greater wakefulness after sleep onset are linked with poorer cardiorespiratory fitness and higher resting metabolism, while longer and more efficient sleep are associated with better fitness and lower resting metabolic rate. Our findings suggest that sleep disturbance may be linked to disrupted energy homeostasis, evidenced by excessive energy expenditure at rest and inefficient energy utilization in response to maximal demands. Prospective analyses are necessary to determine the nature of these associations.

Support: This study was supported in part by National Institute on Aging (NIA) grants R01AG050507 and T32-AG027668, the NIA Intramural Research Program (IRP), and Research and Development Contract HHSN-260-2004-00012C.

0140

DIETARY MACRONUTRIENTS AND SLEEP DURATION, SLEEP DISTURBANCE, AND DAYTIME FATIGUE

Barker, M.¹ St-Onge, M.² Seixas, A.³ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹

¹University of Arizona, Tucson, AZ, ²Columbia University, New York, NY, ³New York University, New York, NY.

Introduction: We examined nationally-representative data on macronutrients associated with multiple dimensions of sleep health.

Methods: Data were obtained from the 2015–2016 National Health and Nutrition Examination Survey, (N=5,266 adults). Standard 24-h dietary recall procedures were analyzed to establish daily consumption of protein, carbohydrates, sugar, fiber, total fat, and saturated fat. Self-reported habitual sleep duration was categorized as very short (<5h), short (5–6.5h), normal (7-8h), and long (>8h). Sleep disturbance and daytime tiredness/fatigue were self-reported as either none, mild, moderate, or severe. Weighted multinomial logistic regressions with sleep variables as outcome/dependent variable and percent of each macronutrient as independent variable were adjusted for age, sex, race/ethnicity, education, and body mass index.

Results: Increased protein was associated with a decreased likelihood of very short sleep (RRR=0.01, p=0.019) and severe fatigue (RRR=0.06, p=0.020). Increased carbohydrates was associated with an increased likelihood of very short (RRR=61.17, p=0.001), short (RRR=3.96, p=0.017), and long (RRR=2.58, p=0.041) sleep, severe sleep disturbance (RRR=9.37, p=0.010) and fatigue (RRR=7.61, p=0.009). Increased sugar was associated with an increased likelihood of very short (RRR=24.17, p=0.001), short (RRR=3.29, p=0.017), and long (RRR=2.22, p=0.046) sleep, as well as mild (RRR=2.36, p=0.041) and severe (RRR=10.70, p=0.001) sleep disturbance, and severe fatigue (RRR=12.98, p<0.0005). Increased fiber was associated with a decreased likelihood of long (RRR=0.01, p=0.032) sleep and severe sleep disturbance (RRR<0.01, p<0.0005), as well as moderate (RRR<0.01, p=0.026) and severe (RRR<0.01, p<0.0005) fatigue. Increased fat was associated with a decreased likelihood of very short sleep (RRR=0.01, p=0.010). Increased saturated fat was associated with a decreased likelihood of very short sleep (RRR<0.01, p=0.017).

Conclusion: Protein and fiber were associated with better sleep profiles overall and carbohydrate and sugar were associated with worse sleep, as well as increased prevalence of sleep disturbances and fatigue.

Support: Dr. Grandner is supported by R01MD011600

A NOVEL, ORALLY AVAILABLE OREXIN 2 RECEPTOR-SELECTIVE AGONIST, TAK-994, SHOWS WAKE-PROMOTING EFFECTS FOLLOWING CHRONIC DOSING IN AN OREXIN-DEFICIENT NARCOLEPSY MOUSE MODEL

Ishikawa, T. Suzuki, M. Kimura, H.

Takeda Pharmaceutical Company Limited, Fujisawa, Kanagawa, JAPAN.

Introduction: The use of an orexin 2 receptor (OX2R) agonist may be a promising approach for the treatment of narcolepsy type 1. TAK-994 is a novel, orally available OX2R-selective agonist with >700-fold selectivity against orexin 1 receptor. Single administration of TAK-994 ameliorates narcolepsy-like symptoms such as fragmentation of wakefulness and cataplexy-like episodes in orexin/ataxin-3 mice, a narcolepsy mouse model with orexin deficiency. In this study, we evaluated the effect of chronic dosing with TAK-994 on sleep/wakefulness states in orexin/ataxin-3 mice.

Methods: Orexin/ataxin-3 mice were grouped into two cohorts: a control group and a 14-day treatment group. In the control group, vehicle was administered orally to mice three times a day: zeitgeber time 12 (ZT12), ZT15 and ZT18, for 14 days. In the 14-day treatment group, TAK-994 was administered orally to mice at ZT12, ZT15 and ZT18 for 14 days. Electroencephalogram/electromyogram analysis was performed on day 1 and day 14 (ZT12-ZT21), and the subsequent sleep phase (ZT0-ZT10).

Results: On day 1, TAK-994 significantly increased wakefulness time, accompanied by a decrease in non-rapid eye movement (NREM) sleep time and rapid eye movement (REM) sleep time, in orexin/ataxin-3 mice compared with the control group. On day 14, TAK-994 also significantly increased wakefulness time, and decreased NREM sleep time and REM sleep time in orexin/ataxin-3 mice. There were no changes in the time spent in wakefulness, NREM sleep and REM sleep during the subsequent sleep phase after chronic dosing with TAK-994.

Conclusion: Wake-promoting effects of TAK-994 were observed following chronic dosing for up to 14 days in orexin/ataxin-3 mice with no rebound of sleep. Overall, there was no clear difference in efficacy between the single and repeated administration of TAK-994 in orexin/ataxin-3 mice.

Support: This work was conducted by Takeda Pharmaceutical Company Limited.

0142

TAK-925, AN OREXIN 2 RECEPTOR-SELECTIVE AGONIST, ENHANCED CORTICAL AROUSAL IN A NARCOLEPSY MOUSE MODEL DIFFERENT FROM EFFECTS OF MODAFINIL

Kimura, H. Ishikawa, T.

Takeda Pharmaceutical Company Limited, Fujisawa, Kanagawa, JAPAN.

Introduction: Patients with narcolepsy type 1 (NT1) suffer from distressing symptoms such as excessive daytime sleepiness (EDS) and cataplexy. Modafinil is widely used as a therapy for NT1; however, it has limited efficacy for EDS and no efficacy for cataplexy. TAK-925 is an orexin 2 receptor (OX2R)-selective agonist which improves multiple symptoms of narcolepsy such as fragmentation of wakefulness and cataplexy-like episodes, and also reduces weight gain, in orexin/ataxin-3 mice, a narcolepsy mouse model.

An early proof of concept study showed TAK-925 increased wakefulness compared to placebo in individuals with NT1; however, a head-to-head comparison between TAK-925 and modafinil in NT1 has not been performed to date. In this study, we carefully compared the wake-promoting effects of TAK-925 and modafinil in orexin/ataxin-3 mice.

Methods: TAK-925 or modafinil was administered to orexin/ ataxin-3 mice at zeitgeber time 12, and the sleep/wakefulness states were evaluated based on electroencephalogram (EEG) and electromyogram measurements. EEG spectral analysis was performed by fast Fourier transform during wakefulness. EEG frequency band was divided into five frequency bands: delta, theta, alpha, beta, and gamma.

Results: Both TAK-925 and modafinil significantly increased wakefulness time, and ameliorated fragmentation of wakefulness, in orexin/ataxin-3 mice during active phase. In contrast, TAK-925, but not modafinil, significantly decreased delta power, and increased alpha and gamma power during wakefulness in orexin/ ataxin-3 mice, suggesting a shift in EEG power density toward higher frequencies.

Conclusion: TAK-925, but not modafinil, enhanced cortical arousal and suppressed signs of somnolence and drowsiness. In a phase 1 study in individuals with NT1, TAK-925 was found to have pronounced effects on the maintenance of wakefulness test, reaching a total duration of 40 minutes wake time at some doses tested. Spectral analysis will be evaluated in future studies in NT1 patients.

Support: This work was conducted by Takeda Pharmaceutical Company Limited.

0143

VIGILANCE DECLINES FOLLOWING SLEEP DEPRIVATION ARE ASSOCIATED WITH TWO PREVIOUSLY IDENTIFIED DYNAMIC CONNECTIVITY STATES

Teng, J. Ong, J. Patanaik, A. Zhou, J. Chee, M. Lim, J. Duke-NUS Medical School, Singapore, SINGAPORE.

Introduction: Dynamic functional connectivity (DFC) analysis of resting-state fMRI data has been successfully used to track fluctuations in arousal in the human brain. Changes in DFC have also been reported with acute sleep deprivation. Here, we demonstrate that dynamic connectivity states (DCS) previously related to arousal are reproducible, and are associated with individual differences in sustained attention declines after one night of total sleep deprivation.

Methods: 32 participants underwent two counterbalanced restingstate fMRI scans: during rested wakefulness (RW) and following total sleep deprivation (SD). They also completed the Psychomotor Vigilance Test (PVT), a sustained attention task that is highly sensitive to the effects of sleep loss. SD vulnerability was computed as the decrease in response speed (Δ RS) and increase in lapses (Δ lapse) in SD compared with RW.

Dynamic functional connectivity analysis was conducted on rs-fMRI data. Connectivity matrices were clustered to obtain 5 prototypical DCS. We calculated the proportion of time participants spent in each of these DCS, as well as how often participants transitioned between DCSs. Relationships between SD vulnerability and connectivity metrics were then correlated.

Results: We recovered two DCS that were highly similar ($\rho = .89$ -.91) to arousal-related DCS observed in previous work (high arousal state (HAS); low arousal state (LAS)).

After sleep deprivation, the proportion of time spent in the LAS increased significantly (t29=3.16, p=.0039), while there was no significant change in HAS (t29=-1.43, p=.16). We observed significantly more state transitions in RW compared with SD. Change in LAS and HAS across sleep conditions correlated significantly with SD vulnerability ($\Delta LASx\Delta RS$: r=-0.64, p<.0001; $\Delta LASx\Delta lapse$: r=0.43, p=.018; $\Delta HASx\Delta RS$; r=0.43, p=.019; $\Delta HASx\Delta lapse$; r=-0.39, p=.033). Finally, $\Delta\%$ transitions was correlated with ΔRS but not Δ lapse.

Conclusion: This study adds to the evidence that two specific reproducible DCS are robust markers of arousal and attention, and may be useful indicators of SD vulnerability.

Support: This work was supported by the National Medical Research Council, Singapore (STaR/0015/2013), and the National Research Foundation Science of Learning (NRF2016-SOL002-001).

0144

RELATIONSHIP BETWEEN SLEEP EEG K-COMPLEX SLOW WAVE COUPLING AND NEXT-DAY THALAMIC ACTIVITY DURING PSYCHOMOTOR VIGILANCE TEST IN OBSTRUCTIVE SLEEP APNEA

Parekh, A. A. Kam, K. Mullins, A. Fakhoury, A. Castillo, B. Roberts, Z. Hedden, T. Varga, A. W. Rapoport, D. M. Ayappa, I. Icahn School of Medicine at Mount Sinai, New York, NY.

Introduction: There is large inter-individual variability in the relationship between obstructive sleep apnea (OSA) severity and lapses in vigilance as measured using psychomotor vigilance test (PVT). We have previously shown that overnight sleep EEG K-complex slow wave coupling (Δ SWAK) exhibits a dose-responsive relationship with next-day lapses in vigilance in OSA on and off treatment. We hypothesized that a variable thalamic dysfunction in OSA explains difference in lapses in vigilance and alterations in Δ SWAK across individuals.

Methods: Five newly diagnosed severe OSA subjects (mean apnea-hypopnea index [AHI4%=57.1±22.8/hr.]) with excessive daytime sleepiness (Epworth Sleepiness Scale=11±3.4) underwent nocturnal polysomnography followed by PVT testing within a 3T SKYRA MRI scanner. The PVT task inside the scanner (PVT-fMRI) was adapted to match the gold standard PVT-192 device. Each fMRI scanning session consisted of 2 10-min PVT runs interleaved with 2 control conditions wherein the subject pressed the response button at random intervals absent of a visual stimulus. fMRI data was analyzed in 2-step procedure (individual time-series followed by group analysis) using Analysis of Functional Neuroimages (AFNI) software package. To estimate thalamic activity during PVT-fMRI, parameter estimates of the %change in blood-oxygen-level-dependent (BOLD) signal using the contrast PVT-Control were used as the primary metric. The region of interest was limited to the bilateral thalamus using the Eickhoff-Zilles macro labels from the MNI N27 template.

Results: In a preliminary test, PVT performance for the subjects inside the scanner was not significantly different from that outside the scanner (PVTLapses_{IMRI}=7.3 \pm 2.1 vs. PVTLapses_{PVT192}=6.4 \pm 3.6 mean \pm std; PVTLapses=reaction time > 500 ms.). Within subjects, a trend toward lower thalamic recruitment was observed during PVT-fMRI (-0.17 \pm 0.2%; p=0.1). Further, lower thalamic activity during PVT-fMRI also showed a trend to lower overnight Δ SWAK (mean -1.2±1.4) values (r = 0.61, p = 0.17).

Conclusion: In severe OSA subjects with excessive daytime sleepiness, we observed a trend to reduced thalamic activity during daytime PVT. Overnight EEG K-complex slow wave coupling showed a similar trend with next-day thalamic activity during PVT, however the small sample size may have limited our ability to detect this association with statistical significance.

Support: AASM Foundation 199-FP-18; NIH K24HL109156

0145

PRE-SLEEP COGNITIVE AROUSAL DECREASES FOLLOWING A 4-WEEK INTRODUCTORY MINDFULNESS COURSE

Hassirim, Z.¹ Lim, E. C.² Lo, J. C.¹ Lim, J.¹

¹Duke-NUS Medical School, Singapore, SINGAPORE, ²Brahm Centre, Singapore, SINGAPORE.

Introduction: Mindfulness-based training has shown potential in reducing anxious and ruminative thoughts before sleep, and improving sleep quality. However, traditional 8-week programs have limited acceptability and uptake. In this study, we aimed to test the effects of a short introductory mindfulness training course on pre-sleep arousal and sleep quality.

Methods: Enrollees in a 4-week Mindfulness Foundation Course were invited to participate in the study and were allocated to one of two groups: intervention (N = 57) and waitlist control (N = 39). 101 participants enrolled in the experiment and 96 completed the protocol (mean(sd) age = 49.5(1.5), 56 female). Participants completed the Pittsburgh Sleep Quality Inventory (PSQI) and the presleep arousal scale (PSAS), and were monitored by actigraphy for a week at baseline and post-intervention. To test the effect of the intervention, outcome variables were subjected to repeated-measures ANCOVA with group as a between-subject variable, and age, gender, and years of education as covariates using intent-to-treat analysis.

Results: PSQI scores improved across both groups (treatment: t_{56} =4.25, p<.001, mean(sd) = 6.93(3.25)); waitlist: t_{38} =3.27, p=.002, mean(sd) = 7.15(3.55)); however, there was no significant interaction between group and time. There was a significant group by time interaction in the cognitive arousal subscale of the PSAS ($F_{1,90}$ =4.71, p=.03), Post-hoc tests revealed a significant decrease in the treatment but not the waitlist group (treatment: t_{50} =3.17, p=.001; waitlist: t_{30} =0.20, p=.84). The decrease in cognitive arousal correlated with the decrease in PSQI scores in the treatment group only (r =.3, p=.007). Finally, a statistically significant interaction favoring the treatment group was also observed in actigraphically measured WASO ($F_{1,82}$ =6.18, p=0.015).

Conclusion: The study suggests that a 4-week introductory mindfulness course has moderate effects on reducing cognitive arousal prior to sleep, and that these effects are correlated with improvements in subjective sleep quality.

Support: This study was funded from a STaR investigator grant (NMRC/STaR/0015/2013) and the National Research Foundation (Singapore) Science of Learning Grant (NRF2016-SOL002-001).

EFFICACY AND SAFETY OF SINGLE DOSE OF TS-142, A NOVEL AND POTENT DUAL OREXIN RECEPTOR ANTAGONIST, IN INSOMNIA PATIENTS

Uchiyama, M.¹ Kambe, D.² Imadera, Y.² Sunaga, H.² Hasegawa, S.² Nogi, T.² Kajiyama, Y.² Yoshida, S.² Ogo, H.² Uchimura, N.³

¹Nihon University School of Medicine, Tokyo, JAPAN, ²Taisho Pharmaceutical Co., LTD, Tokyo, JAPAN, ³Kurume University School of Medicine, Fukuoka, JAPAN.

Introduction: TS-142 is a novel dual orexin receptor antagonist (DORA) developed for the treatment of insomnia. Here we report its pharmacokinetic profile in the healthy subjects and its efficacy and safety in patients with insomnia.

Methods: A phasel study was conducted to clarify pharmacokinetic profile, in which various doses of TS-142 (1–30 mg) were orally administered once to thirty two healthy subjects. Subsequently, a phase 2a study utilizing polysomnography (PSG) was carried out in patients with primary insomnia, in which 5, 10, or 30 mg of TS-142, or placebo was randomly administered in a double-blind manner. Karolinska Sleepiness Scale (KSS) and Digit Symbol Substitution Test (DSST) were also examined in the morning after PSG.

Results: Following single administration of TS-142, plasma concentration of unchanged compound reached maximum within 2.50 h (median), and then eliminated rapidly, giving mean elimination half-life between 1.32 and 3.25 h. Twenty-three patients with insomnia completed the Phase2a study. Both latency to persistent sleep (LPS) and wake after sleep onset (WASO) were significantly improved with TS-142 at all doses, in comparison with placebo (-42, -42 and -45 for LPS [min] and -28, -35 and -55 for WASO [min] in 5, 10, 30 mg, respectively). KSS and DSST administered in the morning indicated no serious hangover effects. No serious adverse events were observed in these trials.

Conclusion: The phase 1 trial showed favorable pharmacokinetic profiles. The phase 2a trial demonstrated that TS-142 was efficacious in objective sleep onset and maintenance with minimal next-day residual effects. TS-142 was generally well tolerated in both studies.

Support: Taisho Pharmaceutical. Co., Ltd.

0147

DYNAMIC ALTERATIONS IN FUNCTIONAL CONNECTIVITY BETWEEN SLEEP- AND WAKE-PROMOTING REGIONS OF THE HUMAN BRAIN AT THE SLEEP ONSET PERIOD

Ishii, T.¹ Koike, T.² Nakagawa, E.² Sumiya, M.² Sadato, N.² ¹Kyoto University Graduate School of Medicine, Kyoto, JAPAN, ²National Institute for Physiological Sciences, Okazaki, JAPAN.

Introduction: The sleep onset period, involving so-called stage N1 sleep largely, is characterized by a reduction in the amount of alpha activity compared to wakefulness. Various kinds of physiological and psychological changes are also apparent, such as slow eye movements, changes in muscle tonus, and the hypnagogic dream-like mentation. These phenomena are thought to be the reflection of dynamic alterations in the brain during the transition period, however, details of these changes have still been uncovered.

Methods: We aimed to investigate a dynamic shift in the brain connectivity at sleep onset using the method of EEG-fMRI simultaneous recording. Twenty-three healthy subjects participated. EEG/fMRI were recorded simultaneously during an hour's nap in a 3T-MRI scanner and real-time monitoring of EEG was performed. To record the transition period between multiple times, an experimenter inside a scanner room touched a subject's foot for inducing arousal when a shift to NREM sleep stage 1 was observed. EEG data were scored according to the AASM criteria. Based on sleep stages defined by polysomnographic findings, we investigated alterations in functional connectivity of sleep- and wake- promoting regions within the hypothalamus and other areas including the thalamus.

Results: Posterior alpha power showed significant positive correlation with BOLD signals in the anterior and medial dorsal thalamus. Connectivity between the thalamus and cortical regions reduced sharply in the descent to sleep stage. Meanwhile, BOLD signals of the sleep- and wake- promoting regions within the hypothalamus fluctuated with certain temporal lags from fluctuations of alpha rhythm at sleep onset.

Conclusion: Present findings provide preliminary evidence of dynamics of wake- and sleep- promoting regions in the human brain in vivo. Our data also support the hypothesis that reduced thal-amocortical connectivity which limits the capacity to integrate information is associated with the transition of consciousness at sleep onset.

Support: None

0148

SEROTONERGIC DORSAL RAPHE NEURONS REGULATE HYPERCAPNIA INDUCED AROUSAL THROUGH 5HT2A RECEPTORS ON THE PARABRACHIAL NEURONS

Kaur, S. Thomas, R. C. Saper, C. B.

Beth Israel Deaconess Medical Centre and Harvard medical School, Boston, MA.

Introduction: Serotoninergic dorsal raphe neurons (DR^{Sert}) are CO2 responsive, and mice lacking serotonin have impaired arousal to CO2. We showed that the neurons in external lateral parabrachial nucleus containing calcitonin gene related peptide (PBel^{CGRP}), are required for CO2-arousal. PBel^{CGRP} neurons also receive serotoninergic innervation from the DR^{Sert}. 5HT_{2A} agonist restores CO2 responsiveness in mice lacking serotonin, suggesting that DR^{Sert} may modulate CO2 arousal by acting on 5HT_{2A} receptors possibly on the PBel neurons.

Methods: We used serotonin transporter (Sert)-Cre mice to optogenetically inhibit DR^{Sert} neurons and their terminals in the PBel. We injected AAV-FLEX-ArchT into the DR and implanted an optical fiber just above it in one set of Sert-Cre mice and bilaterally in the PBel in another set. All mice were instrumented for sleep and optogenetics and were tested for EEG arousals to 10% CO2. Latencies of arousal were compared with optogenetic inhibition of either the DR neurons or their terminals in the PBel with a 593nm laser light. We further tested whether a 5HT_{2A} agonist (TCB-2) can reverse blockade of CO2 arousal in mice where DR^{Sert} terminals in PBel were inhibited. Finally, TCB-2 was injected in mice with PBel^{CGRP} deletions and arousal latency to CO2 was compared.

Results: Compared to the control (Laser-OFF) condition, arousal latency to CO2 was significantly increased by photoinhibition of either the DR^{Sert} neurons (n=6; latency- 40.9 \pm 6.4 vs. 13.81 \pm 0.69 sec; F_{3.17}= 11.5; P< 0.001) or their terminals in PBel (n=8;

SLEEP, Volume 43, Abstract Supplement, 2020

latency-34.9 ± 2.3 sec vs. 16.62 ± 0.97 sec, $F_{1, 14} = 56.9$; P< 0.001). This was reversed by the 5HT_{2A} agonist TCB-2 (5mg/kg), as it reduced the latency to CO2 arousal in mice with photoinhibition of terminals in PBel from 35.48 ± 7.31 sec to 16.24 ± 1.06 sec ($F_{3,9} = 8.05$; P= 0.006), but had no effect in mice with PBel^{CGRP} neurons deletions.

Conclusion: The serotonin system modulate CO2-arousals by the DR^{Sert} input to the PBel. TCB-2 reversed the effect of inhibition of DR^{Sert} terminals in the PBel, but not in mice with PBel^{CGRP} deletions, suggests that DR^{Sert} modulate PBel^{CGRP} neurons through $5HT_{\gamma_a}$ receptors.

Support: NIH- 2P01 HL095491 and NS112175

0149

NIGHTLY SLEEP CHARACTERISTICS ARE ASSOCIATED WITH NEXT-DAY MINDFULNESS

Lee, *S.*¹ *Mu*, *C.*¹ *Gonzalez*, *B*. *D.*² *Vinci*, *C*. *E.*² *Small*, *B*. *J.*¹ ¹University of South Florida, Tampa, FL, ²Moffitt Cancer Center, Tampa, FL.

Introduction: Previous research shows that insufficient and poor sleep is associated with perceiving more stressors the following day. Sleep may also be associated with daily mindfulness, a state in which one is highly aware and focused on the present moment without evaluating or judging that moment. The association between high mindfulness and better sleep is well-established; yet, less is known about the temporal directionality between sleep and mindfulness. This study examined whether nightly sleep predicts next-day mindfulness, and vice versa.

Methods: Participants were 60 middle-aged adults working as a full-time nurse at a cancer hospital (M_{age} =35.35±11.83). Using ecological momentary assessments for 14 days, we asked participants about their previous night's sleep upon waking and participants completed the 5-item state Mindful Attention Awareness Scale an average of 3 times/day. Multilevel modeling examined variance at the between- and within-person levels and tested two temporal directions simultaneously: better sleep predicting mindfulness and mindfulness predicting better sleep.

Results: Daily mindfulness, sleep duration, sleep sufficiency, and sleep quality displayed 34%, 85%, 82%, and 85% within-person variation, respectively. At the within-person level, daily mindfulness was greater on days following longer than usual sleep duration (B=0.39hrs or 23min, p<.01) and greater than usual sleep sufficiency (B=0.26, p<.001). The within-person link between sleep sufficiency and mindfulness remained even after controlling for the strong association of workdays with less sleep sufficiency. Conversely, mindfulness was not predictive of sleep outcomes. At the between-person level, participants who had greater sleep sufficiency and higher sleep quality overall reported greater mindfulness. These associations remained after adjusting for sociodemographics, dayshift vs. nightshift, and workdays vs. non-work days.

Conclusion: Sufficient sleep duration and perceived sleep sufficiency may be antecedents of how mindful individuals are the following day. Future analyses will test whether the daily link between sleep and mindfulness contributes to health outcomes.

Support: This work was supported, in part, by the University of South Florida College of Behavioral & Community Sciences Internal Grant Program (PI: Lee, Grant No. 0134930).

0150

COMPARING TWO MEASURES OF SLEEP DEPTH/ INTENSITY

Younes, M.¹ Schweitzer, P. K.² Griffin, K.² Walsh, J. K.² Balshaw, R.³

¹Sleep Disorders centre, University of Manitoba, Winnipeg, MB, CANADA, ²Sleep Medicine & Research Center, St. Luke's Hospital, Chesterfield, MO, Chesterfield, MO, ³Centre for Healthcare Innovation, Rady Faculty of Health Science, University of Manitoba,, Winnipeg, MB, CANADA.

Introduction: There is currently no well-validated method for evaluating objective sleep depth/intensity. Delta power is thought to reflect sleep depth based upon limited evidence. Odds-ratio-product (ORP) is a recently introduced continuous measure of sleep depth. We compared delta spectral power (delta) and ORP as measures of sleep depth/intensity during manipulations that altered sleep depth (sleep restriction with placebo or with a delta-promoting drug). We hypothesized that ORP will provide a more robust measure of sleep depth.

Methods: This is a secondary analysis of data from a study in which forty-one healthy subjects were sleep restricted and randomized to receive placebo or gaboxadol 15mg. Participants underwent consecutive in-laboratory sleep studies on two baseline, four sleep restriction (5 hours) and two recovery nights. The relation between delta or ORP during any given 30s epoch and sleep depth, operationally defined as the probability of arousal / awakening occurring during the next 30 seconds (arousability), was assessed.

Results: Mean ORP values differed significantly among the four sleep / wake stages, but delta power did not differentiate wake, N1 and N2. The relation between ORP and arousability was linear across the entire range of ORP whereas delta power detected differences in arousability only with delta values < 300 μ V². Correlations with arousability in individual subjects were stronger with ORP (p < 0.0001). Receiver operating characteristic analysis found the ability to predict imminent arousal to be significantly greater with ORP than with delta power for all experimental conditions (p < 0.0001). The increase in sleep depth with restriction alone was detected on the second day of restriction by ORP (p < 0.01) but not by delta.

Conclusion: As compared to delta power, ORP is more discriminating among sleep stages, more sensitive to sleep restriction, and more closely associated with arousability. These observations indicate ORP better reflects sleep depth/intensity. **Support:** None

0151

THE DUAL OREXIN RECEPTOR ANTAGONIST ALMOREXANT BLOCKS THE SLEEP-DISRUPTING EFFECTS OF METHAMPHETAMINE IN MALE RHESUS MONKEYS

Berro, L. F.¹ Rowlett, J. K.¹

¹University of Mississippi Medical Center, Jackson, MS, ²University of Mississippi Medical Center, Jackson, MS.

Introduction: Individuals with stimulant use disorder show a high prevalence of sleep problems. In the laboratory, stimulant drugs have been shown to affect sleep parameters in human and nonhuman primates, even when administered many hours before bedtime. Although the mechanisms underlying the relationship between stimulant use/abuse and sleep impairment remain unclear,

recent research has implicated the orexin (also called "hypocretin") system as a critical regulator of sleep-wake states. The aim of the present study was to investigate the effects of the dual orexin receptor antagonist (DORA) almorexant on the sleep-disrupting effects of methamphetamine in rhesus monkeys.

Methods: Male adult rhesus monkeys (*Macaca mulatta*, n=4) were fitted with primate collars to which Actiwatch monitors were attached. Actigraphy recording was conducted during baseline conditions and on the night after acute morning (9h) administration of vehicle or methamphetamine (0.03, 0.1 or 0.3 mg/kg, i.m.). During a second set of treatments, vehicle or almorexant (1, 3 or 10 mg/kg, i.m.) were administered in the evening (16:30h, 1.5h before "lights off") following morning (9h) administration of methamphetamine (0.3 mg/kg, i.m.).

Results: Morning methamphetamine administration dosedependently impaired sleep in rhesus monkeys, with the dose of 0.3 mg/kg significantly increasing sleep latency and decreasing sleep efficiency. Evening administration of almorexant improved both actigraphy-based sleep measures after morning administration of methamphetamine in a dose dependent manner.

Conclusion: Our findings indicate that orexin receptor systems are involved in methamphetamine-induced sleep disruption. The exact role of the two orexin receptors in this effect, alone or together, remains to be determined. This study suggests that DORAs can be effective in treating sleep impairment in individuals with methamphetamine use disorder or under stimulant prescription for other sleep and psychiatric disorders.

Support: Supported by UMMC Research Enhancement Funds.

0152

INSOMNIA SEVERITY AND DAYTIME COMPLAINTS: WHAT IS TO BE LEARNED WHEN THESE DOMAINS ARE DISCORDANT?

Perlis, M. L.¹ Boyle, J. T.^{2,1} Vargas, I.³ Giller, J.¹ Seewald, M.¹ D'Antonio, B.¹ Muench, A.^{1,4} Williams, N. J.⁵ Rosenfield, B.² Klingman, K.⁶

¹Behavioral Sleep Medicine Program, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA, ²Department of Clinical Psychology, Philadelphia College of Osteopathic Medicine School of Professional and Applied Psychology, Philadelphia, PA, ³Department of Psychological Sciences, University of Arkansas, Fayetteville, AR, ⁴Chronobiology and Sleep Institute, University of Pennsylvania, Philadelphia, PA, ⁵Center for Healthful Behavior Change, Department of Population Health, NYU School of Medicine, New York, NY, ⁶College of Nursing, State University of New York Upstate Medical University, Syracuse, NY.

Introduction: If illness severity and daytime dysfunction are construed as categorical entities, it is possible to conceptualize the relationship between these variables in terms of a 2x2 matrix where the resultant cells represent a concordant dimension (quadrants 2 & 4 [high-high and low-low]) and a discordant dimension (quadrants 1 & 3 [high-low and low-high]). The question for the present analysis was, what percentage of subjects populate each quadrant and is it the case that the discordant dimension contains only a small percentage of subjects? **Methods:** Illness severity and daytime dysfunction data was collected from individuals with sleep continuity complaints in archival/community-based sample (N = 4680; 60% female; Ages 18–89) (www.sleeplessinphilly.com). Illness severity was operationalized as Total Wake Time (TWT; [SL+WASO+EMA=TWT]) and daytime dysfunction was operationalized as the composite score of six daytime symptoms items. Median splits were calculated for each variable and subjects were typed accordingly (HH, LL, HL, & LH).

Results: Surprisingly, the sample was relatively equally distributed into the two dimensions; 38% and 23% for the concordant dimension and 13% and 26% for discordant dimension.

Conclusion: The 39% of subjects in the discordant groups might be thought of as complaining good sleepers (LH) and noncomplaining poor sleepers (HL). Other investigators have identified the LH subjects as individuals with "insomnia identity". Alternatively, it is possible to characterize the whole dimension as being related to a mismatch between the individual's sleep need and sleep ability. Those who need a lot, may suffer a lot, in the face of only a little (LH) whereas those who need a little, may suffer only a little, in the face of a lot (HL).

Support:

0153

PATIENT-DEFINED INSOMNIA SEVERITY: HOW MUCH WAKEFULNESS IS PROBLEMATIC?

D'Ant

onio, B.¹ Boyle, J. T.^{2,1} Seewald, M.¹ Giller, J.¹ Muench, A.^{1,3} Vargas, I.^{4,5} Williams, N. J.⁶ Klingman, K.⁷ Perlis, M. L.¹ ¹Behavioral Sleep Medicine Program, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA, ²Department of Clinical Psychology, Philadelphia College of Osteopathic Medicine School of Professional and Applied Psychology, Philadelphia, PA, ³Chronobiology and Sleep Institute, University of Pennsylvania, Philadelphia, PA, ⁴Department of Psychological Sciences, University of Arkansas, Fayetteville, AR, ⁵Sleep and Stress Research Laboratory, University of Arkansas, Fayetteville, AR, ⁶Center for Healthful Behavior Change, Department of Population Health, NYU School of Medicine, New York, NY, ⁷College of Nursing, State University of New York Upstate Medical University, Syracuse, NY.

Introduction: While Insomnia Disorder is formally defined in the ICSD-3 and DSM-5, neither diagnostic system adopts quantitative criteria for illness severity. Interestingly, quantitative criteria are provided for frequency and chronicity (i.e., ≥ 3 days / week for \geq 3 months). For research purposes, illness severity has long been defined using the "30 minute rule" (SL and/or WASO and/or EMA of \geq 30 minutes is the threshold for clinical relevance). In the present analysis, this threshold was assessed for its significance to patients. Methods: Sleep continuity disturbance (SCD; SL, WASO, and EMA) and problem endorsement data were collected from an archival/community-based sample (N = 4680; 60% female; Ages 18-89 years; www.sleeplessinphilly.com). Problem endorsement was evaluated through questions that included, "Do you consider this a problem?" after participants reported length of SL, WASO, and EMA. Problem endorsement percentages were calculated for 5 minute bins for between 0 and 65 minutes, with one additional bin for > 65 minutes. The temporal bins were compared for significant deviations using absolute (percent of subjects at 0-5 and 5-10 minutes) and moving references (last significant percent).

Results: The first temporal bin to differ from the absolute reference for SL, WASO, *and* EMA was the 26–30 minute bin. At this threshold, 87%, 70%, <u>and</u> 94% of the subjects' identifying SL, WASO and EMA as being problematic (and was deemed statistically different from "normal" [0–10 minute values]).

Conclusion: These data suggest that the "30 minute rule" (which is of unknown provenance) roughly corresponds to the level of illness severity (lowest common threshold) identified by patients as problematic. While the threshold for SL and EMA show a clear majority, the lower percentage of subjects for WASO suggests that people are more tolerant of middle of the night wakefulness. **Support:**

0154

ROLE OF NORADRENERGIC PROJECTION TO THE PREOPTIC AREA IN REGULATION OF AROUSAL

Antila, H. Kwak, I. Covarrubias, I. Baik, J. Hong, J. Stucynski, J. Weber, F. Chung, S.

University of Pennsylvania, Philadelphia, PA.

Introduction: Locus coeruleus (LC) is a noradrenergic nucleus in the brainstem involved in the regulation of attention, arousal, mood and sensory gating. LC projects to multiple brain regions and recent development of novel systems neuroscience tools allows the dissection of projection-specific LC function in more detail. One of the regions with noradrenergic projection is the preoptic area of the hypothalamus (POA). POA has been shown to contain neurons that are important for regulating sleep, and we have examined the function of the LC projection to the POA in sleep and arousal.

Methods: We used optogenetics, chemogenetics, fiber photometry and in vivo electrophysiology to study the function of LC noradrenergic projection to the POA.

Results: Norepinephrine release in the POA fluctuates with brain state changes indicating that the LC to POA projection may be involved in regulating sleep and arousal. Optogenetic stimulation of LC fibers in the POA promotes wakefulness. Furthermore, optogenetic stimulation of the LC fibers in the POA modulates the activity of sleep- and wake-active neurons.

Conclusion: We have identified the role of the LC noradrenergic projection to the POA in the regulation of brain states. Stimulation of the LC fibers in the POA promotes wakefulness and modulates the activity dynamics of sleep- and wake-active neurons in the POA. Our results provide more detailed information about the role of this specific projection, which has been known to exist for a long time, but with insufficient in vivo evidence of its precise function. **Support:** Sigrid Juselius foundation, Alfred P. Sloan Research

Fellowship in Neuroscience, The Whitehall foundation grant, McCabe Fund Award, NARSAD Young Investigator Award.

0155

SUVN-G3031, A POTENT AND SELECTIVE HISTAMINE H3 RECEPTOR INVERSE AGONIST - DIFFERENTIATING FEATURES OVER CURRENT TREATMENTS OF NARCOLEPSY

Shinde, A. Subramanian, R. Palacharla, R. Pandey, S. Benade, V. Jayarajan, P. Bojja, K. Nirogi, R. Suven Life Sciences, Hyderabad, INDIA.

Suven Life Sciences, Hyderabad, INDIA.

Introduction: Majority of pharmacological agents used in the treatment of narcolepsy have several limitations. Both nonclinical and clinical evidences suggest usefulness of the histamine H3 receptor (H3R) inverse agonists for the treatment of narcolepsy and addressing several of the current limitations.

Methods: Extensive nonclinical studies were carried out for SUVN-G3031 and other pharmacological agents that are currently being used for the treatment of narcolepsy. Nonclinical parameters like

inter-species binding affinity, selectivity profile, in vivo and in vitro ADME features, nonclinical efficacy, neurochemistry and safety were compared. Results: SUVN-G3031 has no inter-species variation in binding affinity at H3R with less than 50% inhibition at 1 µM against 70 other targets. Unlike pitolisant, SUVN-G3031 has no significant binding affinity at sigma 1 and 2 receptor. SUVN-G3031 has no inhibition and induction liability towards major CYP enzymes and transporters. Pitolisant is reported to be a CYP3A4, CYP2B6, and CYP1A2 inducer and a CYP2D6 and OCT1 inhibitor. SUVN-G3031 has robust wake promoting effects. SUVN-G3031 showed negligible affinity towards hERG channel with $IC_{50} > 10 \ \mu M$ and had no effects on any ECG parameters in dog telemetry study. SUVN-G3031 did not show convulsion in rats up to the tested dose of 100 mg/kg, p.o. Most of the pharmacological agents used for the treatment of narcolepsy have abuse liability; SUVN-G3031 produced no change in the striatal and accumbal dopamine levels in rats suggesting no propensity to induce abuse liability. Unlike competing H3R inverse agonists, SUVN-G3031 has no effects on fertility and embryo-fetal development up to the highest tested doses.

Conclusion: Nonclinical studies demonstrate superiority of SUVN-G3031 over pharmacological agents currently used in the treatment of narcolepsy. SUVN-G3031 is being evaluated in a Phase 2 study as monotherapy for the treatment of narcolepsy with and without cataplexy (ClinicalTrials.gov Identifier: NCT04072380).

Support: None

0156

ACTIVATION OF GLUTAMATERGIC PPT NEURONS AND THEIR PROJECTIONS PROMOTES AROUSAL, AND DISTINCT WAKE BEHAVIORS

Kroeger, D. Thundercliffe, J. A. Phung, A. Geraci, C. DeLuca, R. Bragg, S. Arrigoni, E. Scammell, T. E. BIDMC / Harvard Medical School, Boston, MA.

Introduction: The pedunculopontine tegmental (PPT) region in the brainstem is crucial for the regulation of sleep/wake states. We recently showed that chemogenetic activation of glutamatergic PPT neurons promotes wakefulness for several hours. Here we used optogenetic activation of these neurons to further investigate the mechanisms and pathways through which PPT glutamatergic neurons produce wakefulness. **Methods:** Using vGlut2-cre mice, we transfected neurons in the PPT region with a viral vector coding for cre-dependent ChR2 tagged with fluorescent mCherry and implanted bilateral optical fibers above the PPT nuclei as well as EEG/EMG leads. Two weeks later, we administered blue laser light to activate ChR2-expressing neurons and recorded sleep/wake states.

Results: Activation of ChR2-expressing glutamatergic neurons during NREM sleep rapidly elicited wakefulness in a stimulation-frequency dependent manner, with higher frequencies producing wake more quickly and with longer duration. Random, automated stimulation for 10 s at 5 Hz over 24 h revealed that activation of glutamatergic PPT neurons produces rapid arousals form NREM sleep. Importantly, stimulation did not wake mice from REM sleep, suggesting that glutamatergic PPT signaling does not interfere with REM sleep. To map the target areas through which PPT glut neurons produce wakefulness, we used a viral tracer to visualize PPT glutamatergic projections, and then optogenetically stimulated terminals in 1) basal forebrain, 2) lateral hypothalamus, 3) thalamus, and 4) substantia nigra. We found that stimulating terminals in all of these regions woke mice from NREM sleep, and stimulating terminals in the basal forebrain and lateral hypothalamus produced a

number of active wake behaviors such as locomotion. In contrast, stimulation of PPT glut soma and terminals in the thalamus and substantia nigra results mainly in quiet wakefulness.

Conclusion: Glutamatergic PPT neurons potently promote arousal from NREM sleep but not REM sleep, and the resulting wake behavior is modulated by different projection targets. **Support:** NIH grant P01 - HL095491

0157

THE RELATIONSHIP BETWEEN REACTION TIME, INSOMNIA SEVERITY, SLEEPINESS, AND DYSFUNCTIONAL BELIEFS ABOUT SLEEP

Gencarelli, A. M. Zurlinden, T. Nicoletta, A. Winters, A. Sorrell, A. Corbett, Q. Everhart, E. East Carolina University, Greenville, NC.

Introduction: Poor sleep quality has adverse effects ranging from decreased focus to increased work-related injuries. The Perceptual Vigilance Task (PVT), a measure of reaction time (RT) used to assess alertness is commonly used in sleep research. This study focuses on the relationship between dysfunctional sleep-related cognitions (Dysfunctional Beliefs and Attitudes About Sleep Scale [DBAS]), insomnia severity (Insomnia Severity Index [ISI]), and sleepiness (Epworth Sleepiness Scale [ESS]) and their association to PVT RT.

Methods: 162 participants were recruited from East Carolina University. Inclusion criteria: right-handed adults; exclusion criteria: history of brain injury, seizure disorder, or vision impairment. Age range 18–39 (M = 20.15; SD = 3.01); 81 (49.1%) females. **Results:** ISI was correlated with PVT RT for inter-stimulus interval delay times of 1000ms r(162) = .155, p = .05, 2000ms r(162) = .204, p = .009, 5000ms r(162) = .164, p = .04, and 6000ms r(162) = .181, p = .02. DBAS was correlated with PVT RT for delay times of 2000ms r(162) = .204, p = .021, 3000ms r(162) = .160, p = .04, 4000ms r(162) = .170, p = .03, 6000ms r(162) = .171, p = .030, 7000ms r(162) = .219, p = .005, and 8000ms r(162) = .158 p = .045. ESS was not correlated with PVT. A regression was calculated to predict reaction time at 7000ms delay based on the DBAS (F(1,151) = 2.51, p = .01), with an R^2 of .12.

Conclusion: There is a diminishing association found between insomnia severity and RT during inter-stimulus delay times (>6000ms). Dysfunctional beliefs about sleep correlate with RT through 8000ms delay, eventually predicting RT. Regardless of severity of sleep disturbance, sleep-related bias may affect subjective feelings of wakefulness and objective levels of alertness (e.g., one who believes they are not obtaining sufficient sleep may act accordingly).

Support: N/A

0158

LOSS OF CONNEXIN 36 ELICITS ABNORMALITIES IN THALAMOCORTICAL NETWORK ACTIVITY RELEVANT TO NEUROPSYCHIATRIC DISORDERS

McNally, J. M.¹ Thankachan, S.¹ Uygun, D. S.¹ Basheer, R.¹ ¹VA Boston Healthcare System-Harvard Medical School, West Roxbury, MA, ²VA Boston Healthcare System-Harvard Medical School, West Roxbury, MA.

Introduction: Neuronal gap-junctions are extensively expressed in mammalian forebrain and suggested to contribute to stateregulation and thalamocortical network activity. However, the physiological role of gap-junctions on these processes remains poorly understood. Connexin-36 (Cxn36) is highly expressed in the brain, representing a mechanism for electrical coupling of inhibitory neurons. We examined the effects of global Cnx36 deletion on sleep/wake and spontaneous and evoked EEG activity.

Methods: We recorded *in vivo* EEG/EMG in Cxn36KO mice and littermate controls. Electrodes were stereotaxically implanted above frontal cortices. We analyzed sleep/wake states and algorithmically detected sleep spindles over 24 hours. Mice underwent auditory stimulation paradigms including the auditory steady state response (ASSR; 1 second train 20-50Hz clicks, 100 reps., 85dB) and mismatch negativity (MMN; 2.5kHz standard 90%, 10kHz deviant 10%, 300ms ISI, 90dB). Social behavior and investigation-evoked EEG activity were also assessed via the social habituation task (repeated 5 min exposures to novel mouse).

Results: Cnx36KO mice exhibited limited sleep/wake abnormalities (n=7/group). Power spectra of EEG revealed significant impairments in spontaneous gamma-band activity (30-80Hz; All States, Light & Dark Phases), and beta activity (15-25Hz; All States, Light Phase). Sigma activity (10-15Hz) was significantly decreased (NREM and REM, Light phase). This was particularly pronounced during NREM-REM transitions. Despite no changes in spindle density, both spindle amplitude and duration were significantly decreased in Cnx36KOs. Cxn36KOs exhibited a blunted gamma-band response to acute ketamine (15mg/kg; IP), impaired 30 & 40Hz ASSR, and an abnormal response in the MMN task (decrease ERP peak amplitude & gamma). Finally, Cxn36KO mice exhibit impaired social habituation and significantly decreased investigation evoked slow gamma-band activity (30 - 55Hz).

Conclusion: Our data suggest Cxn36 plays a critical role in regulating thalamocortical network activity. Further, impairments in Cnx36KO mice reflect abnormalities in neuropsychiatric disorders, including schizophrenia, implicating Cnx36 containing gap junctions as a novel therapeutic target.

Support: Research supported by VA CDA Award BX002130 (JMM), VA Merit Awards BX004500 (JMM), BX001404 (RB), and NIMH RO1 MH39683 (Ritchie E. Brown).

0159

REDUCED REM SLEEP PERCENT IN FREQUENT CANNABIS VERSUS NON-CANNABIS USERS

*Carr, M.*¹ *Borcsok, R.*² *Taylor, M.*² *Segust, S.*² *Pigeon, W.*¹ *Bradshaw, C.*²

¹University of Rochester, Rochester, NY, ²Swansea University, Swansea, UNITED KINGDOM.

Introduction: THC (the main psychoactive component of cannabis) has been shown to suppress REM sleep and decrease sleep latency, although this is not consistently replicated. Increased dream vividness is reported to occur in abstinent cannabis users, although dream quality in active users is unstudied. The current study aimed to assess the effects of cannabis use compared to non-use on objective sleep measures, dream reports, and self-reported anxiety, memory, and sleep quality. To collect objective sleep data we piloted the use of a portable PSG headband that allows EEG and EOG recording at home.

Methods: 12 regular cannabis users (> 3 days per week) & 9 non-users (aged 19 - 27; 43% female) participated; participants used no other drugs or alcohol on study nights. The most common form of cannabis use was smoking in joints with tobacco (range = 1 - 15 per day). Participants wore the PSG headband (the Hypnodyne ZMax) over 2 nights at home (2^{nd} night used for analysis), and were instructed to awaken 4 times across the night to fill out brief dream reports. Objective sleep measures included TST, Sleep latency, REM latency, and REM percent. Self-report measures included the Pittsburgh Sleep Quality Index, Everyday Memory Questionnaire, and State-Trait Anxiety Index. Dream measures included

recall frequency, word length, and three attributes rated on a 1–7 Likert scale - sensory vividness, emotional intensity, and bizarreness.

Results: There were no group differences on self-report measures. Cannabis users showed longer REM latency (t=2.23, p=.04) and lower REM% (U=22, p=.02); there were no other objective group differences. Cannabis users reported higher bizarreness in their dreams (t=2.07, p=.05); there were no other dream differences.

Conclusion: The study presents a novel approach to assess sleep at home and cannabis use. Significant differences emerged between users and non-users on REM latency, REM%, and dream bizarreness. **Support:** N/A

0160

CHEMOGENETIC ACTIVATION OF HYPOTHALAMIC TACHYKININ 1-EXPRESSING NEURONS REDUCES SLEEP AND INCREASES RESISTANCE TO ISOFLURANE

Reitz, S. Wasilczuk, A. Z. Beh, G. Kelz, M. B.

Department of Anesthesiology and Critical Care, University of Pennsylvania, Philadelphia, PA.

Introduction: The hypothalamic preoptic area (POA) is a heterogeneous region of the brain, containing intermingled populations of excitatory and inhibitory neurons expressing a wide variety of molecular markers. Of its many important homeostatic roles, the POA modulates arousal including natural states (sleep and wakefulness) as well as drug-induced (anesthetic-induced unconsciousness) states. Although sleep and anesthetic-induced unconsciousness are undoubtedly distinct, multiple lines of evidence demonstrate that shared neuronal circuits regulate both states. Previous work identified a population of sleep-promoting, tachykinin 1-expressing (Tac1) neurons within the POA. Given evidence in the VLPO and SON, we hypothesized that Tac1 POA neurons would be another site of convergence for facilitating entry into both NREM sleep and anesthetic-induced unconsciousness. Methods: Sleep and isoflurane sensitivity were assessed in Tac1-Cre mice expressing either a Cre-dependent excitatory hM3Dq DREADD or a control fluorophore in the POA. Sleep was assessed using beam break actigraphy, while isoflurane sensitivity was assessed using video tracking as well as a loss of righting reflex assay to construct dose-response curves. Results: Unexpectedly, activation of Tac1 POA neurons with 3mg/ kg CNO reduced cumulative sleep time by 55% compared to controls (p<0.0001). Decreased sleep time was due to a 59% reduction in sleep bout duration (p<0.01), as the number of sleep bouts remained unchanged. Activity, measured as distance traveled, also increased by 167% compared to vehicle (p<0.0001). Activation of these neurons also increased resistance to isoflurane on both induction (p<0.0001) and emergence (p<0.0001). Additionally, activation during a continuous steady-state exposure to isoflurane destabilized the unconscious state.

Conclusion: Our results support the concept that POA Tac1 neurons function as a point of convergence for neural circuits regulating arousal from endogenous and drug-induced states of unconsciousness, while also illustrating the complexity of sleep and wake regulation within the hypothalamus. **Support:** R01-GM088156-06, T32-HL007953

0161

GREATER NEGATIVE AFFECTIVITY PREDICTS SHORTER INFANT SLEEP DURATION

Butler, B. P.¹ Burdayron, R.¹ Laganière, C.¹ Béliveau, M.² Dubois-Comtois, K.³ Pennestri, M.¹

¹Department of Educational and Counselling Psychology, McGill University, Montreal, QC, CANADA, ²Département de psychologie, Université de Montréal, Montreal, QC, CANADA, ³Département de psychologie, Université du Québec à Trois-Rivières, Trois-Rivières, QC, CANADA. **Introduction:** Certain temperament characteristics in infants have been shown to be associated with infant sleep patterns. However, other sleep-related practices, such as co-sleeping and breastfeeding, are also known to be associated with sleep in infancy and are not always taken into account in studies assessing the association between temperament and sleep. Thus, this study aims to examine the associations between infant temperament and sleep parameters at six months of age, while controlling for co-sleeping and breastfeeding practices.

Methods: Mother-infant dyads (n=60) were recruited in the metropolitan Montreal area and consented to participate in the study. Infant sleep was reported by mothers at six months of age (± 1 month) using sleep diaries (two-week period). Total nocturnal and longest consecutive sleep duration were retrieved from the diaries and averaged through the two-week period. Temperament was measured with the Infant Behaviour Questionnaire-Revised (IBQ-R). Sleep-related parental practices (co-sleeping and breastfeeding) were measured using the Sleep Practices Questionnaire (SPQ). Multiple regression analyses were conducted to determine the degree to which the temperament composite negative affectivity predicted total nocturnal and longest consecutive sleep duration while controlling for breastfeeding and co-sleeping practices.

Results: Regression analyses revealed significant regression models for total nocturnal (F(3,44)=6.25, p=.001, R²=.30), and longest consecutive sleep duration (F(3,44)=6.26, p=.001, R²=.30). Greater infant negative affectivity predicted shorter nocturnal sleep duration (β ;;=-0.28, p=.034) and shorter longest consecutive sleep duration (β ;:=-0.34, p=.010) after adjusting for breastfeeding and co-sleeping practices.

Conclusion: Findings suggest infants with greater negative affectivity sleep for fewer hours during the night and have shorter periods of consecutive sleep, even when sleep-related parental practices are considered. These results provide further support for the relationship between infant temperament and sleep at six months of age. Future research should investigate the relationship between infant temperament and sleep using paternal report in addition to maternal report.

Support: SSHRC

0162

MENTAL STRESS COMPROMISES HUMAN SLEEP THROUGH A BIOLOGICAL, NOT PSYCHOLOGICAL, PATHWAY

Brindle, R. C.¹ Ahmad, M.² Evans, K.¹ Hatfield, A.¹ Holthouser, S.¹ ¹Washington and Lee University, Department of Cognitive and Behavioral Science, Lexington, VA, ²Washington and Lee University Neuroscience Program, Lexington, VA.

Introduction: The extent to which mental stress causes sleep disturbance is unknown as experimental studies of stress and sleep have yielded mixed results. Potential mechanisms linking stress to poor sleep are also poorly characterized. The current study aimed to 1) assess the impact of experimentally-induced mental stress on daytime sleep and 2) test candidate mechanisms including physiological and emotional stress reactivity, stress rumination, attentional threat bias, and insensitivity to future consequences.

Methods: Participants (N=30) were randomized to a control (n=14) or stress group (n=16). Both groups were given a 60-minute nap opportunity at midday (\approx 13:30). Prior to sleep, participants in the stress group completed a socially evaluative mental arithmetic stress task and were instructed that they would be required to give a brief speech upon awakening. Sleep was monitored with polysomnography and scored

according to standard AASM criteria. Measures of heart rate (HR), blood pressure (BP) and self-reported stress were recorded during the stress task. Self-reported stress rumination was measured upon awakening. Attentional threat bias was measured using an emotional dot probe and performance on the Iowa Gambling Task quantified insensitivity to future consequences.

Results: Acute mental stress significantly increased HR and BP (all p<.001, all Cohen's d>1.24) and participants reported significant increases in self-reported stress (p<.001). The stress group exhibited longer sleep latency (p=.038, d=.82), shorter sleep duration (p=.044, d=.78), and worse sleep continuity (p=.045, d=.79). Subjective sleep quality was not different across groups (p=.39, d=.32). Of all candidate mechanisms, physiological reactivity was the only one significantly related to sleep measures. Greater HR reactivity predicted longer sleep latency (r=.37), shorter sleep duration (r=.59), and worse sleep continuity (r=.59).

Conclusion: Acute mental stress caused significant disturbances in a single episode of daytime sleep. The degree of disturbance was, to an extent, predicted by the amount of physiological reactivity to stress.

Support: This work was supported a Washington and Lee University Summer Lenfest Grant and the Summer Research Scholars Program.

0163

INVESTIGATING THE ROLE OF VASOACTIVE INTESTINAL PEPTIDE-CONTAINING NEURONS OF THE VENTROMEDAL PREOPTIC AREA IN SLEEP-WAKE CONTROL

Venner, A. Fuller, P. M.

Harvard Medical School/ Beth Israel Deaconess Medical Center, Boston, MA.

Introduction: A role for vasoactive intestinal peptide (VIP) in promoting rapid eye movement (REM) sleep has been suggested, but the anatomical location of the neurons that release VIP to promote REM sleep has not been identified. Here, we investigated the role of VIP-containing cell groups in the ventromedial preoptic area (VMPO^{VIP}) in sleep-wake regulation. The VMPO has also previously been implicated in thermoregulation and the febrile response.

Methods: We first investigated the native firing activity of VMPO^{VIP} neurons, over repeated sleep-wake cycles, using *in vivo* fiber photometry in *VIP-ires-Cre* mice. We next examined the afferent and efferent profile of this cell group using conditional retrograde (pseudotyped modified rabies) and anterograde (adeno-associated viral vector-based) tracers. We finally utilized a chemogenetic strategy to selectively activate VMPO^{VIP} neurons cells while monitoring electroencephalogram/electromyogram activity and core body temperature, in order to determine their role in sleep-wake and thermoregulatory control.

Results: We found that VMPO^{VIP} cells were predominantly and strikingly REM-active, that they received many synaptic inputs from surrounding hypothalamic regions (including the ventromedial hypothalamus, dorsomedial hypothalamus and the arcuate nucleus), and that they targeted established sleep-wake nodes, such as the ventrolateral preoptic nucleus, tuberomammillary nucleus, lateral hypothalamus and ventrolateral periaqueductal gray area. To our surprise, chemogenetic activation of the VMPO^{VIP} cell population had little effect upon all measures of sleep-wake analysed and no effect upon core body temperature. **Conclusion:** We conclude that VMPO^{VIP} neurons do not promote REM sleep per se. However, their REM-active profile and anatomical connectivity suggest that these neurons may play a functional role in generating certain cardinal features of REM sleep, which is an active focus of on-going research in our laboratory.

Support: SRSF CDA #016-JP-17 to A.V. and NS073613, NS092652 and NS103161 to P.M.

0164

EVIDENCE SUGGESTING EARLY AIRWAY COLLAPSE AS CAUSE OF SPONTANEOUS AROUSALS

Rechul, D.¹ Rechul, K.²

¹Yale University, New Haven, CT, ²DWI, Colorado Springs, CO.

Introduction: Spontaneous arousals can occur in response to a number of stimuli like noise, movement, hypoxia, or airway obstruction. Some arousals occur "spontaneously" and in individuals donning a hyper-arousable phenotype, spontaneous arousals can dominate the sleep architecture. While arousal mechanisms for some stimuli have been well described, there is a profound lack of knowledge to explain spontaneous arousals. During clinical testing of a device that was designed by SleepMethods, Inc. to anticipate obstructive sleep apnea events by the ability to sense minute airway caliber changes, it was noted, incidentally that the device would signal impeding airway collapse but a spontaneous arousal followed the signal before an obstructive airway event ever developed. This phenomenon was observed many times within and between subjects, suggesting the possibility that very early airway changes are causing "spontaneous arousals"

Methods: Ten adults (7M;3F) aged 18-80y/o (avg. 54.7y/o) with a known AHI \geq 15/hr (avg. AHI = 42.6/hr) underwent 1 overnight PSG recording while wearing the device. Patients were required to forego their usual CPAP therapy on the night of study in efforts to expose the device to an adequate number of total obstructive events (defined as apneas and hypopneas; RERAs and snores were excluded). Standard PSG analysis was performed. Scoring rules were applied to determine whether signals were true/false positives and/or true/false negatives based on pre-clinical data showing anticipation accuracy for up to 45 seconds prior to an obstructive airway event. Signals designed to herald obstructive events were noted, incidentally, appearing prior to spontaneous arousals.

Results: Preliminary results suggest that early phases of airway collapse, as the airway progresses from patency to clinically significant obstruction, are causing EEG arousals which, by current standards, are considered "spontaneous". Because these findings were incidental to another primary purpose of the clinical study, data analysis is in early stages but currently suggesting at least an associative relationship.

Conclusion: If final data analysis shows statistically significant correlation between early airway collapse and "spontaneous arousals", it may have tremendous implications for patients with hyper-arousability, insomnia, and/or pathologically elevated spontaneous arousal indices by proposing therapies aimed at airway patency maintenance. **Support:** N/A

0165

THE RELATIONSHIP BETWEEN ANXIETY SYMPTOMS AND SLEEP QUALITY: MEDIATING AND MODERATING FACTORS OF PRE-SLEEP AROUSAL AND ANXIETY SENSITIVITY

Nagy, S. Pickett, S. M. Hedge, M. Mesa, J. Mechal, R. Florida State University College of Medicine, Tallahassee, FL.

Introduction: The relationship between anxiety and sleep has been well-established, with many studies demonstrating the relationship

between anxiety and reduced REM time, increased sleep latency, and reduced sleep efficiency. Anxiety sensitivity, or the fear of experiencing anxiety-related sensations, has also been associated with increased sleep latency and sleep dysfunction. This delay in sleep onset may be explained by increased arousal, both physical and cognitive, immediately before sleep related to worry or anxiety. The current study examined the relationship between anxiety and subjective sleep quality through pre-sleep arousal and investigated the moderating effect of anxiety sensitivity.

Methods: Participants (n=322) were recruited from Amazon's MTurk site, with most identifying as female (58.5%) and White/Caucasian (84.4%) and with an average age of 37.51 (SD = 12.12). Participants completed the Daily Assessment of Symptoms - Anxiety Scale (DAS-A), the Anxiety Sensitivity Index (ASI), the Pre-Sleep Arousal Scale (PSAS) and the Pittsburg Sleep Quality Index (PSQI). A moderated mediation analysis was conducted using Model 58 in PROCESS for SPSS.

Results: Results indicated that pre-sleep arousal partially mediated the relationship between anxiety and the dichotomous sleep quality variable while controlling for the covariates of age, and therapy participation, (a1= .186, p<.001, b1= .113, p<.001, c'= .038, p<.001). Additionally, the conditional effects and interactions of the moderating anxiety sensitivity variable were significant in both pathways (a1: F=24.702, p<.001; b1: χ^2 = 5.255, p=0.22).

Conclusion: These results help to identify potential mechanisms and conditions of the relationship between anxiety and sleep quality and contribute to the development of evidence-based interventions for sleep disturbance or other sleep complaints in individuals experiencing anxiety and anxiety symptoms.

Support: N/A

SLEEP INCONSISTENCY RELATED CHANGES IN BRAIN FUNCTION DURING TASK AND REST

Zhang, R.¹ Tomasi, D.¹ Shokri-Kojori, E.¹ Wiers, C. E.¹ Wang, G.¹ Volkow, N. D.²

¹National Institute on Alcohol Abuse and Alcoholism,

Laboratory of Neuroimaging, National Institutes of Health, Bethesda, MD, ²National Institute on Drug Abuse, National Institutes of Health, Bethesda, MD.

Introduction: Sleep deprivation and circadian disruptions impair brain function and cognitive performance, but few studies have examined the effect of sleep inconsistency. Here we investigated how inconsistent sleep duration and sleep timing between weekend (WE) and weekdays (WD) affected behavior and brain function during task and at rest in 56 (30 female) healthy human subjects. **Methods:** WE-WD differences in sleep duration and sleep midpoint were calculated using one-week actigraphy data. All subjects underwent 3Tesla BOLD-fMRI to measure brain activity during a visual attention task (VAT) and in resting-state condition.

Results: We found that WE-WD inconsistency of sleep duration and sleep midpoint were uncorrelated with each other (r=.08, p=.58) and influenced behavior and brain function differently. Our healthy subjects showed relatively small WE-WD differences (WE-WD: 0.59 hours) and benefited from longer WE catch-up sleep, which was associated with better attentional performance (3-ball: β =.30, t=2.35, p = .023; 4-ball: β =.30, t=2.21, p =.032) and greater deactivation of the default mode network (DMN) during VAT (p < .05, cluster-corrected) and greater resting-state functional connectivity (RSFC) between anterior DMN and occipital cortex (p < .01, cluster-corrected). In contrast, inconsistent WE-WD sleep midpoint (WE-WD: 1.11 hours) was associated with worse performance (4-ball: β =..33, t=-2.42, p = .020) and with lower occipital activation during VAT and lower RSFC within the DMN.

Conclusion: Our results document the importance of consistent sleep timing for brain function in particular of the DMN, and provide evidence of the benefits of WE catch-up sleep in healthy adults. **Support:** This work was supported by NIAAA IRP (Y01AA3009). R.Z. received research fellowship from German research foundation (DFG).

0167

THE EFFECT OF NOCTURNAL DINNER TYPE "LIGHT" VERSUS "HEAVY" ON SLEEP, ATTENTION AND MICROBIAL COMPOSITION

Green, A.^{1,2} Sher, S.² Siri, N.² Mizrahci, N.² Koren, O.³ Dagan, Y.^{1,2} ¹The Sleep and Fatigue Institute, Assuta Medical Center, Tel-Aviv, ISRAEL, ²The Research Institute of Applied Chronobiology, The Academic College of Tel-Hai, Tel-Hay, ISRAEL, ³Azrieli Faculty of Medicine, Bar-Ilan University, Safad, ISRAEL.

Introduction: Nutrition and sleep are two essential functions for the physiological existence of the organism. Furthermore, both have an acquired cultural, educational and social behavioral component. This study examined the effect of nocturnal dinner type ("light" vs. "heavy") on the quality and quantity of sleep, attention abilities, fatigue, and mood in the following morning. In addition, the microbial composition was examined.

Methods: Twenty healthy subjects (10 men and 10 women), aged 25–33, were invited to two non-consecutive nights at the Sleep Institute for polysomnography test and filling out questionnaires: KSS; ESS; and Brief Symptom Inventory (BSI), attention abilities

assessed with CPT-III. In one evening, the subjects consumed two hours before bedtime a "light" dinner based on vegetable ingredients (vegetables and vegetable proteins) with 342 calories that contained lentils, feta cheese, beet, and other vegetables. On the other evening, the subjects consumed two hours before bedtime a "heavy" dinner based on carbohydrates, fats, and animal protein with 501 calories that contained hamburger and French fries. In addition, subjects were required to give a microbial test before and after meals.

Results: There was no significant difference in the various sleep parameters between the two nights after each meal type: efficacy (t=-1.51,p=0.15); sleep latency stage 1 (t=1.81,p=0.08); sleep latency stage 2 (t=1.00,p=0.33); REM latency (t=0.57,p=0.57); total sleep time (t=-1.57,p=0.13); number of awakenings (t=0.30, p=0.76). No significant differences were found for: fatigue (KSS) (t=-0.30,p =0.77); sleepiness (ESS) (t=0.76,p =0.45); mood (BSI) (t=0.87,p=0.39); and attention deficit (CPT-III) (t=-0.68,p=0.50). The type of night meal did not show a significant effect on the microbial composition (H=0.059,p= 0.81).

Conclusion: The findings of this study show that, contrary to popular belief, "heavy" dinner did not affect the quality of sleep and functions measured in the study compared to a "light" dinner. In the current research, the population that was examined included only young and healthy subjects, therefore, the results may differ if the research in this field will extend and include other populations such as adults and subjects with different disorders. Future studies are needed to examine the relationships between sleep and nutrtion. **Support:** No support

0168

EFFECTS OF SCHOOL NIGHT SLEEP DURATION AND CIRCADIAN PREFERENCE ON STUDENT TARDINESS: AN INVESTIGATION IN A MIDDLE-SCHOOL AGED SAMPLE *Tran, K. M.¹ Cook, J. D.^{1,2} Blair, E. E.³ Peppard, P. E.⁴ Plante, D. T.^{1,2}* ¹University of Wisconsin School of Medicine and Public Health, Department of Psychiatry, Madison, WI, Madison, WI, ²University of Wisconsin, Department of Psychology, Madison, WI, ²University of Wisconsin, Department of Psychology, Madison, WI, Madison, WI, ³University of Wisconsin-Whitewater, Department of Educational Foundations, Whitewater, WI, Whitewater, WI, ⁴University of Wisconsin School of Medicine and Public Health, Department of Population Health Sciences, Madison, WI, Madison, WI.

Introduction: Sleep and circadian factors play an important role in school attendance, academic performance, and daytime behaviors among adolescents. This investigation assessed school night sleep duration (SNSD) and circadian preference (CP) association with first period tardies (FPT) using a middle-aged sample from the Madison (Wisconsin) Metropolitan School District (MMSD), prior to implementation of a planned district-wide delay in middle school start times. Methods: 4,175 middle-school aged students from 12 MMSD schools completed a sleep survey, which included SNSD and a validated 4-level measure of CP. Self-reported SNSD between 4-and-12 hours served as final sample inclusion criterion. Mixed effects modeling was employed with students nested within school. Linear regression determined SNSD and CP effect on student tardiness. Individual, year-long FPT served as outcome variable. Inclusion of SNSD quadratic term was not statistically indicated. Full model covariates included age, sex, race, parent educational level, homelessness, free and reduced lunch, and special education status.

Results: Final sample included 3,860 students. Univariate regression determined a significant CP association with FPT [β =1.20, 95% CI (0.54, 1.86), F(1,10.41)=13.7, p=0.004), but not SNSD [β =-0.31, 95% CI (-0.70, -0.09), F(1,10.21)=2.5, p=0.14]. SNSD and CP interaction

was not significant. CP significance was maintained in the full model $[\beta=1.24, 95\%$ CI (-0.70, -0.09), F(1,11.21)=13.7, p=0.004]. Evening preference associated with 3.72 more FPT, relative to morning preference. **Conclusion:** Results suggest evening preference is associated with increased risk of tardiness among middle school students. Future research that examines the relationships between delayed school start times, circadian preference, and impact on school tardiness is indicated. **Support:** This research was generously supported by a grant from the Madison Education Partnership (MEP).

0169

DISRUPTION OF SLEEP ARCHITECTURE AND CIRCADIAN RHYTHMS FOLLOWING HIGH FREQUENCY HEAD IMPACTS

Korthas, H. T. Main, B. S. Harvey, A. C. Wicker, E. W. Sloley, S. S. Burns, M. P. Laboratory for Brain Injury and Dementia, Georgetown

University, Washington, DC.

Introduction: Mild Traumatic Brain Injury (mTBI) can cause a broad array of behavioral problems including cognitive and emotional deficits. Sleep disturbances including disrupted sleep latency and efficiency are common amongst human mTBI patients. Crucially, sleep plays a key role in hippocampal learning and memory consolidation, yet the contribution of single and repetitive mTBIs influencing sleep related cognitive outcomes remains unclear.

Methods: To study the effect of repetitive mTBI on sleep and circadian rhythms, C57Bl/6 mice underwent sham or High Frequency-Head Impact (HF-HI, 30 closed head impacts, 5/per day for 6-days) procedures before brains were assessed at 1d, 1m and 2m using a combination of molecular neurobiology (RNA/protein), EEG/ EMG recordings and behavioral analysis.

Results: HF-HI induces learning and memory deficits in the Barnes and T-Maze at both 1d and 1m post injury, in the absence of axonal injury, inflammation, or protein deposition. Disruptions in circadian mRNA expression was identified at multiple time points post HF-HI. RNA analysis of mouse cortex, hippocampus, and hypothalamus for core circadian rhythm genes (Bmal1, Clock, Cryptochrome 2, Period1 and Period 2) was conducted at 1d and 1m post HF-HI. We found dysregulated expression of these core biological clock genes in these regions at both time points. Furthermore, we find distinct changes to sleep architecture chronically post injury. Animals were implanted with EEG and EMG for monitoring at 2m post injury. EEG and EMG signals were coded for wake, NREM, and REM. One-month post injury, HFHI injured mice showed dysregulated sleep architecture compared to sham mice, while both groups had the same total sleep time. We also demonstrate that HF-HI alters EEG activity in awake animals.

Conclusion: Overall, our data shows disruptions in both sleep architecture and expression of circadian rhythm genes following HF-HI. This opens up an important avenue of potential therapeutic intervention following injury.

Support: If deficits in sleep and circadian rhythms can be rescued after mTBI, it may assist in improving symptoms and chronic outcomes after injury.

0170

ASSOCIATION OF HIF-1 ALPHA AND BETA SUBUNITS WITH CLOCK AND BMAL1 PROTEINS IN OBSTRUCTIVE SLEEP APNEA PATIENTS

Gabryelska, A. Sochal, M. Turkiewicz, S. Bialasiewicz, P. Department of Sleep Medicine and Metabolic Disorders, Medical University of Lodz, Lodz, POLAND. Introduction: Obstructive sleep apnea (OSA) is a chronic condition that is characterized by intermittent hypoxia. Key regulator of oxygen metabolism is hypoxia inducible factor (HIF), which consists of oxygen sensitive subunit and continuously produced subunit. Circadian clock is composed of set of genes, which function as activators - CLOCK and BMAL 1, who similarly to HIF are basic helix-loop-helix-PER-ARNT-SIM transcription factors. Therefore, the aim of the study was to assess the relationship between HIF-1alpha, HIF-1beta, CLOCK, BMAL1 and polysomnography (PSG) variables in healthy individuals and severe OSA patients. Methods: The study included 20 individuals, who underwent PSG and based on apnea-hypopnea index (AHI) were divided into severe OSA group (n=10; AHI30; 90% male) and healthy control (n=10; AHI<5; 70% male). All participants had their peripheral blood collected in the evening (9:00-10:00 pm) before and in the morning (6:00-7:00 am) after the PSG. HIF-1alpha, HIF-1beta, CLOCK and BMAL1 protein concertation measurements were performed using ELISA. Results: Significant difference was observed in the following protein measurements between study groups: evening and morning HIF-1 (p=0.020 and p=0.043, respectively), evening HIF-1alpha (p=0.047), evening and morning CLOCK (p=0.037 and p=0.019, respectively) and morning BMAL1 (p=0.016). No differences were observed between morning and evening protein levels in both groups. Evening HIF-1beta corraleted with evening CLOCK and morning BMAL1 (R=0.511, p=0.21 and R=0.594, p=0.006, respectively), while morning HIF-1 with evening BMAL1 (R=474, p=0.35). Furthermore, evening and morning HIF-1 correlated with evening BMAL1 (R=564, p=0.010 and R=0.689, p=0.001, respectively). Additionally, morning CLOCK and BMAL1 correlated with AHI (R=0.510, p=0.022 and R=0.560, p=0.010, respectively) and desaturation index (R=0.487, p=0.209 and R=0.570, p=0.009, respectively). Conclusion: There is significant correlation between both subunits of HIF-1 protein and circadian clock proteins: CLOCK and BMAL1, which further correlate with increased disease severity. This suggests OSA patients are in risk of circadian clock disruption due to present hypoxia.

Support: The study was financed by Polish National Centre Grant no. 2018/31/N/NZ5/03931.

0171

THE IMPACT OF SHORT-TERM BRIGHT GREEN LIGHT EXPOSURE IN PARTIALLY SLEEP-DEPRIVED PERSONS

Pilcher, J. J. Bisson, J. B. Scircle, E. J.

Clemson University; Department of Psychology, Clemson, SC.

Introduction: Many workers and students experience regular sleep deprivation and daytime sleepiness when they are expected to be performing at their best. The purpose of the current research was to investigate the potential effect of short-term bright green light exposure on daytime performance and sleepiness in partially sleepdeprived persons.

Methods: Participants were 30 students (19.4 ± 0.89 years; 20 females). On Day 1, participants were loaned a Fitbit to provide an objective measure of activity/sleep and were instructed to sleep no more than 5 hours that night. On Day 2, participants provided information on their sleep time for the previous night and were randomly assigned to a bright light condition (bright green light, 381 Lux) or a standard light condition (control condition, indoor florescent light, 139 Lux). They completed a simple reaction time task, the Arrow Flankers task to measure cognitive inhibition, and the Stanford Sleepiness Scale during three testing periods (baseline, after 20 minutes of light exposure, and after 60 minutes of

light exposure). Between the testing periods, participants read a non-fiction book while exposed to their respective light condition. At the end of the study, participants returned their FitBit.

Results: The Fitbit and self-report sleep data indicated that participants slept 4.9 hours the night between Day 1 and Day 2. Mixed 2 (light condition) x 3 (testing period) ANOVAs indicated that the bright green light condition resulted in a significant decrease in reaction time on the Flankers task but had no significant effect on simple reaction time or subjective sleepiness.

Conclusion: The current findings suggest that 60 minutes of bright green light exposure could improve performance on a cognitive inhibition task. This suggests that bright green light exposure could be a useful countermeasure for cognitive performance decrements in settings where sleep deprivation is common. **Support:** None to report.

0172

BLUE-LIGHT BLOCKERS AND SLEEP: A META-ANALYSIS OF INTERVENTION STUDIES

Shechter, A.¹ Quispe, K. A.¹ Mizhquiri Barbecho, J. S.¹ Falzon, L.² ¹Columbia University Medical Center, New York, NY, ²Northwell Health, New York, NY.

Introduction: Sleep and circadian physiology are influenced by external light, particularly within the short-wavelength portion of the visible spectrum (~450–480 nm). Most personal light-emitting electronic devices (e.g., tablets, smartphones, computers) are enriched in this so-called "blue" light. Interventions to reduce short-wavelength light exposure to the eyes before bedtime may help mitigate adverse effects of light-emitting electronic devices on sleep. **Methods:** We conducted a meta-analysis of intervention studies on the effects of wearing color-tinted lenses (e.g., orange or amber) in frames in the evening before sleep to selectively filter short-wavelength light exposure to the eyes. Outcomes were self-reported or objective (wrist-accelerometer) measures of nocturnal sleep. Databases (MEDLINE, EMBASE, Cochrane Library, PsycINFO, CINAHL, AMED) were searched from inception to November 2019. PROSPERO Registration: CRD42018105854.

Results: Ten studies were identified (7 randomized controlled trials; 3 before-after studies). Findings of individual studies were inconsistent, with some showing benefit and others showing no effect of intervention. For objective sleep onset latency, there was a significant modest-sized combined effect (Hedge's g=-0.52, 95% CI: -1.27-0.24, Z=-2.94, p=0.003, I²=16.6%, k=3). There was a minor but non-statistically significant combined effect for objective sleep efficiency (Hedge's g=0.24, 95% CI: -0.16–0.64, Z=1.69, p=0.09, I²=23.7%, k=5). There were no significant combined effects for objective measures of total sleep time and wake after sleep onset. For self-reported total sleep time, there was a statistically significant medium-sized combined effect (Hedge's g=0.61, 95% CI: 0.14–1.09, Z=5.56, p<0.01, I²=0%, k=3).

Conclusion: There is mixed evidence that this approach can improve sleep. Relatively few studies have been conducted, and most did not assess light levels or melatonin. The "blue-blocker" intervention may be particularly useful in individuals with insomnia, delayed sleep phase syndrome, or attention-deficit hyperactive disorder. Considering the ubiquitousness of short wavelength-enriched light sources and the potential for widespread sleep disturbance, future controlled studies examining the efficacy of this approach to improve sleep are warranted. **Support:** N/A

0173

SPRING FORWARD, FALL BACK: INCREASED PATIENT SAFETY-RELATED ADVERSE EVENTS FOLLOWING THE SPRING TIME CHANGE

Kolla, B. Coombes, B. J. Morgenthaler, T. I. Mansukhani, M. P. Mayo Clinic, Rochester, MN.

Introduction: "Spring forward," the start of daylight savings time (DST) reduces sleep opportunity by an hour. The resulting sleep deprivation in healthcare workers can increase the potential for medical errors. We examined the change in patient safety-related adverse events (AEs) following the time change in both spring and fall.

Methods: Self-reported AEs that occurred 7 days prior to and following the spring and fall time changes for years 2010–2017 in a large healthcare organization were ascertained. AEs likely resulting from human errors were identified. The change in the number of AEs (all AEs or restricted to those resulting from human error) following the spring and fall time change were modeled using negative binomial mixed models using a random effect to correct for nonindependent observations in consecutive.

Results: Over the 8 year period, there were more AEs (all and human) in the 7 days following the change in time both in spring (All: 2812 V. 2699; Human: 1902 V. 1625) and fall (All: 3207 V. 3007; Human: 2189 V. 2087). However, the only statistically significant increase was for the estimated 18% increase in human errors following time change in spring (95% CI: 6% to 34%; p = 0.004). The 18% AE increase in spring was also significantly greater than the 5% increase in AE in fall (p = 0.018).

Conclusion: There is a significant increase in human error related AEs following the "spring forward" clock change which can jeopardize patient safety. Based on safety considerations, DST might best be eliminated; alternatively, policy makers and healthcare organizations should evaluate measures to mitigate the increased risk during this period. **Support:** NA

0174

EXAMINING CIRCADIAN DISADVANTAGES IN THE NATIONAL BASKETBALL ASSOCIATION'S PLAYOFFS

Pradhan, S.¹ Gregory, K.² Alton, D.¹ Chachad, R.⁴ Flynn-Evans, E. E.² ¹Menlo College, Atherton, CA, ²NASA Ames Research Center, Moffett Field, CA, ³Menlo College, Atherton, CA, ⁴University of California, San Francisco, San Francisco, CA.

Introduction: Prior research on travel in the National Basketball Association's (NBA) regular season has shown that teams journeying west relative to their home base face circadian disadvantages for evening games, while those traveling east have advantages. The current study extends previous research by examining these effects within the NBA playoffs. We hypothesized that teams would have a greater circadian advantage during eastward compared to westward travel. Methods: In 2013, the NBA implemented a 7-game series playoff structure, in which teams play an alternating home/away 2-2-1-1-1 format. Data for all 499 postseason games played during the 2013-14 to 2018–2019 seasons were collected from Basketball-Reference and FiveThirtyEight. We investigated the impact of direction of travel based on home base city (same time zone, westward, eastward) and time zones traveled on game outcomes, Elo rating differences (i.e., a team quality metric based on wins and losses), win probability, and team scoring.

Results: Teams had lower win probabilities following 3-hour westward than same time zone and all eastward travel, while 3-hour eastward travel related to higher probabilities of winning compared to same time and all westward travel (p < .001, d > .95). Teams travelling westward with 2-hour time changes lost significantly more games than those experiencing 1-hour westward (p = .04, OR = 2.45), 1-hour eastward (p = .05, OR = 2.34), and 3-hour eastward changes (p = .02, OR = 4.68). Scoring was significantly higher following eastward travel compared to both westward (p = .001, d = 0.60) and same time zone travel (p = .003, d = 0.44). There were no differences in team quality based on direction of travel or number of time zones traveled, and game outcomes based on overall direction of travel (p > .05).

Conclusion: Direction and magnitude of travel were related to win probability, team scoring, and game outcomes, whereby teams travelling eastward and within the same time zone gained an advantage over those travelling westward. Adjustment to travel and time changes appear to influence in-game performances and outcomes in the NBA playoffs.

Support: None

0175

LIGHT IMPROVES ALERTNESS AND MOOD DURING THE SLEEP INERTIA PERIOD FOLLOWING SLOW WAVE SLEEP

Hilditch, C. J.¹ Feick, N. H.¹ Wong, L. R.¹ Bathurst, N. G.¹ Flynn-Evans, E. E.²

¹Fatigue Countermeasures Lab, SJSU Research Foundation, Moffett Field, CA, ²Fatigue Countermeasures Lab, NASA Ames Research Center, Moffett Field, CA.

Introduction: Waking from sleep, especially slow wave sleep (SWS), is associated with reduced alertness known as sleep inertia. Light improves alertness during sleep deprivation and circadian misalignment. In this study, we assessed the efficacy of light to improve alertness and mood immediately after waking from SWS.

Methods: Twelve participants kept a sleep schedule of 8.5 h for 5 nights and 5 h for one night prior to the overnight laboratory visit (confirmed by actigraphy). Participants went to bed at their scheduled habitual bedtime in the laboratory and were monitored by standard polysomnography. After at least 5 min of SWS, participants were awoken and exposed to either red ambient light (control) or blueenriched bright light (light) for 1 h. During this time, participants completed a subjective scale of alertness (Karolinska Sleepiness Scale, KSS) and visual analogue scales (VAS) of mood at 2 min, 17 min, 32 min, and 47 min after waking. Following this sleep inertia measurement period, all lights were turned off and participants were allowed to return to sleep. They were then awoken again from their subsequent SWS period and exposed to the opposite condition (control or light). A linear mixed-effects model with fixed effects of condition, time, and condition*time and a random effect of participant was used to determine the impact of light across the testing period. An average of baseline responses (pre-sleep) was included as a covariate.

Results: Compared to the control condition, participants exposed to blue-enriched bright light reported feeling more alert (KSS: $F_{1,77}$ =4.955, p=.029; VAS_{alert}: $F_{1,77}$ =8.226, p=.005), more cheerful (VAS_{cheerful}: $F_{1,77}$ =8.615, p=.004), less depressed (VAS_{depressed}: $F_{1,77}$ =4.649, p=.034), and less lethargic (VAS_{lethargic}: $F_{1,77}$ =5.652, p=.020).

Conclusion: Exposure to blue-enriched bright light immediately after waking from SWS may help to improve subjective alertness and mood. Future analyses will explore whether these findings extend to effects on cognitive performance.

Support: Naval Postgraduate School Grant. NASA Airspace Operations and Safety Program, System-Wide Safety Project.

0176

SHORT SLEEP DURATION AND POOR SLEEP QUALITY PREDICT BLUNTED WEIGHT LOSS IN A BEHAVIORAL WEIGHT LOSS INTERVENTION

Kline, C. E.¹ Lambiase, M. J.¹ Conroy, M. B.² Brooks, M. M.¹ Kriska, A. M.¹ Barinas-Mitchell, E. J.¹

¹University of Pittsburgh, Pittsburgh, PA, ²University of Utah, Salt Lake City, UT.

Introduction: Short sleep duration and poor sleep quality have each been associated with obesity and weight gain. However, less is known regarding how sleep may impact attempted weight loss. The purpose of this study was to investigate the associations between sleep duration and sleep quality, both independently and in combination, with weight loss in a 12-month behavioral weight loss intervention.

Methods: Young to middle-aged adults who were overweight or obese (N=296) completed a 12-month behavioral weight loss intervention, with weight assessed at baseline, 6 and 12 months. Sleep duration and quality were derived from the Pittsburgh Sleep Quality Index. Analyses examined the change in sleep over time and the association between baseline sleep and changes in sleep with 6- and 12-month weight loss following adjustment for relevant covariates including age, gender, race, education, baseline body mass index, and baseline risk for sleep apnea.

Results: Participants (with an average baseline weight of 97.0 ± 1.0 kg) lost 6.6 ± 1.1 kg (6.8%) and 6.7 ± 1.2 kg (6.9%) at 6 and 12 months relative to baseline, respectively. Global sleep quality significantly improved over the 12-month intervention (P=.03), but average sleep duration and the prevalence of short sleep duration (<6 h) or poor sleep quality did not change significantly (each P≥.45). Adults with short sleep duration at baseline lost $3.3\pm0.9\%$ less weight than those with ≥6 h sleep duration (P<.001). Adults with poor sleep quality at baseline lost $1.6\pm0.8\%$ less weight than those with good sleep quality (P=.04). When considered together, adults with both short sleep duration and poor sleep quality lost at least 5.0% less weight compared with all other sleep duration/quality group combinations (P<.001).

Conclusion: Our findings highlight the importance of both sleep duration and sleep quality as predictors of behavioral weight loss and suggest that screening for sleep disturbance may be useful to determine who may benefit from additional counseling and resources. **Support:** R01HL077525, K23HL118318

0177

MEASURING SLEEP HEALTH ACROSS SURVEY, ACTIGRAPHY, AND DIARY METHODS IN YOUNG ADULTS

Jakubowski, K. P.¹ Wright, A. G.² Matthews, K. A.^{1,2,3}

¹Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA,
 ²Department of Psychology, University of Pittsburgh, Pittsburgh, PA,
 ³Department of Epidemiology, University of Pittsburgh, Pittsburgh, PA.

Introduction: Sleep health is a relatively new construct including multiple dimensions of nighttime and daytime sleep across the 24-hour day. To date, few studies have investigated sleep health across various types of sleep measures. We collected sleep health data using survey, actigraphy, and daily diary, affording the unique ability to compare the latent factor structure of the sleep health construct across methods in a single sample of healthy young adults. **Methods:** Undergraduates (N=540; 50% female; 71% white; mean age=18) self-reported sleep health via the 6-item RUSATED survey (Buysse, 2014), which queries "typical" sleep patterns including: day-time alertness and nocturnal sleep regularity, satisfaction, timing, efficiency, and duration (0=rarely/never to 2=usually/always); total

score=0–12 (higher=better sleep health). A subsample (N=114) provided 7-day actigraphy and daily diaries, which were used to derive weeklong averages of the aforementioned sleep dimensions. Confirmatory factor analysis was conducted to assess model fit of three factors (survey, actigraphy, diary), each including withinmethod indicators of the six sleep dimensions; note the actigraphy model included diary satisfaction. Acceptable model fit was assessed using established criteria (e.g., Bentler, 1990): RMSEA < .08, CFI > .90, and TLI > .90. Maximum likelihood estimation was used.

Results: Average RUSATED total score was 7.1 (SD=2.5). Average actigraphy [diary] sleep dimensions were: Alertness (i.e., proportion of days napped)=21% [25%]; Duration=6.2hr [7.1hr]; Timing (i.e., midpoint)=5:00am [3:30am]; Regularity (i.e., SD of midpoint)=56 min [40 min]; Efficiency= 82% [95%]; Satisfaction="average" to "good". The survey model demonstrated acceptable fit (RMSEA [90%CI] = .06 [.03, .09], CFI=.96, TLI=.92) after correlating regularity and timing, as suggested by modification indices; factor loadings [λ (SE)] ranged from .27(.05) for efficiency to .71(.04) for satisfaction. Fit was poor for the diary model, whereas the actigraphy model failed to converge.

Conclusion: Sleep health models demonstrated adequate fit using survey but not actigraphy or diary data. The lack of acceptable fit for the latter may reflect differences in the measurement timeframe (e.g., "typical" sleep vs. 7-day averages) or the smaller sample size for the actigraphy/diary measures. Determining how to best measure sleep health and ultimately apply it to health-relevant outcomes is a valuable research agenda. **Support:** T32HL07560; T32MH018269

0178

THE CYCLE OF DAILY STRESS AND SLEEP: SLEEP MEASUREMENT MATTERS

Slavish, D. C.¹ Asbee, J.¹ Veeramachaneni, K.² Messman, B.¹ Scott, B.¹ Walker, J.¹ Sin, N. L.³ Taylor, D. J.⁴ Dietch, J.⁵ ¹University of North Texas, Denton, TX, ²St. Louis University, St. Louis, MO, ³University of British Columbia, Vancouver, BC, CANADA, ⁴The University of Arizona, Tucson, AZ, ⁵Palo Alto Veterans Affairs Health Care System, Palo Alto, CA.

Introduction: Disturbed sleep can be both a cause and a consequence of increased stress. Yet intensive longitudinal studies have demonstrated that sleep assessed via sleep diaries and actigraphy is inconsistently associated with daily stress. We expanded this research by examining daily associations between sleep and stress using a three-fold approach to assess sleep: sleep diaries, actigraphy, and ambulatory single-channel electroencephalography [EEG].

Methods: Participants were 80 adults (M age = 32.65 years, 63%) female) who completed 7 days of sleep and perceived stress assessments in a naturalistic setting (resulting in 560 possible measurement occasions). Multilevel models were used to examine bidirectional associations between daily stressor occurrence (0 =stressor did not occur, 1 = stressor occurred) and stressor severity (0 = not at all severe to 3 = very severe) and sleep parameters assessed via diary, actigraphy, and EEG (e.g. total sleep time [TST], sleep efficiency [SE], and sleep onset latency [SOL], wake after sleep onset [WASO]). Results: Participants reported at least one stressor on 37% of days. Compared to days without a stressor experienced, days with a stressor were associated with a 14.4-minute reduction in actigraphy-determined TST the subsequent night ($\beta = -0.24$, p = 0.030). Nights with greater sleep-diary determined WASO were associated with greater next-day stressor severity ($\beta = 0.01$, p = .026). No EEG-determined sleep parameters were associated with next-day stressor occurrence or severity, or vice versa.

Conclusion: Daily stress and sleep disturbances occurred in a bidirectional fashion, though specific results varied by sleep measurement technique and sleep parameter. Together, our results highlight that type of sleep measurement matters for examining associations with daily stress. We urge future researchers to treat sleep diaries, actigraphy, and EEG as complementary — not redundant — sleep measurement approaches.

Support: Funding for this study included NIH/NIAID R01AI128359-01; DoD-VA 1I01CU000144-01; the Foundation for Rehabilitation Psychology; and General Sleep Corporation.

0179

POOR SLEEP QUALITY INCREASES ODDS OF ON-DUTY INJURIES IN POLICING

Riedy, S. M.^{1,2} Fekedulegn, D.³ Vila, B.^{4,5} Andrew, M.³ Violanti, J.⁶ ¹Sleep and Performance Research Center, Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ²Unit for Experimental Psychiatry, Division of Sleep and Chronobiology, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, ³Bioanalytics Branch, Health Effects Laboratory Division, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Morgantown, WV, ⁴Sleep and Performance Research Center, Washington State University, Spokane, WA, ⁵Department of Criminal Justice and Criminology, Washington State University, Spokane, WA, ⁶Department of Epidemiology and Environmental Health, School of Public Health and Health Professions, University at Buffalo, The State University of New York, Buffalo, NY.

Introduction: Shiftwork is inevitable in law enforcement. Officers are scheduled around-the-clock to protect and serve communities. Many police departments are also understaffed; consequentially, officers' work schedules often include long work hours. Shift work and long work hours can result in sleep loss, poor sleep quality, and fatigue. In turn, these factors can impair police officers' operational performance. We investigated whether sleep loss and poor sleep quality increase odds of on-duty injuries or disciplinary actions in policing.

Methods: Officers (n=113) that started their careers as police officers at the Buffalo Police Department between 1994–2001 were studied. Work and injury data were obtained for each officer starting with their hire date and continuing day-by-day for 15-years. Between 2004–2009, officers reported any disciplinary actions in the prior two years and their sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI) and Survey Screen for Apnea. Data were analyzed using logistic regression with logit link functions (PROC GLIMMIX, SAS 9.4). Covariates included sex, ethnicity, experience, shift type, workload, and secondary employment.

Results: Seventy-four percent of officers had poor sleep quality (PSQI global score \geq 5). Officers with poorer sleep quality had greater odds of injury (OR=1.3 [95% CI: 1.0–1.5], p=0.03). Officers' sleep duration was not a significant predictor of injuries (OR=1.0 [95% CI: 0.3–3.2], p=0.96). Officers with sleep disturbances (OR=3.5 [95% CI: 1.0–11.8], p<0.05) and/or using sleep medications (OR=15.7 [95% CI: 2.8–89.3], p<0.01) had higher odds of injury. None of the variables were significant predictors of disciplinary actions.

Conclusion: Poor sleep quality was prevalent among the officers. The natures of the injuries were likely multi-factorial and complex. Notwithstanding, poor sleep quality was associated with higher odds of on-duty injuries. The source of officers' sleep disturbances (e.g. shift work, insomnia, and/or policing-related stresses) remains to be determined. Support: CDC/NIOSH grant 1R01OH009640-01A1; NIJ grant 2005-FS-BX-0004

0180

SLEEP, SLEEPINESS, AND SLEEP HYGIENE RELATED TO NOMOPHOBIA (NO MOBILE PHONE PHOBIA)

Peszka, J.¹ Michelle, S.² Collins, B. T.² Abu-Halimeh, N.² Quattom, M.² Henderson, M.² Sanders, M.² Critton, J.² Moore, B.² Mastin, D. F.²

¹Hendrix College, Conway, AR, ²University of Arkansas at Little Rock, Little Rock, AR.

Introduction: Previously, active phone use at bedtime has been implicated in disrupted sleep and related complaints. To improve sleep, a recommendation following such findings is limiting phone use before and during bedtime. However, for those with the characteristic of "nomophobia", fear of being out of mobile phone contact, this recommendation could exacerbate anxiety at and around bedtime and disrupt, rather than improve, sleep. In 2012, an estimated 77% of 18-24-year-olds could be identified as nomophobic. Because of the prevalence of nomophobia and its possible interaction with sleep, we explored the existence of nomophobia in a college-age population and its relationship to sleep, sleepiness, and sleep hygiene behaviors. Methods: 327 university students (age: M=19.7 years, SD=3.78) recruited from introductory psychology courses and campus newsletters were given extra credit or a chance to win \$25 gift cards for participation. Participants completed demographic information, the Nomophobia Questionnaire (NMP-Q), the Epworth Sleepiness Scale (ESS), the Pittsburgh Sleep Quality Index, questions regarding associated features of inadequate sleep hygiene, and the Sleep Hygiene Index. Additional sleep hygiene questions assessed frequency of active and passive technology use during sleep time.

Results: 89.4% of the participants had moderate or severe nomophobia. Greater nomophobia was significantly related to greater daytime sleepiness (ESS) (r(293)=.150, p<.05), associated features of poor sleep (daytime sleepiness: r(297)=.097, p<.05, and avolition: r(297)=.100, p<.05), more maladaptive sleep hygiene behaviors including active technology use during sleep time (r(298)=.249, p<.05), long daytime naps, inconsistent wake and bed times, using bed for non-sleep purposes, uncomfortable bed, and bedtime cognitive rumination (r's=0.097 to 0.182).

Conclusion: Most participants experienced moderate to severe nomophobia with greater nomophobia associated with greater sleepiness, avolition, and poorer sleep hygiene. Nomophobia is likely to be an important consideration when treating sleep disorders and/or making any sleep hygiene recommendations. **Support:** Hendrix College Charles Brewer Fund for Psychology

0181

STRESSOR REACTIVITY TO INSUFFICIENT SLEEP IN ONCOLOGY NURSES: DOES WORK SHIFT MATTER?

Vigoureux, T. F. Lee, S.

University of South Florida, School of Aging Studies, Tampa, FL.

Introduction: Individuals generally perceive more stressors on days following nights with shorter-than-usual sleep duration. Recent research shows that this daily sleep—stress relationship (i.e., stressor reactivity to insufficient sleep) is stronger for some than for others. Workers in certain occupations, such as oncology nurses, may be more prone to insufficient sleep and/or more stressors. Given the impact of work schedule on sleep, this study examined whether

stressor reactivity to insufficient sleep differed between day and night shift nurses working at a cancer hospital.

Methods: Participants were 39 day-shift and 19 night-shift nurses at a cancer hospital (M_{age} =35.36±12.00). Using ecological momentary assessments for 14 days, we asked participants about their previous night's sleep characteristics and their daily stressor frequency and severity before lunch, during afternoon, and before bedtime. Using multi-level modeling, we tested whether previous night's sleep duration, quality, or sufficiency predicted next day's total stressor frequency or severity. For analyses with significant within-person effects, we extracted a reactivity slope for each participant. We used t-tests to examine whether day and night shift nurses differed in reactivity. **Results:** There were significant within-person associations of sleep duration with (a) stressor frequency (b=-.07, p<.001) and (b) stressor severity (b=-.76, p<.001), but no associations of sleep

quality or sufficiency with stressor frequency or severity. Day and night shift nurses did not differ in either of these operationalizations of stressor reactivity to insufficient sleep, or on any stressor or sleep variables except for average sleep duration (M_{night} =6.87±2.57 vs. M_{dav} =8.02±1.84, p<.001).

Conclusion: These findings suggest that the phenomenon of perceiving more stressors in response to insufficient sleep exists regardless of work shifts. Given the previously found association between stressor reactivity to insufficient sleep and body mass index in middle-aged workers, further analyses will test how this reactivity is associated with health outcomes in oncology nurses.

Support: This work was supported, in part, by the University of South Florida College of Behavioral & Community Sciences Internal Grant Program (PI: Lee, Grant No. 0134930).

0182

THE SLEEP REGULARITY QUESTIONNAIRE: DEVELOPMENT AND PRELIMINARY PSYCHOMETRIC PROPERTIES

Donovan, E. K. Dzierzewski, J. M.

Virginia Commonwealth University, Richmond, VA.

Introduction: Sleep is a critically important behavior which influences diverse aspects of health, functioning, and longevity. An increasing literature suggests the importance of sleep regularity, also referred to as sleep inconsistency, sleep variability, or intraindividual variability in sleep. Given there is no brief, subjective measure of sleep regularity, the purpose of this study was to examine the psychometric properties of an in-development, tenitem Sleep Regularity Questionnaire (SRQ).

Methods: In an online study of sleep and health, participants $(n = 3284; M_{age} \text{ (SD)} = 42.74(16.72); 47.8\%$ female; 77.1% white) completed the in-development SRQ, as well as other sleep-related measures including the Insomnia Severity Index (ISI) and the Pittsburgh Sleep Quality Index (PSQI).

Results: An exploratory factor analysis on a random half of the sample revealed a two factor structure, with four items representing "circadian regularity" and two items representing "sleep disturbance regularity." A confirmatory factor analysis on the other random half of the sample fit the two factor model with good model fit indices ($X^2 = 50.9$, df = 7, p < .001; RMSEA = .06; CFI= .99; NFI = .99; IFI = .99; TLI = .98). The SRQ was negatively associated with poor sleep quality measured via the PSQI (r = -.37, p < .001) and negatively associated with insomnia severity measured via the ISI (r = -.40, p < .001).

Conclusion: The SRQ appears to be a valid instrument for the assessment of sleep regularity in adults that is related to, but distinct from, other established sleep constructs. Future research will

benefit from examining test-retest reliability of the measure as well as assessing the validity of the SRQ as a measure of objective sleep regularity by comparing it to conventional diary, actigraphy, and/ or polysomnography methods of sleep assessment.

Support: This work was supported by the National Institute on Aging of the National Institutes of Health under Award Number K23AG049955 (PI: Dzierzewski). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

0183

SOCIAL JET LAG AND CHRONOTYPE: IMPLICATIONS FOR ACTIVITY AND REST RHYTHM AND LIGHT EXPOSURE

Vallim, J. R. Amaral, F. G. D'Almeida, V.

Universidade Federal de São Paulo, São Paulo, BRAZIL.

Introduction: Social jet lag is associated with contemporary lifestyle with harmful impacts on health, predisposing individuals to obesity, diabetes, cardiovascular diseases and cognitive impairment. Knowing that this condition is influenced by chronotype and that these are reflected in rhythmicity markers, a better understating of mechanisms and factors related to this condition are helpful to raise awareness to the society, to prevent and treat its consequences. Our goal was to evaluate if chronotype has a moderating effect on social jet lag, investigate if activity-rest rhythm could be affected by social jet lag and whether it could be related to nighttime light exposure.

Methods: 13 subjects (10 women) aged between 23 and 59 years answered Morningness-Eveningness Questionnaire and record activity and rest rhythm by actimetry.

Results: We observed that chronotype is a predictor of social jet lag (p < 0.05) and eveningness leads to greater social jet lag. Sleep duration and time of sleep offset were higher on free days (p < 0.05) and, as expected, higher activity at night was related to greater social jet lag (p < 0.05). We did not find relation between level and time of exposure to light at night and social jet lag (p > 0.05). None of these differences were chronotype-dependent.

Conclusion: Our work was one of the first that addressed the relationship between social jet lag and a rhythmicity marker, the activity-rest rhythm. We also demonstrated the importance of a better understanding of the evening types, that are more affected by social obligations.

Support: Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES), Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) e Associação Fundo de Incentivo à Pesquisa (AFIP).

0184

PREDICTING STRATEGIC NAPPING IN SURGICAL RESIDENTS BY INDIVIDUAL AND ROTATION CHARACTERISTICS

Devine, J. K.¹ Schwartz, L. P.¹ Hursh, S. R.^{1,2} Mosher, E.³ Schumacher, S.⁴ Boyle, L.⁴ Davis, J. E.⁴ Smith, M.³ Fitzgibbons, S.⁴ ¹Institutes for Behavior Resources, Baltimore, MD, ²John Hopkins University School of Medicine, Baltimore, MD, ³MedStar Institutes for Innovation, Washington, DC, ⁴Georgetown University School of Medicine, Washington, DC.

Introduction: Strategic napping, or napping on-shift, is recommended to reduce fatigue in medical residents. The actual prevalence of strategic napping in relation to residents' workload, schedule, or demographics is largely unquantified. This study objectively measured sleep patterns and work schedules in surgical residents working a variety of service lines over a two-month period in order to identify which resident and service line attributes predict on-shift napping.

Methods: Surgical residents from an academic surgery program in the Washington, DC area provided schedule information, completed the Epworth Sleepiness Scale (ESS), and wore sleep-tracking devices (Zulu Watch) continuously for 8 weeks. Multiple linear regression was performed to predict percent days with on-shift napping from resident demographics (age, gender, post graduate year (PGY), sleep characteristics (ESS, total sleep time (TST), sleep efficiency (SE)), schedule characteristics (shift start time, shift length, rotation length, percent days on-shift, percent night shifts), and service line characteristics (service line category, number of service lines worked).

Results: Twenty-two (n=22) residents completed the study, rotating through at least 1 of 5 different service line categories (Acute Non-Elective Surgery, Elective Surgery, Night Float, Surgical Intensive Care Unit, and Other). Residents slept an average of 6 hours within a 24-hour period (370 ± 129 minutes) with normal SE ($87.13\%\pm7.55\%$). ESS scores indicated excessive daytime sleepiness (11.64 ± 4.03). Ninety-five percent (n=21) of residents napped at least once while on shift. Residents napped on-shift approximately 32% of their working days and were most likely to nap when working between 2300-0500 hours. Earlier shift start times predicted less on-shift napping (B=-0.08,SE=0.04,\beta=-2.40,t=-2.09,p=0.05) while working more night shifts (B=1.55,SE=0.44, β =4.12,t=3.52,p=0.003) and shifts over 24 hours.

Conclusion: Residents take advantage of opportunities to nap on-shift, particularly when working at night. Despite naps, however, residents exhibit insufficient sleep with excessive daytime sleepiness, representing a safety risk to themselves and their patients. **Support:** NA

0185

SLEEP BEHAVIORS AND THOUGHTS AS LINKS BETWEEN SOCIAL RHYTHMICITY AND DEPRESSIVE SYMPTOMS

Sabet, S. M. Dautovich, N. D. Dzierzewski, J. M. Virginia Commonwealth University, Richmond, VA.

Introduction: Disturbances in social circadian rhythms (e.g. regularity of meals and social interactions) and poor sleep are two potential factors that may contribute to the development of mood disorders. To date, no studies have investigated sleep behaviors (e.g. sleep health) and sleep thoughts (e.g. sleep self-efficacy) as potential links between social rhythmicity and mental health outcomes. The current study explored whether (1) higher social rhythmicity predicted fewer symptoms of depression and whether (2) sleep health and sleep self-efficacy act as mechanisms underlying this association. **Methods:** An archival analysis was performed using data from an online study, Investigating Sleep Across Normal Development (ISLAND Study). The sample consisted of 4,261 adults aged 18+. Measures of social rhythmicity (SRM-10), sleep self-efficacy (SES), sleep health (RU SATED), and depressive symptoms (PHQ-2) were utilized. Age and gender were included as covariates in mediation analyses.

Results: The overall model was significant, p < .0001 and 26.4% of the total variance was accounted for by social rhythmicity. Controlling for covariates, higher social rhythmicity was directly associated with fewer depressive symptoms (95% CI [.0326, .0420]). Additionally, both sleep health 95% CI [.0034, .0078] and sleep self-efficacy [.0119, .0169] significantly mediated the association between social rhythmicity and depressive symptoms.

Conclusion: Individuals who have higher levels of daily routine regularity experienced less depressive symptoms than those who are more irregular in their daily routines. Furthermore, individuals who had more regular lifestyle habits were more likely to engage in healthy sleep behaviors and thoughts. Thoughts and behaviors are common

factors that may have an impact on mental health given their daily reoccurrence. Also, as these factors are modifiable they could be targeted to potentially reduce depressive symptoms. Future research should continue to examine the link between social rhythmicity and sleep behaviors and thoughts on various health outcomes.

Support: This work was supported by the National Institute on Aging (K23AG049955, PI: Dzierzewski).

0186

SLEEP QUALITY AND EXPECTATIONS ABOUT SLEEP ARE ASSOCIATED WITH PERFORMANCE IN INDIVIDUAL SPORTS

Carmichael, K. E. O'Connor, P. University of Georgia, Athens, GA.

Introduction: Epidemiological studies on predictors of sport performance are rare. Some athletes report poor sleep the night prior to competition which may influence performance. The purpose of this study was to examine relationships between sleep and athletic performance in a sample of U.S. adults.

Methods: Self-identified athletes (n=635), recruited from Amazon's Mechanical Turk website, completed demographic, sleep, and athletic performance questions. Participants were at least 18 years old and competed in at least one athletic competition during the prior 6 months. Sleep quality and perceived performance were rated on 5-point Likert scales (e.g., 1=sleep quality/performance was very much worse than usual to 5=very much better than usual). Additional questions asked about plausible confounding variables, including the presence of bodily pain, expectations about whether sleep influences sport performance, and intensity of effort given during the competition compared to usual effort.

Results: The sample was 42% female and 71% Caucasian with 94% between the ages of 18 and 39 years; 358 (56.4%) participated in an individual sport and 277 (43.6%) were team sport athletes. The most common sports were running (35%), basketball (14%), and soccer (6%). For individual sport athletes, three variables predicted a significant amount of the variance in perceived performance (R² = 0.32, F(3, 354) = 56.30, p < .001): intensity of effort (β = 0.44, p < 0.001), sleep quality the night prior to competition (β = 0.14, p < 0.01), and expectations for performance change following a good night's sleep (β = 0.14, p < 0.01). Team sport athletes' perceived performance was predicted by the intensity of effort (β = 0.46, p < 0.001) and percentage of time spent in competition (β = 0.14, p = 0.01; R² = 0.26, F(2, 271) = 47.50, p < .001). **Conclusion:** Among individual sport athletes, and after adjusting for the intensity of their effort, prior night sleep quality and expectations regarding sleep and performance aided in predicting performance and performance aided in predicting performance size of the performance stress of the performance specifies and performance and performance aided in predicting performance specifies and performance and perform

ations regarding sleep and performance aided in predicting perceived performance. These relationships did not exist among team sport athletes, perhaps because of the greater error in estimating performance during team sport competition.

Support:

0187

THE EFFECTS OF NOCTURNAL AIRCRAFT NOISE ON SELF-REPORTED SLEEP

Casario, K. Howard, K. Smith, M. G. Rocha, S. White, M. Basner, M. University of Pennsylvania, Philadelphia, PA.

Introduction: Nocturnal traffic noise can fragment sleep through cortical arousals and induce self-reported sleep disturbance. Here we present data gathered around Atlanta International Airport in a pilot field study on the effects of aircraft noise on sleep.

Methods: N=34 subjects participated in a five night in-home study. Every night, subjects recorded noise inside their bedroom, and completed questionnaires the following morning containing items on sleep latency; number of awakenings; sleepiness (Stanford Sleepiness Scale); 11-point scales on sleep quality, tiredness, ease of falling asleep and calmness or restlessness of sleep; and a 5-point scale on sleep disturbance by noise. We analyzed the effect of both the average ($L_{AEq,sleep}$) and maximum ($L_{AS,max,sleep}$) aircraft noise level during a subject's sleep period for each questionnaire outcome in repeated measures multiple regression adjusted for the number of aircraft noise events during sleep, sex, age, and if the window was open or closed.

Results: A total of 165 sleep questionnaires (97.1% of expected) were completed. Self-reported awakenings increased by n=0.051 per decibel (dB) $L_{AS,max,sleep}$ (p<0.001). An increase in $L_{AS,max,sleep}$ was associated with a significant increase in tiredness (0.118/dB, p=0.005). There was a significant effect of sex on tiredness in the $L_{AEq,sleep}$ model, whereby men were less tired than women. There were no significant effects of $L_{AEq,sleep}$ on any questionnaire outcomes.

Conclusion: There was some evidence for adverse effects of aircraft noise on self-reported sleep outcomes. Effects were predominantly found for maximum rather than average noise exposure during the sleep period, stressing the importance of individual noise events for sleep. A larger-scale, adequately powered National Sleep Study will be conducted to better understand the observed effects.

Support: This research was funded by the U.S. Federal Aviation Administration Office of Environment and Energy through ASCENT, the FAA Center of Excellence for Alternative Jet Fuels and the Environment, project 017 through FAA Award Number 13-C-AJE-UPENN-011 under the supervision of Natalia Sizov. Any opinions, findings, conclusions or recommendations expressed in this material are those of the authors and do not necessarily reflect the views of the FAA.

0188

MOOD DISORDERS MODERATE THE RELATIONSHIP BETWEEN SLEEP QUALITY AND LEADERSHIP DEVELOPMENT FOR U.S. ARMY OFFICER CANDIDATES DURING ROTC ADVANCED CAMP

*Choynowski, J.*¹ *Pirner, M.*¹ *Mickelson, C.*¹ *Mantua, J.*¹ *Sowden, W. J.*² *Burke, T.*¹ *Capaldi, V. F.*¹ *McKeon, A. B.*¹ ¹Walter Reed Army Institute of Research (WRAIR), Silver Spring, MD, ²Tripler Army Medical Center, Honolulu, HI.

Introduction: U.S. Army Reserve Officer Training Corps (ROTC) Cadets are college students training to be Army Officers. During a month-long capstone course (Advanced Camp), Cadets are rated on their leadership ability. Little work has been done to determine predictors of leadership ability at Advanced Camp. This study examined the effect of poor sleep and mood disorders -- two prevalent factors among college students -- on leadership ability.

Methods: Metrics on leadership, sleep quality, anxiety, and depression, were assessed in 159 ROTC Cadets (22.06 ± 2.49 years; 23.90%female) at Days 1 (Baseline), 14 (Mid), and 29 (Post) of Advanced Camp. Leadership ratings were determined by ROTC Instructors over the course of Advanced Camp (1–5 score; higher score indicates poorer leadership). Predictors were the Pittsburgh Sleep Quality Index, Generalized Anxiety Disorder-7, and Patient Health Questionnaire-9. The relationships between the predictors and leadership scores were tested using linear regression. The interaction between mood disorders and sleep quality on leadership was tested using SPSS Process (Model 1).

Results: Poorer sleep quality at the Post time point (reflecting the prior 2 weeks of sleep) predicted poorer leadership (B=.05,p=.03),

while sleep quality from Baseline (B=.03,p=.14) and Mid (B=.01,p=.67) did not. Higher anxiety and depression scores from all time points predicted poorer leadership (p-values<.03). There was an interaction: higher anxiety and high depression predicted poorer leadership only in the context of poor sleep quality (not good or average sleep quality) [anxiety: R^2 =.04,F(1,159)=6.04 ,p=.02; interaction: R^2 =.03,F(1,155)=5.30,p=.02].

Conclusion: The current study identified a relationship between sleep quality and leadership ratings in ROTC cadets. This relationship was moderated by anxiety and depression. ROTC instructors should encourage ROTC Cadets to take advantage of sleep opportunities at Advanced Camp in order to maximize leadership potential.

Support: Support for this study came from the Military Operational Medicine Research Program (MOMRP) of the United States Army Medical Research and Development Command (USAMRDC). Disclaimer: The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the US Army or of the US

0189

SLEEP DURATION AND SUBJECTIVE RESILIENCE TO SLEEP LOSS PREDICT FUNCTIONAL IMPAIRMENT IN ELITE INFANTRYMEN DURING MILITARY TRAINING

Mickelson, C. A.¹ Mantua, J. R.¹ Burke, T. M.¹ Choynowski, J.¹ Bessey, A. F.² Naylor, J. A.³ Krizan, Z.⁴ Sowden, W. J.⁵ Capaldi, V. F.¹ McKeon, A. B.¹

¹Walter Reed Army Institute of Research (WRAIR), Silver Spring, MD, ²Clemson University, Clemson, SC, ³75th Ranger Regiment, Joint Base Lewis-McChord, WA, ⁴Iowa State University, Ames, IA, ⁵Tripler Army Medical Center, Honolulu, HI.

Introduction: Insufficient sleep during military operations is detrimental to cognition, physical performance, and general health outcomes. However, there is evidence of inter-individual differences in resilience to sleep loss. Therefore, some Soldiers may be more resilient to the effects of sleep loss than others. This study examined the relationship between sleep duration, resilience, and Soldier well-being during a deployment-readiness training event.

Methods: Seventy-six male Soldiers (aged 25.7±4.0y) from the 75th Ranger Regiment participated while undergoing a two-week training exercise. Surveys were administered at the completion of training and queried subjective measures of sleep duration during training, functional impairment (Walter Reed Functional Impairment Scale), and resilience to sleep loss (Iowa Resilience to Sleepiness Test; iREST). The independent relationships between sleep duration, resilience to sleep loss, and functional impairment were assessed using linear regressions. The interaction between sleep duration and resilience to sleep loss was assessed using SPSS Process (Model 1).

Results: Shorter sleep duration during training predicted higher functional impairment at the end of training (B=-.71, p=.001). Less resilience to sleep loss also predicted higher functional impairment (B=.07,p=.004). An interaction between sleep duration and resilience predicted Soldier impairment (R² change: .06; F(1,68)=.17,p=.03). Individuals with both shorter sleep duration during training and less resilience had the highest functional impairment. Those with more sleep, and those with high resilience and less sleep, both had lower functional impairment.

Conclusion: This study suggests the iREST can be used as a quick, subjective screening tool to indicate who may be most vulnerable to the effects of sleep loss. Identifying individual resilience to sleep loss may be useful in the military context for prescribing sleep

strategies before and during missions in order to enhance Soldier readiness and performance.

Support: Support for this study came from the Military Operational Medicine Research Program (MOMRP) of the United States Army Medical Research and Development Command (USAMRDC).

0190

IMPACT OF MENOPAUSE-RELATED SLEEP FRAGMENTATION ON DAYTIME SLEEPINESS AND NEUROBEHAVIORAL PERFORMANCE: RESULTS OF AN EXPERIMENTAL MODEL

Grant, L. K.^{1.2} Cohn, A.^{3,4} Abramson, M.⁴ Russell, J. A.⁴ Wiley, A.^{4,5} Coborn, J. E.^{4,5} Nathan, M. D.⁴ Scheer, F. A.^{1,2} Klerman, E. B.^{1,2,6} Kaiser, U. B.³ Rahman, S. A.^{1,2} Joffe, H.^{1,2,4,5} ¹Division of Sleep Medicine, Department of Medicine, Harvard Medical School, Boston, MA, ²Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, Boston, MA, ³Division of Endocrinology, Diabetes and Hypertension, Department of Medicine, Brigham and Women's Hospital, Boston, MA, ⁴Women's Hormones and Aging Research Program, Department of Psychiatry, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, ⁵Mary Horrigan Connors Center for Women's Health and Gender Biology, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, ⁶Department of Neurology, Massachusetts General Hospital, Boston, MA.

Introduction: Cognitive performance may be adversely affected during the menopause transition from hot flash-induced sleep fragmentation even without changes in sleep duration. We examined the effects of experimentally-induced sleep fragmentation without shortened sleep duration on daytime sleepiness and neurobehavioral performance in women in a high and low estradiol (E2) state.

Methods: Seven pre-menopausal women (29.4 \pm 3.8 years) participated in two 6-day inpatient studies repeated in a high-E2 (midto-late follicular phase) then low-E2 state (gonadotropin-releasing hormone agonist-induced E2 suppression - similar to levels during menopause) ~6 weeks apart. Sleep was uninterrupted on nights 1–2 [8-h time-in-bed (TIB)] and fragmented on nights 3–5 (9-h TIB) using an auditory stimulus delivered every 15 min that sustained wake for 2 minutes, producing 1-h total wake after sleep onset. Wakefulness was confirmed by event-markers during polysomnographically-recorded sleep episodes. Daytime subjective sleepiness (Karolinska Sleepiness Scale; KSS) and neurobehavioral performance (Psychomotor Vigilance Task; PVT) were assessed every 2–3 hours on study days 2–5. The effects of study day and E2 state on KSS scores and PVT measured reaction time (RT) and attentional failures (RT>500ms) were examined using linear mixed models.

Results: Participants reported feeling sleepier (+10%), had longer RTs (+22ms), and more attentional failures (+53%) after sleep fragmentation than after uninterrupted sleep (all p<0.001). While there was no main effect of E2 state, there was a differential effect of sleep fragmentation by E2 state on PVT, but not sleepiness, such that the increase in RT and attentional failures in response to sleep fragmentation was only observed in the high-E2 state (p<0.001).

Conclusion: Eight hours of total sleep time may not be sufficient to maintain subjective sleepiness and PVT performance levels when sleep is not consolidated. These findings have important implications for understanding the role of sleep and E2-modulated cognitive impairment during the menopause transition.

Support: This work was supported by the NIH: 5R01 AG053838-02 (HJ) and K24-HL105664 (EBK).

0191

SOCIAL JETLAG AND SLEEP HABITS ON WEEKENDS MODERATE THE RELATIONSHIP BETWEEN PERSONAL STANDARDS PERFECTIONISM AND ACADEMIC PERFORMANCE IN YOUNG ATHLETES

Caron, J. Roy, J. Godin, R. Gaudreault, P. Forest, G. Laboratoire du sommeil, Département de psychoéducation et de psychologie, Université du Québec en Outaouais, Gatineau, QC, CANADA.

Introduction: Research suggests that young athletes may present different levels of perfectionism, which affect sport and academic performances. Sleep is also a variable that can affects grades. The aim of the present study was to investigate the relationship between personal standards (PS) perfectionism, sleep and school performance in young athletes.

Methods: 27 young athletes (13-16y) wore an actigraph for a week and completed an adapted version of the Frost Multidimensional Perfectionism Scale at the beginning, middle and end of the school year. Sleep habits during weekdays (WD) and weekends (WE), and social jetlag (SJ) were extracted from the actigraphy. Mean performance of the two main school subjects were taken from the final report at the end of the school year (*M*grades). A linear regression was done between PS and *M*grades. Then, we used Hayes' PROCESS Macro V3.4 to examine the role of sleep as a moderator of the relation between PS and *M*grades.

Results: PS significantly predicts *M*grades (β =.59, *p*=.001; R_a^2 =.34, *p*=.001). The addition of the interactions terms, first between SJ and PS, then, between WE bedtimes and PS, and finally, between WE waketimes and PS, explained a significant increase in variance in *M*grades (ΔR^2 =.14, *F*(1,23)=31.81, *p*<.001; ΔR^2 =.17, *F*(1,23)=25.99, *p*<.001; ΔR^2 =.10, *F*(1,23)=12.43, *p*=.002, respectively). Therefore, when SJ is higher than 39min, WE bedtimes are after 10:21PM and WE waketimes are after 7:12AM, PS and *M*grades are significantly related.

Conclusion: These results show that when higher SJ and later WE bedtimes and waketimes are present, low PS are associated with low grades and high PS are associated with high grades. In contrast, when a small SJ, earlier WE bedtimes and waketimes are present, PS are not associated with grades. These results suggest that young athletes may be more vulnerable to the effect of sleep disturbances on grades depending on various factors such as perfectionism. **Support:** N/A

0192

SLEEP CHARACTERISTICS AND MOOD OF PROFESSIONAL ESPORTS ATHLETES: A MULTI-NATIONAL STUDY

Lee, S.¹ Bonnar, D.² Roane, B.³ Gradisar, M.² Jang, E.¹ Suh, S.¹ ¹Sungshin Women's University, SEOUL, KOREA, REPUBLIC OF, ²Flinders University, Adelaide, AUSTRALIA, ³University of North Texas, Fort Worth, TX.

Introduction: Esports is becoming increasingly professionalized, yet research on performance management is remarkably lacking. The present study aimed to investigate sleep and mood in professional esports athletes.

Methods: Participants were 17 professional esports athletes from South Korea (8), Australia (4) and the US (5) who played First Person Shooter games (mean age 20 ± 3.5 years, 100% male). All participants wore a wrist-activity monitor for 7–14 days, and completed subjective sleep and mood questionnaires.

Results: Based on data from the wrist-activity monitory, participants averaged 409 ± 37 minutes of total sleep time, and $87\pm1\%$ of sleep efficiency per night. All participants had significantly delayed sleep patterns (Average bed Time 3:41 am and wake Time 11:11 am). Participants had an average SOL of 26.15 minutes and prolonged wake after sleep onset of 51.91 (\pm 31.84) minutes. Korean players had significantly higher depression scores compared to the other groups (p=.006) and trained longer than the Australian or US teams (13.38 vs. 4.75 vs. 6.10 hours, respectively). Depression scores were strongly correlated with number of awakenings, wake after sleep onset and training time per day (ps<.05).

Conclusion: As the first exploratory study in the esports field, the study indicates that esports athletes show delayed sleep patterns and have prolonged wake after sleep onset. These sleep patterns may be associated with mood (depression) and training time. There may also be cultural differences that contribute to sleep disturbance in this population.

Support: Korean Society of Sleep Medicine

0193

SLEEP HEALTH ACROSS RELIGIONS: A CONSIDERATION OF BIDIRECTIONAL PROCESSES

Fergason, K. Rowatt, W. Scullin, M. K. Baylor University, Waco, TX.

Introduction: The psychology of religion literature indicates that religious engagement is beneficial to physical and mental health. Such effects might be mediated by sleep health, which causally affects mood, cognitive, and immune functioning. However, few studies have investigated whether religiosity is associated with better sleep, and no studies have considered the reverse causal direction: better sleep may impact religious behaviors or perceptions. Methods: We conducted a secondary data analysis of 1,501 participants in Wave 5 of the Baylor Religion Survey (BRS-5). Completed in Spring 2017, the BRS-5 used Address Based Sample methodology to derive a population-based sample. The survey included questions on religious affiliation, behaviors, and perceptions (e.g., certainty of Heaven). Additionally, participants rated their difficulty falling asleep and their average total sleep time. We investigated whether participants were meeting AASM/SRS consensus guidelines of 7-9 hours/night.

Results: Religious affiliation was associated with sleep duration, but not in the predicted direction. Atheists/Agnostics (73%) were significantly more likely to report meeting consensus sleep duration guidelines than religiously-affiliated individuals (65%), p<.05. For example, Atheists/Agnostics reported better sleep duration than Catholics (63%, p<.01) and Baptists (55%, p<.001). Atheists/Agnostics also reported less difficulty falling asleep at night than Catholics (p=.02) and Baptists (p<.001). The effects persisted when controlling for age and were particularly evident in members of African American congregations. Perceptions of getting into Heaven were significantly higher in participants who obtained better sleep duration, p<.05, but interestingly, such beliefs/perceptions were unrelated to difficulty falling asleep at night, suggesting that better sleep may lead to these perceptions rather than vice versa.

Conclusion: In contrast to predictions, religious affiliation was associated with significantly poorer sleep health. Poor sleep health has implications for physical and mental health, and seemingly also religious perceptions/beliefs. Future experimental work is required to disentangle the causal direction of sleep-religiosity associations.

Support: The Baylor Religion Survey was supported by the John Templeton Foundation.

0194

BEDTIME TECHNOLOGY USE AND NEW QUESTIONS FOR THE SLEEP HYGIENE INDEX

Mastin, D.¹ Abu-Halimeh, N.¹ Collins, B. T.¹ Critton, J.¹ Henderson, M.¹ Michelle, S.¹ Quattom, M.¹ Sanders, M.¹ Moore, B.¹ Peszka, J.² ¹University of Arkansas Little Rock, Little Rock, AR, ²Hendrix College, Conway, AR.

Introduction: We examined the relationship between bedtime active and passive social technology use (self and bedpartner) and daytime sleepiness/sleep. We generated questions to differentiate participants with and without bedpartners and updated passive personal, active bedpartner, and passive bedpartner social technology questions of the Sleep Hygiene Index.

Methods: 327 students (age: M=19.7 years, SD=3.78) recruited through psychology courses and campus newsletters received extra credit or chances to win \$25 gift cards. Participants completed demographic information, the Epworth Sleepiness Scale (ESS), the Pittsburgh Sleep Quality Index, questions regarding associated features of inadequate sleep hygiene, and the Sleep Hygiene Index. Five questions assessed active and passive social technology use, presence of a bedpartner, and awareness of bedpartner active and passive social technology use during sleep time.

Results: 61.8% and 62.7% of students reported frequently or always using active and passive bedtime social technology, respectively; and 23.5% and 29.1% reported noticing a partner's active or passive use. More frequent active technology use was significantly related to greater daytime sleepiness (ESS) (r(305)=.193, p<.05), sleep disturbances (PSQI-global: r(302)=.120, p<.05), and associated features of inadequate sleep hygiene (daytime sleepiness, worry about sleep, mood disturbance, avolition, and reduced cognition (r(306)=.212, p<.05)). Neither passive use nor passive or active partner use was significantly related to any sleep/sleepiness variables.

Conclusion: We continue to find students are frequent users of bedtime social technology which is related to daytime sleepiness, disrupted sleep, and related complaints. Passive and partner active/ passive bedtime technology use may not have a significant impact on daytime sleepiness. It is possible younger participants are not good judges of passive or partner technology use or this younger population is resilient to these disruptions. **Support:** none

0195

SOCIAL DETERMINANTS OF SLEEP: INSIDE RELATIONSHIPS WITH SIGNIFICANT OTHERS

Mousavi, Z. Tran, M. Kuhlman, K. R. University of California, Irvine, Irvine, CA.

Introduction: Social relationships impact health through different mechanisms. Sleep problems are prevalent among adults in the USA, negatively impacting all-cause mortality, and increasing the risk for chronic diseases such as depression, cardiovascular disease, and cancer. This study aimed to assess whether the quality of an individual's relationship with their significant other including support and strain, subjective relationship quality, joint decision making, marital risk, and conflict are associated with clinical, subjective, and objective measures of sleep.

Methods: Participants were selected from the Midlife in the United States (MIDUS) study if they had complete data on subjective sleep quality from the MIDUS II biomarker project and shared a bed with their partner (*n*=751, 49.5% female, M_{age} =53.4, SD_{age} =11.2, range= 34–83). Subjective sleep quality was measured using the PSQI. A subset of these participants (*n*=246, 50.8% female) also completed 7-days of daily diary and actigraphy.

Results: Sleep disturbances were pervasive: 44.1% (*n*=331) of participants reported clinically meaningful sleep disturbances (PSQI>=5). Among the smaller sample of participants with 7-day sleep data, sleep disturbances were even more prevalent (60.9%). There were significant bivariate associations between higher support and lower strain with better subjectively and objectively sleep outcomes. Better subjective relationship quality was also associated with better subjectively measured sleep outcomes. Higher marital risk and more disagreement with partner were associated with poorer sleep. When accounting for all marital relationship factors, participants with higher support and lower strain given to partner had a better long-term sleep quality, b=1.93, SE=.54, p<.001. Relationship quality was also associated with better daily sleep quality and daily reports of feeling rested, b = -.12, SE=.05, p=.023, and b=.11, SE=.048, p=.023, respectively. These relationships remained significant after accounting for age, current employment status, recent major health events, average daily caffeine, alcohol, exercise, and napping.

Conclusion: These findings support the importance of considering social determinants of sleep, suggesting that relationships with significant others may impact health through sleep quality. This highlights the importance of sleep as a transdiagnostic physiological mechanism that could be enhanced through improvements to relationships with significant others.

Support: N/A

0196

DIFFERENCES IN SLEEP DURATION AND ALERTNESS AMONG INTERNAL MEDICINE INTERNS COMPARING INTENSIVE CARE UNIT TO GENERAL MEDICINE ROTATIONS: A SECONDARY ANALYSIS OF THE ICOMPARE TRIAL

Cordoza, M.¹ Basner, M.¹ Asch, D. A.^{1,2} Shea, J. A.¹ Bellini, L. M.¹ Carlin, M.¹ Malone, S. K.¹ Desai, S. V.³ Sternberg, A. L.³ Tonascia, J.³ Volpp, K. G.^{1,2} Mott, C. G.⁴ Mollicone, D. J.⁴ Dinges, D. F.¹ ¹University of Pennsylvania, Philadelphia, PA, ²Corporal Michael J. Crescenz Veterans Affairs Medical Center, Philadelphia, PA, ³Johns Hopkins University, Baltimore, MD, ⁴Pulsar Informatics, Philadelphia, PA.

Introduction: Little is known about the impact of specific rotations on medical residents' sleep. The purpose of this analysis was to examine the difference in sleep duration and alertness among internal-medicine resident interns during intensive care unit (ICU) compared to general medicine (GM) rotations.

Methods: This is a secondary report of a randomized noninferiority trial of 63 United States internal-medicine residency programs. Programs were assigned to either standard duty-hour (80h workweek/16h shifts) or flexible (80h workweek/no shiftlength limit) policies. Interns were followed for 2 weeks during either a GM or ICU rotation. The primary outcome was sleep duration/24h (actigraphy). Secondary outcomes were sleepiness (Karolinska Sleepiness Scale [KSS]) and alertness (number of Brief Psychomotor Vigilance Test [PVT-B] lapses). Data were averaged across days (thirteen 24-hour periods). Linear mixed-effect models with random program intercept were used to determine the association between each outcome by rotation, controlling for age, sex, and policy followed.

Results: N=386 interns were included (mean age 27.9 \pm 2.1y, 194 (50.3%) males), with n=261 (67.6%) in GM, and n=125 (32.4%) in ICU. Average sleep duration was 7.00 \pm 0.08h and 6.84 \pm 0.10h for GM and ICU respectively (p=.09; 95%CI -0.02;0.33h). Percent of days with self-reports of excessive sleepiness were significantly more likely for ICU vs GM from 12am-6am (ICU: 20.2%; GM: 12.5%) and 6am-12pm (ICU: 20.5%; GM: 14.3%). GM had significantly more days with no excessive sleepiness (GM: 40.5%; ICU: 28.1%). Average KSS was 4.8 \pm 0.1 for both GM and ICU (p=.60; 95%CI -0.18;0.32). Average number of PVT-B lapses were 5.5 \pm 0.5 and 5.7 \pm 0.7 for GM and ICU respectively (p=.83; 95%CI -1.48;1.18 lapses). There were no significant differences in PVT-B response speed or false starts between rotations.

Conclusion: Interns in ICU may experience more excessive sleepiness compared to GM interns, especially in early morning hours. However, sleep duration and alertness were not significantly different between rotations.

Support: Funded by the National Heart, Lung, and Blood Institute and American Council for Graduate Medical Education

0197

CHRONOTYPE IS INFLUENCED BY BEHAVIORAL CHOICES AND CAN FLUCTUATE ACROSS THE SEMESTER IN STEM STUDENTS

Porro, A.¹ Luster, T.¹ Gao, C.¹ George, C.¹ Parizi-Robinson, M.¹ Quigley, D.¹ Zinke, P.¹ Scullin, M. K.¹

¹Baylor University, Waco, TX, ²Baylor University, Waco, TX, ³Baylor University, Waco, TX.

Introduction: A delay in endogenous biological rhythms is assumed to cause undergraduate students to be "night owls," but neurodevelopmental effects may only partially explain chronotype (circadian preference). Instead, perceived chronotype in students may result from poor sleep hygiene practices including bedtime social media use, afternoon caffeine consumption, and daytime napping. If so, then chronotype should be malleable in students to the extent that behavioral choices change.

Methods: We surveyed 1,120 undergraduate students who were enrolled in STEM courses across up to 3 time points during the semester. The survey assessed perceived chronotype (morning/ evening type), global sleep quality, and daily habits that impact alertness and sleep hygiene (e.g., social media usage and timing, caffeine consumption and timing, and napping behavior).

Results: Relative to Morning Types, students who perceived themselves as being Evening Types showed 23.1% greater bedtime social media usage (t=3.14, p=.002), 35.1% greater daytime napping duration (t=4.44, p<.001), and a 44 minute later average time of caffeine consumption (even though total caffeine consumption was reduced; t=2.30, p=.022). Evening Types also reported lower subjective health (t=3.55, p<.001), with 14.2% of the association between chronotype and subjective health being mediated by bedtime social media use (direct effect: b=0.050, p=.002; indirect effect: b=0.009, p<.05). Ninety-one students reported switching from being Evening Types at baseline to Morning Types at a later survey; those who switched to Morning Types used less social media and consumed less caffeine after 5pm and they showed significant improvements across the semester in sleep duration, sleep quality, and exam scores (ps<.05).

Conclusion: Perceived chronotype is related to social media and caffeine consumption behaviors and is modifiable. Students who

perceive themselves as night owls may find better health and academic success if they behave like morning larks. **Support:** National Science Foundation (DRL 1920730)

0198

PSYCHOMOTOR/COGNITIVE EFFECTS, PHARMACOKINETICS AND SAFETY OF V117957, A NOVEL, HIGHLY POTENT AND SELECTIVE PARTIAL AGONIST FOR NOCICEPTIN/ORPHANIN-FQ PEPTIDE (NOP) RECEPTORS, ADMINISTERED IN COMBINATION WITH ALCOHOL IN HEALTHY SUBJECTS

Zhou, M.¹ Harris, S.¹ Cipriano, A.¹ Kapil, R.¹ He, E.¹ Shet, M.¹ Apseloff, G² ¹Imbrium Therapeutics, Stamford, CT, ²Ohio Clinical Trials, Inc., Columbus, OH.

Introduction: V117957 is an investigational nociceptin/orphanin-FQ peptide (NOP) receptor partial agonist designed to treat insomnia by promoting sleep onset and maintenance with minimal residual next-day somnolence or psychomotor impairment. The satisfactory safety/tolerability profile of V117957 has been previously established in ~200 healthy subjects with maximum doses at 30mg following a single oral administration and 10mg once daily for 2 weeks. The present study was conducted to assess the safety/tolerability and pharmacokinetics (PK) of V117957 with co-administered alcohol.

Methods: A randomized, double-blind, double-dummy, placebocontrolled, balanced six-period crossover design was employed. Single doses (2mg, 6mg) of V117957 and placebo were administered orally to healthy subjects in the morning with and without alcohol (0.7g/ kg). Pharmacodynamic (PD) effects of V117957 were assessed, and safety/tolerability and PK interactions were also characterized. The primary PD endpoints (body sway, Digit Vigilance Test, and numeric working memory) were measured through 12 hours postdosing.

Results: Forty-eight subjects were enrolled and randomized; 46 completed. Compared with placebo, alcohol alone showed an impairment on psychomotor/cognitive performances through 2 hours postdose. V117957 alone showed a dose-dependent impairment. Compared with V117957 alone and alcohol alone, co-administration of alcohol and V117957 showed greater impairment until 8 hours postdose. No subject discontinued due to an adverse event (AE). No clinically meaningful treatment-emergent (TE) changes in clinical laboratory values, vital signs, SpO₂ measurements, or 12-lead ECG results were observed. The most common TEAE was somnolence. All plasma and urine PK parameters for V117957 and alcohol were comparable when V117957 or alcohol was administered alone or in combination.

Conclusion: Single oral doses of V117957, 2mg or 6mg, administered alone or in combination with alcohol in healthy subjects resulted in no notable PK interaction between V117957 and alcohol. A dose-effect relationship in the magnitude and duration of impairment was observed for most psychomotor/cognitive performance parameters. Greater effects of V117957 with alcohol were observed for most psychomotor/cognitive performance parameters. **Support:** Funded by Imbrium Therapeutics, a subsidiary of Purdue Pharma L.P.

0199

SLEEP AND RISK TAKING BEHAVIOR IN UNITED STATES ARMY SOLDIERS: A FOUR STUDY MEGA-ANALYSIS

Mantua, J.¹ Sowden, W. J.² Mickelson, C.¹ Choynowski, J. J.¹ Bessey, A. F.⁴ Burke, T. M.¹ Capaldi, V. F.¹ McKeon, A. B.¹ ¹Walter Reed Army Institute of Research, Silver Spring, MD, ²Tripler Army Medical Center, Honolulu, HI, ³Walter Reed Army Institute of Research, Silver Spring, MD, ⁴Clemson University, Clemson, NC. **Introduction:** In military service members, high risk-taking behavior (RTB; e.g., looking to start a fight, reckless driving) leads to injury, judicial reprimand, and removal from military service. Consequently, reducing RTB has become a priority of the United States (U.S.) Army, and identifying modifiable antecedents of RTB has become critical. In non-military populations, in-lab studies have shown sleep restriction/deprivation leads to risky decision-making. We assessed whether sleep duration/quality and RTB are related in U.S. Army soldiers in operationally-relevant settings.

Methods: Sleep and RTB questionnaire data were collected in 4 unique samples: U.S. Army soldiers from an Armored Brigade Combat Team, Reserve Officer Training Corps (ROTC) Cadets, Special Operations infantrymen, and elite mountain warfare instructors. We aggregated data to conduct a mega-analysis, which is a combined analysis of original raw data. We assessed whether RTB (assessed with an in-house measure of soldier-specific RTB) was correlated with nightly sleep hours (n=2175), Insomnia Severity Index (n=1076), and Pittsburgh Sleep Quality Index scores (n=503). Next, using a linear regression, we assessed whether sleep duration was a predictor of RTB while controlling for relevant demographic factors (age, gender, marital status, combat experience, years of education, rank, years of service; n=1198).

Results: Higher RTB was correlated with lower sleep duration (r=.23,p<.001), more insomnia symptoms (r=.29,p<.001), and poorer sleep quality (r=.20,p<.001). In the full model, lower age (B=.02,p=.03) and higher combat experience (B=.05,p=.006) predicted higher RTB. Sleep duration remained a significant (and the strongest) predictor of RTB (B=.18,p<.001).

Conclusion: Military leaders should work to build in more sleep opportunities and remove environmental sleep disruptors during training and deployment operations. Leaders should also monitor soldier behavior after military operations that require sleep loss in order to reduce RTB, and, consequently, increase the readiness of the force.

Support: This work was supported by the Military Operational Medicine Research Program (MOMRP). The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the US Army or of the US Department of Defense. This abstract has been approved for public release with unlimited distribution.

0200

TRAIT MINDFULNESS MODERATES THE WITHIN-PERSON RELATIONSHIP BETWEEN SLEEP AND PAIN IN NURSES

Mu, C. Lee, S.

University of South Florida, School of Aging Studies, Tampa, FL.

Introduction: Sleep and pain have a bidirectional relationship in clinical populations; however, we know less about the daily association in non-clinical but potentially vulnerable populations. Nurses are prone to poor sleep and pain symptoms due to work schedules and occupational stress. Implications from mindfulness-based interventions suggest that mindfulness may play a role in improving sleep and subsequently, reducing pain. The current study examined the within-person relationship between sleep and pain in nurses, and whether trait mindfulness moderates the relationship.

Methods: Participants were 60 nurses employed at a cancer hospital (M_{age} =35.35±11.83, 32% reported pain). For 14 consecutive days, ecological momentary assessment collected participants' sleep characteristics, pain symptoms (i.e., chest pain, headaches, upset stomach, and other pain), and pain interference with daily activities. Trait mindfulness was measured using the 15-item

Mindful Attention Awareness Scale. Multilevel modeling decomposed variances at the between- and within-person levels.

Results: At the between-person level, after controlling for sociodemographic covariates, more frequent insomnia symptoms (β =0.35) or lower sleep sufficiency (β =-0.19) were associated with more pain symptoms (ps<.05). Inversely, individuals with more pain symptoms reported lower sleep sufficiency (β =-0.41, p<.05). At the within-person level, after nights with poorer sleep quality (β =-0.08, p<.01), lower sleep sufficiency (β =-0.08, p<.01), or shorter sleep duration (β =-0.03, p<.05), participants reported more pain symptoms the following day. There were significant interactions of mindfulness with (a) sleep sufficiency predicting pain interference and (b) sleep duration predicting number of pain symptoms, such that the adverse associations of less sufficient and shorter sleep with more pain were more apparent in those with lower mindfulness.

Conclusion: Although there was a bidirectional association between sleep and pain at the between-person level, sleep was more likely to be the predictor of pain at the within-person level in oncology nurses. The significant moderation by mindfulness suggest that promoting mindfulness among nurses, prone to having poor sleep and pain, may reduce the adverse impact of poor sleep on daily pain.

Support: This work was supported, in part, by the University of South Florida College of Behavioral & Community Sciences Internal Grant Program (PI: Lee, Grant No. 0134930).

0201

MEDICAL MALPRACTICE PAYMENTS FOLLOWING MILD SLEEP LOSS AND THE DAYLIGHT SAVING TIME SHIFT

Lage, C. Gao, C. Scullin, M. K. Baylor University, Waco, TX.

Introduction: The national cost of the medical liability system exceeds \$10 billion/year, but not all medical errors result in a malpractice claim or payment. Malpractice claims are more likely if the medical error is perceived as severe, if the physician is perceived as lacking empathy, and if negative emotional reactions are triggered in the patient (due to individual or contextual factors). In recent experimental work, participants who were mildly sleep restricted showed an increased propensity to want to punish physicians for medical errors and compensate patients the maximum allowed. Building upon this laboratory work, we evaluated 30 years of medical malpractice claims to determine whether the judgment of final compensation increased after the Spring daylight saving time (DST) shift.

Methods: We obtained medical malpractice payment data on 373,643 United States cases from the National Practitioner Data Bank (NPDB). We contrasted inflation-adjusted payments across states that have DST shifts relative to non-DST control states (e.g., Arizona, Hawaii). We compared post-Spring DST payments to payments during the two weeks before/after the DST, and averaged payments for the remainder of the year.

Results: The total number of claims was unrelated to Spring DST, however, the size of malpractice payments significantly increased the week following the spring DST shift relative to non-DST control states and relative to the averaged payments for the remainder of the year. Spring DST was associated with an inflation-adjusted relative increase in malpractice payments by \$7,836 to \$61,809 per case (depending on comparison). Malpractice payments did not change in relation to the Fall DST shift.

Conclusion: Mild sleep restriction alters the cognitive and emotional regulation processes that underpin perceptions of medical error severity, willingness to punish, and judgments of appropriate compensation for medical errors. Support: N/A

0202

CHRONOTYPE MODERATES THE RELATIONSHIP BETWEEN PERSONALITY AND ACADEMIC PERFORMANCE IN YOUNG ATHLETES

Collins-Rancourt, M. A. Roy, J. Gaudreault, P. Godin, R. Forest, G. Université du Québec en Outaouais, Gatineau, QC, CANADA.

Introduction: Recent studies have shown that chronotype is associated with personality in adolescents. Other studies suggest that athletes are more conscientious, and that this personality trait is associated with higher academic performance among adolescents. The aim of the present study was to investigate the relationship between personality, chronotype and academic performance in young athletes. **Methods:** 27 young athletes and 13 young non-athletes (13-16y) completed the Horne & Östberg Morningness-Eveningness Questionnaire (MEQ) and the NEO-PI-3. Grades were taken from their final report at the end of the school year (*M*grades=year-mean performance on the two main school subjects). First, T-tests were conducted to compare both groups on personality traits. Then, a moderation analysis was conducted using Hayes' PROCESS Macro V3.4 to examine the moderation effect of the chronotype on the relationship between conscientiousness and *M*grades.

Results: Results show that young athletes are significantly more conscientious (M=4748±11,67) than non-athletes (M=38,31±9,59) (t(38)=2,46,p=.019). Conscientiousness and chronotype were entered in a regression analysis to predict Mgrades. The addition of the interaction term between chronotype and conscientiousness, to the regression analysis, explained a significant increase in variance in Mgrades (ΔR^2 =.34, F(1,23)=19.38, p<.001). Therefore, when the score is inferior to 57.75 at the MEQ (toward evening type), conscientiousness and Mgrades are significantly related (β =0.28, t(23)=2.07, p=0.05). **Conclusion:** These results show that the chronotype moderates the relationship between conscientiousness and academic performance in young athletes. Specifically, in more evening types, higher level of conscientiousness is associated with higher grades and lower level of conscientiousness is associated with lower grades. On the

other hand, for more morning types, the level of conscientiousness is not associated with grades. These results suggest that conscientiousness may be a protective factor against the impacts of adolescence sleep changes and disturbances, on academic performance. **Support:** -

0203

TO AND FROM THE NIGHT SHIFT: RISKY ON-THE-ROAD DRIVING IN NIGHT SHIFT WORKERS

Murugan, N. Sagong, C. Cuamatzi Castelan, A. S. Moss, K. Roth, T. Drake, C. L. Cheng, P. Henry Ford Health System, Detroit, MI.

Introduction: Drowsy driving is a common occupational hazard for night shift workers (NSWs). While sleep loss is commonly identified as the primary culprit of drowsy driving, another critical factor to consider is circadian phase. However, the role of circadian phase in driving safety has not been well characterized in NSWs. This study examined if dim light melatonin offset (DLMOff, i.e. the cessation of melatonin secretion) is also a relevant phase marker of susceptibility to four different subtypes of risky on-the-road driving behaviors.

Methods: On-the-road driving was monitored over 8 weeks via a mobile application that tracked risky driving behaviors using

accelerometer and GPS data from cell phones (N=15; 3052 total driving events recorded). Risky driving behaviors included: 1) frequency of hard-braking events, 2) frequency of aggressive-acceleration events, 3) duration of excessive-speeding, and 4) duration of phone-usage. At week 2, participants spent 24 hours in-lab where hourly saliva samples were collected and assayed for melatonin, and DLMOff was calculated. Phase angle of driving events relative to DLMOff was used as the predictor in nested mixed-effects regressions, with risky driving behaviors as the outcome variables.

Results: The most common occurrences of risky driving were phone-usage and hard-braking. On average, NSWs had 46.7% and 42.0% of driving events with at least one occurrence of phone-usage and hard-braking, respectively. Rates of aggressive-acceleration and speeding were 24.4% and 20.4%. Positive phase angles (i.e. driving after DLMOff) were associated with reduced rates of hard-braking and aggressive-acceleration, but not of phone-usage and excessive-speeding. Specifically, rates of hard-braking and aggressive-acceleration decreased by 4.5% (p<.01) and 3.4% (p=.05) every two hours following DLMOff, respectively.

Conclusion: The study suggests DLMOff appears to be an important variable for predicting accident risk in NSWs. If replicated, circadian phase should be considered in recommendations to increase occupational health and safety of NSWs.

Support: Support for this study was provided to PC by NHLBI (K23HL138166).

0204

THE IMPACT OF SLEEP ON WELL-BEING AND DIURNAL PERFORMANCE IN ELITE AUSTRALIAN FOOTBALL LEAGUE ATHLETES

Facer-Childs, E. R. Drummond, S. P. Rajaratnam, S. W. Monash University, Melbourne, AUSTRALIA.

Introduction: The ultimate goal in the sports world is achieving optimal health and continuous high-level performance through an adequate balance of training load and recovery e.g. rehab, nutrition, well being and sleep. Athletes often encounter situations that negatively impact their ability to sleep well, disrupt their biological rhythms and increase mental stress e.g. late competition times, travel and high training load. Therefore, there is a need to increase our understanding of how individual variability could be impacting recovery and performance in elite sports.

Methods: This study examined the relationships between individual sleep and circadian patterns, well-being and performance variables in Australian Football League (AFL) athletes. Actigraphy combined with daily sleep diaries were used to gather objective sleep data over a period of 14 days. Performance tests were conducted in the morning (between 07:00 - 08:00am) and afternoon (between 14:00 - 15:00) on days 3, 5, 7, 10, 12 and 14. Performance measures included a strength test (force plate jump), a skilled based accuracy test (goal kicking) and a reaction time test (psychomotor vigilance task).

Results: Preliminary results show that sleep and circadian parameters differed significantly between individuals and were correlated with measures of well-being and diurnal performance.

Conclusion: The Australian Football League (AFL) is one of the largest growing sports industries in Australia and New Zealand with annual revenues reaching a billion dollars. These findings add to the growing literature showing how sleep impacts performance in elite athletes and highlights the need to take sleep and time of day into account. This is of critical importance to the global sports industry, who are constantly seeking marginal gains. **Support:** n/a

0205

REMOTE COLLECTION OF DAILY LIFE INFORMATION FOR JAPANESE RESIDENTS

Hirai, N. Kubo, M. Sakurai, Y. Komatsuzaki, K. Tazawa, R. Tokyo Medical and Dental University, Tokyo, JAPAN.

Introduction: Japanese medical students spend their school days under a dense curriculum. It is often difficult to maintain their healthy lifestyle. After graduation, most of them participate in clinical training programs as residents. It is also difficult to maintain a normal life because they are engaged in different specialized departments every few months. Such an environment is considered prone to mental health problems. In fact, one in four residents are reported to be newly depressed two months after the start of clinical training (Maeno T, et al. 2008). These mental health issues are thought to be affected by changes in daily life, but it is difficult to know such changes. In order to investigate changes in their daily lives, we developed a data collection system related to mental health via the Internet.

Methods: The subjects were 22 medical students who graduated from our university in March 2017. They were asked to wear activity tracker wrist bands from December 2016. They were also asked to answer the questionnaire on a web site every week as much as possible, and the responses were collected via the Internet together with the activity data. The first eight months of the observation period, including four months before the start of clinical training and four month after the start of clinical training, are divided into four quarters every two months, and the averaged sleep time and responses to the questionnaire in each period were investigated.

Results: The average number of days that the sleep was effectively recorded during each two months was 28–48. The average number of responses to the questionnaire during each two months was 6.0–7.2. **Conclusion:** Residents in the initial clinical training period should be very busy, but the system we have developed seems to have worked well with them for the first four months after the start of clinical training. Whether this system would work as well a longer period is a further problem.

Support: This study is supported by KAKENHI 16K01753.

0206

SLEEP OPPORTUNITY AND DURATION ARE RELATED TO RISK INJURY IN ELITE ATHLETES

Shaw, L.¹ Cohen, R.² Altman, Y.³ Eyal, S.³ Baharav, A.^{3,2} ¹United States Olympic & Paralympic Committee, Colorado Springs, CO, ²Wingate Institute, Netanya, ISRAEL, ³HypnoCore, Petach Tiqva, ISRAEL.

Introduction: Sleep is essential to musculoskeletal recovery, acquisition of new skills and emotional regulation in athletes. Insufficient sleep is detrimental to performance. Recent publications indicate that sleep duration is related to risk for injury in young athletes. We aimed at analyzing the relation between sleep opportunity and duration and the likelihood of an injury among adult elite athletes.

Methods: We studied 7,237 nights recorded with the Sleeprate application by 71 adult elite athletes from diverse sports, during the period September 2018-October 2019. Night recordings included perceived and measured sleep parameters. In addition, athletes reported their previous day nap duration, injuries and illness status. Out of the total number of nights, 4,205 included reported injury status with no injury and no illness for the previous night. Nightly total time in bed (TIB), TIB including reported naps (TIB24hr) and measured total sleep time (TST) were examined.

Results: Average TIB was significantly shorter (508 ± 77 minutes, mean \pm STD) in healthy days preceding injuries than in healthy days preceding days with no injury (525 ± 70 minutes, p<.001). Similar results were found when comparing the TIB24hr (injury: 517 ± 83 minutes, no injury: 543 ± 76 minutes, p<.001) and TST (injury: 443 ± 72 minutes, no injury: 457 ± 69 minutes, p<.001).

Conclusion: Average sleep opportunities of the elite athletes in this study were in accordance with their age and workouts load. The time athletes allow themselves as an opportunity for sleep is inversely correlated to the chances of developing an injury. These findings corroborate published research regarding sleep duration and risk of injury in athletes, yet our findings are based on real life data of elite athletes, and demonstrate the importance of sleep as part of the elite or professional athlete's routine, suggesting that even as little as around 20 minutes of added sleep may be efficient in preventing injury. **Support:** N/A

0207

THE EFFECT OF NAPS ON INHIBITORY CONTROL AND SUSTAINED ATTENTION IN EARLY CHILDHOOD

Andre, C. J. Mendelevich, E. Santiago, A. Spencer, R. M. University of Massachusetts Amherst, Amherst, MA.

Introduction: Sleep in adults and school-age children has been shown to improve regulatory behaviors. Specifically, slow wave sleep (SWS) disruptions have been positively associated with decreased levels of sustained attention and inhibitory control in adults, while REM sleep has been associated with inhibitory control in typically developing children. However, it is unknown whether midday naps confer a similar benefit in preschool-aged children, particularly since REM sleep is often lacking in their naps. In this study, we used a Go/No-Go task to determine whether SWS during early childhood naps benefits sustained attention and inhibitory control. We also explored nap habituality as a factor given that habitual and non-habitual nappers have different sleep architecture in adults and children.

Methods: Preschool children (N=22, 38–69 months) completed a Go/No-Go task, after which they either napped with polysomnography (nap condition) or stayed awake (wake condition) for an equivalent amount of time (within subject; order counterbalanced; \sim 1 week apart). After their nap and wake sessions, they completed the Go/No-Go task again.

Results: When controlling for nap frequency, participant performance (accuracy) post-session did not differ across conditions. However, by examining only the habitual nappers (5–7 days/week, N=9), we found a moderate positive correlation between percent of sleep spent in SWS and post-nap accuracy (r=0.335, p=0.037). Interestingly, we did not see the same relationship with non-habitual nappers (0–4 days/week, N=13) and found a weak negative correlation with SWS (r=0.007, p=0.031).

Conclusion: The findings suggest that habitually napping children show a benefit of nap SWS on regulatory behaviors while non-habitual nappers do not. Such results have important translational significance for early education settings. **Support:** NIH R01 HL111695.

0208

THE EFFECT OF MASTICATION ON PSYCHOMOTOR VIGILANCE PERFORMANCE

Hansen, D. A.¹ Hudson, A. N.¹ Lawrence-Sidebottom, D.¹ Maislin, G.² Miquel, S.³

¹Washington State University Spokane, Spokane, WA, ²Biomedical Statistical Consulting, Wynnewood, PA, ³Mars Wrigley, Global Innovation Center, Chicago, IL.

Introduction: Sustained attention is important for optimal neurobehavioral performance, but many biological and environment factors (e.g., circadian rhythm, distraction) may cause sustained attention deficits. Mastication (chewing) has been suggested to provide a countermeasure to sustained attention deficits. To investigate this, we conducted a randomized, within-subjects, cross-over study of sustained attention with a mastication condition and a control condition. Methods: N=58 adults (ages 18-45; 38 females) completed a 5h in-laboratory study. Subjects entered the laboratory at 09:00. Following training on performance tasks, they had a 1h break before beginning the first of two test bouts at 11:00. Each test bout was 40min long and included subjective rating scales, the Sustained Attention to Response Task, and the Psychomotor Vigilance Test (PVT). Here we focus on PVT lapses of attention (RT > 500 ms), false starts, and mean reaction time (RT) as measures of sustained attention. In between test bouts, subjects had a 1h break inside the laboratory. During one of the two test bouts, subjects were instructed to chew a piece of gum at a steady, comfortable rate. Mastication activity was verified via electromyography (EMG). Half of the sample was assigned to the mastication condition during the first test bout, the other half during the second test bout. Results: Controlling for order of conditions, there were no significant differences between conditions for PVT lapses (F156=0.40, P=0.54) or false starts ($F_{1,56}$ =0.10, P=0.80). Mean RT was higher in the mastication condition by 8.9 ± 2.5 ms (F_{1.56} =12.68, P<0.001). Conclusion: Using this test paradigm, we were unable to detect any significant improvement in PVT performance, although mastication resulted in a very small increase in mean RT. However, subjects were not sleep-deprived, distracted, or otherwise perturbed. A follow-up study under conditions of sleep deprivation and/or with longer task duration may provide further insight into the countermeasure potential of mastication.

Support: Mars Wrigley Confectionery, U.S., LLC

0209

THE EFFECT OF A NAP ON EMOTIONAL REACTIVITY IN INDIVIDUALS WITH A CHRONIC MILD TRAUMATIC BRAIN INJURY

Kurdziel, L. B. Maier, E. Limone, N. Azzarto, E. Merrimack College, North Andover, MA.

Introduction: Mild Traumatic brain injuries (mTBIs) affect ~1-3 million people per year in the US alone. Mild TBIs can have lasting (>1 year) impacts on emotional reactivity and regulation. Sleep has also been shown to be significantly altered in individuals with a mTBI, even when tested over a year since the injury. Sleep quality is strongly linked with emotional stability and emotional memory. Therefore, one possible mediating factor between emotional reactivity and mTBIs is sleep. Reduced sleep quality following a mTBI may impair the emotional regulation that typically occurs across sleep. Thus, increasing total sleep time through a nap may help to alleviate some of the emotional symptoms. This study assessed whether individuals with a chronic mTBI showed differences in brain activity associated with emotional regulatory circuits, performance on an emotional reactivity task, and sleep physiology across a nap compared to controls. Methods: Participants were 53 young adults (mTBI nap group: n=9; control nap group: n=16; mTBI wake group: n=11; control wake group: n=17). Following a nap, or an equivalent bout of wake (both recorded with polysomnography), participants completed an emotional Go/No-Go task in which they were asked to respond when a particular emotional valence was presented (neutral, fearful, or happy), and withhold a response when a different valence was presented.

Results: There was a significant main effect of emotion on reaction time (F(2, 98)=26.55, p < 0.001). Participants were slowest to respond to the neutral images. There was also a significant three way interaction between emotion, group, and condition (F(2,98)=4.085, p = 0.02).

Conclusion: While these results are preliminary, they support that both napping and mTBIs may impact emotional reactivity. Further, napping may help alleviate some of the chronic emotional dysregulation associated with mTBIs.

Support: Zampell Family Faculty Fellow

0210

EVALUATING THE SENSITIVITY OF SLEEP MEASURES FOR MONITORING PILOT FATIGUE IN OPERATIONAL SETTINGS

van den Berg, M. J.¹ Wu, L. J.¹ Gander, P. H.¹ Santos-Fernandez, E.² Signal, L.¹

¹Massey University, Wellington, NEW ZEALAND, ²Queensland University of Technology, Brisbane, AUSTRALIA.

Introduction: Previously, combined data analyses of four pilot fatigue monitoring studies including 237 pilots flying long-haul and ultra-long range (ULR) flights found no association between pilots' actigraphic sleep in flight and psychomotor vigilance task (PVT) performance at top-of-descent (TOD; beginning of the landing phase of flight). The present study aimed to determine whether measures of in-flight sleep recorded with polysomnography (PSG) are more sensitive predictors of pilots' PVT performance near TOD than actigraphic measures.

Methods: Data were re-analysed from 41 Singapore Airlines A340-500 pilots (median age 47, range 29–58 years) monitored on a ULR trip between Singapore and Los Angeles (average flight duration outbound = 15.6 hrs; inbound = 17.2 hrs). In-flight sleep was recorded simultaneously with PSG (scored in 30-second epochs) and actigraphy (recorded in 30-second epochs and scored in conjunction with sleep diary information). PSG- and actigraphy-determined time awake were calculated as the duration between the end of the last epoch scored as sleep (PSG) or software-scored sleep interval (actigraphy) and the start time of the 10-minute PVT completed near TOD.

Results: Linear mixed modelling indicated that after controlling for flight sector and intra- and inter-individual variability, neither PSG-derived total in-flight sleep (F_(1, 44.4) = 0.006, p= 0.941) and time awake (F_(1, 34.3) = 0.431, p= 0.516), nor actigraphic total in-flight sleep (F_(1, 51.1) = 0.161, p= 0.69) and time awake (F_(1, 34.9) = 0.23, p= 0.634) were associated with PVT response speed at TOD.

Conclusion: In this context, actigraphy produced identical findings to polysomnography and remains a valid alternative for monitoring in-flight sleep of groups of pilots during ULR flights. Further research is needed to determine whether PVT performance is a discriminatory measure of fatigue-related impairment in pilots. **Support:** This analysis was supported by the Massey University College of Health Research Fund. The Singapore Airlines study was funded by the Singapore Civil Aviation Authority. We thank Dr Jarnail Singh for permission to use these data.

0211

SLEEP-PREPARATORY BEHAVIORS MODULATE SLEEP PHYSIOLOGY IN MICE

Tyan, J. L. Sotelo, M. I. Markunas, C. M. Morrow, J. G. Eban-Rothschild, A. University of Michigan, Ann Arbor, MI.

Introduction: Prior to sleep, animals perform various sleeppreparatory behaviors, yet little is known about their contribution

to sleep physiology. Sleep hygiene, which involves proper sleep preparation, is an effective treatment for insomnia in humans. The high prevalence of sleep disorders and drawbacks of available pharmacological interventions necessitate a better understanding of the ecological and evolutionary contexts of sleep. Nest-building is a sleep-preparatory behavior performed by many species. In this study, we aimed to determine whether the presence of a nest modulates sleep. Specifically, we investigated the effects of a nest on sleep/ wake architecture and activity in wake-promoting neurons in mice. Methods: To examine the role of nesting in sleep/wake architecture. we recorded EEG/EMG activity over 24 hrs (n=14, 7 males and 7 females) in the presence/absence of a nest. To determine whether the lack of a nest activates wake-promoting neurons, we utilized TRAP (targeted recombination in active populations) technology to label neurons activated by nest removal (n=4 mice per experimental group). Results: Mice without nests exhibited increased latencies to NREM and REM sleep and spent less time asleep during the inactive/light phase. Mice without nests also exhibited shorter episodes of NREM and REM sleep and more transitions between arousal states. Additionally, our preliminary results suggest that nest removal significantly increases population activity in multiple brain regions, including several cortical and thalamic regions.

Conclusion: Our findings support the hypothesis that the presence of a nest facilitates and consolidates sleep. The causal role of specific neuronal populations in sleep fragmentation in the absence of a nest remains to be elucidated. Taken together, our findings provide the first evidence for a role of sleep-preparatory behaviors in the facilitation and consolidation of sleep and could shape the development of novel treatments for sleep disorders.

Support: This work is supported by the Sloan Alfred P. Foundation, the Brain and Behavior Research Foundation, and the Eisenberg Translational Research Award.

0212

QUESTIONNAIRE ASSESSMENT OF INTRAINDIVIDUAL VARIABILITY IN SLEEP: INCONSISTENT SLEEP CAN BE WORSE THAN SHORT AVERAGE SLEEP

Gao, C. Luster, T. Bermudez, V. Porro, A. Scullin, M. K. Baylor University, Waco, TX.

Introduction: The consequences of short sleep are welldocumented, but recent evidence indicates that night-to-night consistency in sleep may be just as important. For the current work, we developed an intraindividual variability in sleep (IIV) questionnaire to make IIV measurement possible in single-time-point studies. We investigated whether self-reported IIV explained unique variance in sleep quality and health beyond average total sleep time (TST), focusing on a critical transition period (first semester of college) in which high variability was predicted.

Methods: First-semester college students (N=126, M_{age} =18.28, 75.40% females) completed an IIV questionnaire in which participants indicated their average sleep duration, then estimated how much their sleep duration deviated from their average duration for each day of a typical week. We quantified IIV as the mean day-to-day change in sleep. Participants also completed standard questionnaires on global sleep quality, social jetlag, daytime sleepiness, depression, and stress.

Results: Participants reported substantial IIV in their sleep durations (M=1.77 hours, SD=0.86) that was largely distinguishable from measures of social jetlag (r=.25) and average TST (r=-.18). Patterns of IIV differed across race/ethnicities: in white/Asian students, IIV was strongly associated with social jetlag (r=-.44) whereas in underrepresented minority students, IIV and social jetlag were separate constructs (r=-.03), suggesting that fluctuations in the latter group occur across all days of the week. Greater IIV was associated with significantly worse global sleep quality (r=.24, p=.01), stress (r=.20, p=.03), and depression, r=.20, p=.03). These associations were significant after adjusting for average TST, and only marginally reduced when controlling for social jetlag.

Conclusion: IIV in sleep/wake patterns can be captured using a questionnaire, and such measurement provides unique explanatory power to understanding sleep quality and mental health. Future research is needed to compare IIV questionnaire data to actigraphy data and to understand the underlying mechanisms by which inconsistent sleep detrimentally affects individuals.

Support: National Science Foundation (NSF 1920730)

0213

THE ASSOCIATION BETWEEN WORK-RELATED JOB DEMANDS AND DAYTIME IMPAIRMENT IN PROFESSIONAL FIREFIGHTERS

Soto, P. Dzierzewski, J. M. Dautovich, N. D. Ravyts, S. G. Perez, E. Donovan, E. K.

Virginia Commonwealth University, Richmond, VA.

Introduction: Sleep is an important predictor of daytime functioning and is impaired in first responders. The present study investigated whether job demands were associated with daytime impairment in professional firefighters. We hypothesized that the frequency of emergency calls would predict daytime impairment above and beyond years of service, sleep apnea risk, and sleep duration.

Methods: Participants were 267 (251 males; mean age=41.94) firefighters from the Richmond, Virginia Fire Department who completed pen-and-paper surveys in small groups assessing sleep duration (Pittsburgh Sleep Quality Index; PSQI), sleep apnea risk (STOP-BANG), job demands (number of emergency calls received per day), and sleep-related impairment (Patient Reported Outcomes Measurement Information System 8-item short-form; PROMIS). A three block hierarchical regression was used to assess the contribution of job demands to daytime impairment.

Results: The final model significantly predicted sleep-related daytime impairment, F(4,260)=11.51, p<.001, $R^2=.15$. Each block in the model accounted for significant change in variance, years of service and sleep apnea risk ($R^2=.05$), sleep duration ($\Delta R^2=.08$), number of calls ($\Delta R^2=.01$). Number of emergency calls significantly predicted daytime impairment ($\beta=.14$) above and beyond number of years of service ($\beta=-.24$), sleep duration ($\beta=-.29$), and sleep apnea risk ($\beta=.03$). **Conclusion:** With 2–18 emergency calls per 24-hour period, the results suggest that job-related demands are a unique contributor to daytime functioning in professional firefighters. As such, it will be important for interventions aimed at improving sleep in professional firefighters to incorporate information unique to the profession (i.e., job specific demands and intensity of work) as specific treatment factors.

Support: This work was supported by the National Institute on Aging (K23AG049955, PI: Dzierzewski).

0214

THE RELATIONSHIP BETWEEN WORK STRESS AND SLEEP QUALITY IN SHIFT WORKING NURSES: THE INTERMEDIATE EFFECT OF CIRCADIAN RHYTHM AMPLITUDE AND STABILITY

Wu, S. Wu, C. Wang, X. Fei, W. Ma, Y. Fu, Y.

Shanghai University of Traditional Chinese Medicine, Shanghai, CHINA.

Introduction: Research suggests that work stress may be an important factor in poor sleep in shift working nurses. However, evidence is limited about the underlying mechanism. Circadian rhythm might be effective. The purpose of this study was to explore the relationship between work stress and sleep quality in shift working nurses and the mediation effect of circadian rhythm amplitude and stability respectively. **Methods:** A total of 529 nurses working in shifts from seven hospitals were selected by convenient sampling in Shanghai, China. Participants were assessed with the Circadian Type Inventory (CTI), Chinese Nursing Stressor Scale (CNSS), Pittsburgh Sleep Quality Index (PSQI). The Amos24.0 software was applied to construct structural equation models with circadian rhythm amplitude (LV) and circadian rhythm stability (FR) as intermediate variables respectively.

Results: About 73.3% (388/529) of the nurses' PSQI scores were above 5, indicating poor sleep quality. The correlations between work stress, circadian rhythm amplitude, circadian rhythm amplitude and sleep quality were all significant (all P<0.01).Sleep quality total score was positively correlated with work stress(γ =0.348) and circadian rhythm amplitude (γ =0.330). Work stress was positively correlated with circadian rhythm amplitude (γ =0.297,P<0.01). Sleep quality total score was negatively correlated circadian rhythm amplitude (γ =-0.204,P<0.01);Work stress was negatively correlated with circadian rhythm amplitude (γ =-0.284,P<0.01).Work stress showed significant indirect effects on sleep quality through the mediating effect of circadian rhythm amplitude (β =0.188,P<0.01) and circadian rhythm amplitude (β =0.082,P<0.05).

Conclusion: It suggests that nurses working in shifts have poor sleep quality. Work stress and the amplitude and stability of circadian rhythm could affect sleep quality, and circadian rhythm amplitude and stability could mediate the relationship between work stress and sleep quality.

Support: The work was supported by Xinglin Young Talent Program of Shanghai University of Traditional Chinese Medicine(to Caiqin Wu) and The 12th Innovative activities of college students of Shanghai Traditional Chinese Medicine(2019SHUTCM094).

0215

SLEEP DURATION ASSOCIATED WITH DEGRADED PERFORMANCE IN MARKSMANSHIP AND WEAPON STABILITY DURING A 72-HOUR MISSION TRAINING EXERCISE

Brown, S. A.¹ Burke, T. M.²

¹Combat Capabilities Development Command Soldier Center, Natick, MA, ²Behavioral Biology Branch, Center for Military Psychiatry and Neuroscience, Walter Reed Army Institute of Research (WRAIR), Silver Spring, MD.

Introduction: Military operations require vigilance and performance under stressful conditions while functioning with little to no sleep. Previous links between marksmanship performance and sleep restrictions have been shown primarily in novice Soldier populations, with significant decrements in reaction time and decision making skills rather than lethality, mobility, or weapon handling metrics. Additionally, previous research has focused on isolated static marksmanship tasks in laboratory settings. The research presented here assessed the relationship between changes in Soldier sleep duration as measured by actigraphy, and marksmanship performance when conducting a 72-hour military field training exercise. Methods: Forty-six volunteers [42 males (age 24.5±4.2y; mean±SD)] across three platoons participated in this assessment of Soldier performance during a company-wide mission exercise. Sleep characteristics were collected via actigraphy throughout the mission. Marksmanship performance was assessed during pre-,

mid-, and post-mission, utilizing an operationally relevant course that integrated a static self-paced shooting task with a dynamic, fast-paced multiple target task. Marksmanship was assessed in areas of lethality (e.g. accuracy, shot dispersion), mobility (e.g. acquisition, engagement times), and weapon handling stability (e.g. movement of barrel during aiming).

Results: Analysis utilized a multivariate analysis of covariance (MANCOVA) with platoon as the independent variable and total sleep time over the previous 24-hours as the covariant, during the midsession assessment. The results indicated that prior sleep does significantly modulate marksmanship performance in areas of lethality and stability (ps<.05), but not mobility. Although all individuals received fewer hours of sleep than typical during the mission, those who received less sleep had greater inaccuracy and dispersion in their shot groups, and more barrel movement during weapon handling.

Conclusion: Marksmanship is a key military skill required of all Soldiers. This study has linked degradations in marksmanship lethality and stability performance in the field to naturally occurring sleep restrictions due to platoon-group variances during mission activities. Additionally, it is the first study to link weapon handling stability degradations to sleep loss. Further analysis will explore performance degradations associated with aspects of sleep quality, as well as individual platoon differences, such as leadership, qualifications, and resiliency.

Support: CCDC Soldier Center and Military Operational Medicine Research Program.

0216

IS THERE A DAILY RHYTHM IN ALCOHOL CRAVING AND DOES IT VARY BY CIRCADIAN TIMING?

Hisler, G. Pedersen, S. Clark, D. Rothenberger, S. Hasler, B. University of Pittsburgh, Pittsburgh, PA.

Introduction: People with later circadian timing tend to consume more alcohol, potentially due to altered rhythms in when and how much they crave alcohol throughout the day. However, whether circadian factors play a role in alcohol craving has received scant attention. Here, we investigated if the daily rhythm of alcohol craving varied by circadian timing in two independent studies of late adolescent and young adult drinkers.

Methods: In Study 1, 32 participants (18–22 years of age; 61% female; 69% White) completed momentary reports of alcohol craving five times a day for 14 days. Participants wore wrist actigraphs and completed two in-lab assessments of dim light melatonin onset (DLMO). Average actigraphically-assessed midpoint of sleep on weekends and average DLMO were used as indicators of circadian timing. In Study 2, 231 participants (21–35 years of age; 28% female; 71% White) completed momentary reports of alcohol craving six times a day for 10 days. Average midpoint of self-reported timein-bed on weekends was used to estimate circadian timing.

Results: Multilevel cosinor analysis revealed a 24-hour daily rhythm in alcohol craving which was moderated by circadian timing in both studies (p's<0.05). In both Study 1 and 2, people with later circadian timing had a later timed peak of craving. In Study 1, but not Study 2, later circadian timing predicted a blunted amplitude in craving.

Conclusion: Findings support a daily rhythm in craving that varies by individual differences in circadian timing. Because craving is an important predictor of future alcohol use, the findings implicate circadian factors as a useful area to advance alcohol research and potentially improve interventions.

Support: R21AA023209; R01DA044143; K01AA021135; ABMRF/The Foundation for Alcohol Research.

0217

ADOLESCENTS' EVENING PREFERENCE IS ASSOCIATED WITH SPECIFIC SLEEP HYGIENE BEHAVIOURS

*Gauthier-Gagne, G.*¹ *Dimakos, J.*² *Somerville, G.*³ *Boursier, J.*⁴ *Gruber, R.*²

¹Douglas Mental Health University Institute, Montreal, QC, CANADA, ²McGill University, Montreal, QC, CANADA, ³Riverside School Board, Montreal, QC, CANADA, ⁴Heritage Regional High School, St. Hubert, QC, CANADA.

Introduction: Circadian tendencies are associated with individual differences in preferred timing of behaviour. Sleep hygiene encompasses a variety of habits that are necessary for healthy. Given the later bedtimes of individuals with evening circadian preferences, more time is spent being awake in the evening and this could be associated with increased or longer engagement in poor sleep hygiene. Specific sleep hygiene practices that are common in adolescents with high evening preferences may therefore be a target to improve sleep. However, the relationship between specific sleep hygiene behaviours and circadian preferences in adolescents has not been examined. The objective of this study was to examine the associations between circadian preference and specific domains of sleep hygiene behavior. Methods: 127 adolescents (86 female) between 13 and 18 years old (M = 14.83, SD = 1.20) participated in the study. Circadian preferences were measured by the Morningness-Eveningness subscale of the School Sleep Habits Survey. Sleep hygiene was measured using the Adolescent Sleep Hygiene Scale (ASHS).

Results: Higher eveningness scores were significantly negatively associated with the ASHS physiological, behavioural arousal, cognitive emotional arousal, sleep environment, sleep stability, daytime sleep, substances use factors (r = -.20, p = <.05, r = -.27, p = <.01, r = -.32, p = <.01, r = -.18, p = <.05, r = -.41, p = <.01, r = -.28, p = <.01, r = 0.20, p = <.05 respectively) and with total sleep hygiene score (r = -.45, p = <.01).

Conclusion: Higher eveningness preferences in adolescents is significantly associated with poorer sleep hygiene in all domains with the exception of bedtime routine. Behavioural arousal, cognitive emotional, and sleep stability domains show the strongest inverse correlations. These findings could be used to inform the development of tailored sleep health interventions for adolescents with strong evening tendencies **Support:** Social Sciences and Humanities Research Council (SSHRC) support for Dr. Reut Gruber.

0218

BIOMATHEMATICAL MODELING PREDICTS FATIGUE RISK IN GENERAL SURGERY RESIDENTS

Schwartz, L. P.¹ Devine, J. K.¹ Hursh, S. R.¹ Mosher, E.² Schumacher, S.³ Boyle, L.³ Davis, J. E.³ Smith, M.² Fitzgibbons, S.³ ¹Institutes for Behavior Resources, Baltimore, MD, ²MedStar Institute for Innovation, Washington, DC, ³Georgetown University School of Medicine, Washington, DC.

Introduction: Fatigue and its effects on performance have long been a concern in medicine. Evidence exists that current duty-hour restrictions for resident trainees have a limited impact on physician wellbeing and patient safety, prompting renewed efforts to address this threat. In this study, sleep patterns of general-surgery residents were used to optimize a biomathematical model of performance for use as a tool for fatigue risk management with residents.

Methods: General surgery residents based at a multi-hospital, general surgery residency program were approached for

participation in this study. Enrolled residents wore actigraph devices for 8 weeks and completed subjective sleep assessments. Sleep data and shift schedules were then input into the Sleep, Activity, Fatigue and Task Effectiveness (SAFTE) Model to assess predicted cognitive performance. Performance was compared to an "effectiveness" level of 77 (equivalent to a blood-alcohol content of 0.05g/dL). Eight hours of sleep debt was considered "below reservoir criteria". Results: Sleep actigraphy data was collected from 22 general surgery residents. Modeling results showed that as shift lengths increased, effectiveness scores generally decreased, and the time spent below criterion (77) increased. Additionally, 11.13% of time on shift was below the effectiveness criterion and 42.7% of shifts included time spent below the reservoir criterion. Adjustments to the sleep prediction were made based on actual sleep, and performance predictions from actual sleep and the adjusted model were significantly correlated (p<.0001).

Conclusion: Despite adherence to national standards limiting work hours, current surgical resident sleep patterns and shift schedules create concerning levels of fatigue. This study illustrates how biomathematical fatigue models can predict resident physician sleep patterns and performance. Modeling represents a novel and important tool for medical educators seeking to create shift schedules that maintain physician preparedness and minimize fatigue risk. **Support:** N/A

0219

SUBJECTIVE SLEEP QUALITY MEASURED BEFORE, PREDICTS PSYCHOLOGICAL SAFETY AFTER SIMULATED COMBAT TRAINING. TESTING THE BIDIRECTIONAL LINK BETWEEN SLEEP AND SOCIAL PROCESSES IN THE MILITARY OPERATIONAL CONTEXT

Sowden, W.^{1,2} St. Pierre, M.³ Mickelson, C.² Mantua, J.²

¹Tripler Army Medical Center, Honolulu, HI, ²Walter Reed Army Institute of Research, Silver Spring, MD, ³Massachusetts General Hospital, Boston, MA.

Introduction: The research on sleep in the social-psychological domain is sparse. Gordon and colleagues (Gordon, Mendes, & Prather, 2017) proposed a bidirectional relationship between sleep and social processes. The current research tests this model in the military by examining the relationship between subjective sleep quality and an important social cognitive process in the contexts of military teams - psychological safety (i.e., an individual's perceptions of interpersonal threat in their work environment; Edmondson, 1999).

Methods: One hundred and twenty-eight U.S. Army tank crewmen were surveyed prior to (T1), and immediately after (T2), participating in a two-week simulated combat training exercise. Each survey included the seven-item Insomnia Severity Index (ISI; Bastien et al., 2001), which served as a measure of subjective sleep quality (SSQ), and Edmonson's seven-item Psychological Safety Questionnaire which measured team psychological safety (TPS). A cross-lagged panel model tested the effects of SSQ and TPS over the course of the training.

Results: Both SSQ and TPS were stable over the two time points, SSQ_{T1}: M=2.83, SD=.85, α =.83; SSQ_{T2}: M=2.63, SD=.83, α =.83; B=.387, SE=.08, β =.397, p<.001, and TPS_{T1}: M=3.7, SD=.72, α =.79; TPS_{T2}: M=3.67, SD=.75, α =.77; B=.619, SE=.07, β =.600, p<.001, respectively. Although SSQ and TPS were weakly related to one another at both time points, r_{T1} =.122, p=.086 and r_{T2} =.171, p = .028, only the cross-lagged path between SSQ_{T1} predicting TPS_{T2} was significant, B=.129, SE=.06, β =.147, p=.038. The cross-lagged path between TPS_{T1} predicting SSQ_{T2} was not significant, B=-.098, SE=.094, β =-.086, *p*=.296. Approximately 40% of the variance in CPS, R^2 =.4 as opposed to 17% in SSQ, R^2 =.17, was accounted for by the predictors in the model.

Conclusion: These results provide support for a directional (vs bidirectional) link between SSQ and TPS, insomuch that, in the context of military training, SSQ influences TPS, as opposed to the other way around. Elucidating the directionality of this relationship is not only important for advancing theory, but more importantly, it helps practitioners develop programs and policies that precisely address the right mechanism at the right time to maximize team effectiveness and wellbeing.

Support: This work was supported by the Military Operational Medicine Research Program (MOMRP).

0220

SLEEP DEPRIVATION INCREASES SELF-REPORTED BUT NOT BEHAVIORAL AVOIDANCE

*Campbell, R. L.*¹ *Lindsay, E.*¹ *Vance, A.*¹ *Nguyen, A.*¹ *Feldner, M.*² *Leen-Feldner, E.*¹

¹University of Arkansas, Fayetteville, AR, ²Canopy Growth Corporation, Smith Falls, ON, CANADA.

Introduction: A common form of emotion regulation is avoidance, in which attention toward negative stimuli results in avoiding (Elliot, 2006). Dysfunctional avoidance is linked to negative outcomes in various forms of psychopathology (Kashdan, Barrios, Forsyth, & Steger, 2006). Sleep challenges have been identified as a mechanism in numerous mental health disorders (Kryger, Roth, & Dement, 2017). These two mechanisms may be related. We hypothesized sleep deprived individuals would demonstrate more avoidance compared to baseline and a sleep as usual group as indexed by lower scores on a behavioral approach task (BAT) and more self-reported avoidance.

Methods: Fifty-two undergraduates (mean age: 18.87, white: 45, female: 35) without mental health disorders, sleep apnea symptoms, or use of medications that may impact sleep or wakefulness were recruited. Participants completed a Cognitive-Behavioral Avoidance Scale (CBAS modified) in which all questions were modified to elucidate present moment avoidance (ex. I would avoid attending social activities) and a BAT in which they were presented with a bedpan made to look and smell dirty. They were asked to complete seven hierarchical levels of engagement ex. (1) touching it with a tissue, (7) touching it with both hands then touching your face. The task ended when a participant declined to complete a step or they completed all seven. They were randomly assigned to 26 hours of sleep deprivation or sleep as usual. Students completed the CBAS modified and the BAT the next morning.

Results: After conducting a mixed ANOVA, there were no significant differences between or within groups in BAT steps completed. There were significant increases in self-reported behavioral social (p < .001) and nonsocial (p < .001), and cognitive nonsocial (p = .006), and social (p = .031) avoidance in the sleep deprivation group.

Conclusion: The study demonstrated a discrepancy between behavioral and self-report avoidance, suggesting a response bias after sleep loss. This investigation illuminates the effects of sleep loss on the transdiagnostic mechanism, avoidance. Note, there are no psychometric data for the modified CBAS. Future work should examine social forms of behavioral avoidance.

Support: This study was conducted using the University of Arkansas SONA system.

0221

LATE MEALS, SLEEP DURATION, AND SLEEP FRAGMENTATION: FINDINGS FROM THE AMERICAN TIME USE SURVEY

Iao, S.¹ Shedden, K.¹ Jansen, E. C.³ O'Brien, L. M.³ Chervin, R. D.¹ Knutson, K. L.⁴ Dunietz, G. L.¹

¹University of Michigan, Ann Arbor, MI, ²University of Michigan, Ann Arbor, MI, ³University of Michigan, ANN ARBOR, MI, ⁴Northwestern Medicine, Chicago, IL.

Introduction: Sleep hygiene recommendations discourage eating close to bedtime, though published data on the impact are not consistent. Associations between eating or drinking, within 1-hour prior to bedtime, sleep duration and sleep fragmentation were examined in a nationally-representative sample.

Methods: Data from the American Time Use Survey (ATUS), administered to a nationally representative sample of n=201,151 US residents aged ≥ 15 years were utilized. In an annual phone interview, ATUS participants were asked to record their activities during a 24-hour period (04:00am to 04:00am on the interview day) and were randomly selected to report weekdays or weekend activities. The present analysis included data from 2003–2018 and restricted to weekday respondents (n=124,242). Reporting of eating/drinking activities within 1-hour prior to bedtime was considered as a dichotomous variable (yes/no). Sleep fragmentation was defined as any awakening during the primary sleep episode (yes/no). Linear and logistic regression models, adjusted for age, sex, cohabitation, education and employment, were used to examine associations between eating/drinking and sleep duration or sleep fragmentation.

Results: In this ATUS sample, 56% of respondents were female and the mean age was 45 years. Mean sleep duration was 8.02 (0.007) hours, and 6% of survey participants ate/drank within 1-hour prior to bedtime. Overall, eating/drinking within 1-hour prior to bedtime was associated with longer sleep duration (p<0.01). Women and men who ate/drank within 1-hour prior to bedtime, in comparison to those who did not, had 35 minutes and 26 minutes longer sleep duration (p-value<0.0001) Eating/drinking activities within 1-hour prior to bedtime were associated with 1.8 higher odds of fragmented sleep (p<0.001).

Conclusion: In this large population-based survey, weekday eating or drinking within 1-hour prior to bedtime was associated with sleep fragmentation and longer sleep duration. Causal pathways would be difficult to discern, though sleep fragmentation could lead to compensatory increases in sleep duration. **Support:** None

0222

THE ASSOCIATION OF HABITUAL PHYSICAL ACTIVITY WITH 24-HOUR SLEEP OUTCOMES IN PRESCHOOLERS

St Laurent, C. W. Spencer, R.

University of Massachusetts Amherst, Amherst, MA.

Introduction: Sedentary behavior, physical activity (PA), and sleep are health behaviors that contribute significantly to overall and early childhood health. Although interactive relationships of these behaviors have been reported in adults and school-aged children, there is limited evidence that PA is associated with sleep using objective measures in younger children and findings have been mixed. The purpose of this study was to determine if objectively measured PA and sleep behavior outcomes are associated in preschoolers.

Methods: Participants (n=77, age: 4.34 ± 177 ;0.91 years; 55.8% female) were included in this cross-sectional study. Actiwatch

Spectrum monitors (wrist-worn, triaxial accelerometers) were worn 24-hours for 16-days to measure PA (total PA counts, sedentary time [ST], light PA, and moderate-to-vigorous PA [MVPA]) and sleep (24-hour, nighttime, and nap sleep duration, bedtime, wake after sleep onset [WASO], and sleep efficiency).

Results: Multiple linear regression models adjusted for age and wear time during wake periods indicated that greater MVPA was associated with less nighttime sleep duration (β =-3.48, p<0.001), less total 24-hour sleep duration (β =-3.38, p<0.001), and a later bedtime (β =0.07, p<0.001). Total PA counts were associated with less nighttime sleep duration (β =-0.0002, p=0.001), less total 24-hour sleep duration (β =-0.0002, p=0.001), less total 24-hour sleep duration (β =-0.0002, p=0.001), and a later bedtime (β =4.83, p=0.001). Greater ST was associated with greater total 24-hour sleep duration (β =1.92, p=0.006) and an earlier bedtime (β =-0.36, p=0.02). Percent time spent in light PA was not associated with any sleep outcomes and no PA variables were associated with nap sleep duration, WASO, or sleep efficiency.

Conclusion: As these findings are in contrast to previous studies reporting null or beneficial associations, further analyses are warranted to examine potential mediators/effect modifiers (e.g., sleep timing, gender, body mass index, and socioeconomic status) and temporal relationships between these movement behaviors in young children. **Support:** NIH R01 HL111695

0223

INTERACTIONS BETWEEN HOME, WORK, AND SLEEP AMONG FULL-TIME FIREFIGHTERS

Watkins, S. L. Shannon, M. A. Hurtado, D. Shea, S. A. Bowles, N. P. Oregon Health and Science University, Portland, OR.

Introduction: Firefighters endure large occupational burdens (e.g., heat, exposure to toxic fumes, witnessed trauma) and generally operate under conditions of chronic sleep deficiency due to long shifts plus disrupted sleep and circadian disruption due to emergency calls during the night. A typical shift for firefighters is 24-hours on/48-hours off, and firefighters are expected to use time-off to recover from any sleep debt. However, firefighters need to balance that recovery with social/family needs and home maintenance. We conducted focus groups and qualitative analysis of responses to understand how firefighters' sleep recoverability is affected by occupational burdens and home/family dynamics.

Methods: Focus groups were conducted via convenience sampling in Portland, Oregon, with full-time firefighters, battalion chiefs, and spouses of firefighters to assess current strategies and coping mechanisms used to manage occupational burdens, home/family obligations, and sleep loss based on their 24-hours on/48-hours off shift schedule. Grounded theory, using NVivo 12 plus, was used to code focus group transcripts to reveal reoccurring concepts that were further grouped into themes.

Results: Major themes that emerged among firefighters and spouses (n=48) centered on spousal resentment of firefighters, driven by understanding a firefighters' heroic occupation and need to recover from accumulated sleep loss and shift schedule, but also wanting a partner physically/emotionally present to share home/social responsibilities. While married firefighters discussed choosing family/home obligations over reducing sleep debt to maintain social/family relationships, single and divorced firefighters spoke of fewer conflicts impeding their ability to prioritize sleep at home.

Conclusion: This study improves our understanding of how work impacts home life in firefighters and can inform future strategies to address work-family conflict and sleep loss concerns, and highlights the importance of managing expectations of time-off to promote a healthier work-life balance.

Support: Oregon Healthy Workforce Center

0224

OPTOGENETIC MANIPULATION OF BASAL FOREBRAIN PARVALBUMIN NEURONS MODULATES VIGILANT ATTENTION AND RESCUES SLEEP DEPRIVATION INDUCED IMPAIRMENTS

Schiffino, F. L.^{1,2} McNally, J. M.^{1,2} Hassler, A. N.^{1,3} Brown, R. E.^{1,2} Strecker, R. E.^{1,2}

¹VA Boston Healthcare System, West Roxbury, MA, ²Harvard Medical School, West Roxbury, MA, ³Stonehill College, Easton, MA.

Introduction: Sleep disruption leads to attention impairments, excessive daytime sleepiness, and is a major contributor to accident rates and decreased workplace productivity. The basal forebrain (BF) region has long been associated with promoting cortical arousal and wakefulness. Recently, selective excitation of BF parvalbumin (PV) GABAergic neurons has been shown to produce high frequency cortical activation and brief periods of wakefulness. Here we test the hypothesis that BF PV neurons are involved in vigilant attention using bidirectional optogenetic manipulations in a signaled reaction time task.

Methods: Brief optogenetic excitation (ChR2) and inhibition (ArchT) of BF PV neurons was applied during a lever release version of the rodent psychomotor vigilance task (rPVT). Mice were trained to hold a lever down to initiate a trial and after a random delay, a 200ms cue light signaled the mouse to quickly release the lever within 1s to receive a sucrose pellet reward. The reaction time between cue light onset and lever release was the primary measure of attentional performance. Sleep deprivation (8h) produced by gentle handling was also investigated. Laser parameters: brief (1s) of continuous (non-pulsatile) laser stimulation was delivered beginning 500ms prior to cue light onset (5mW 473nm blue light for ChR2-mediated excitation; 10mW 530nm green light for ArchT-mediated inhibition).

Results: BF PV excitation led to faster reactions times (N=6, 14% faster, p<.001), interpreted as an enhancement of attention. Sleep deprivation slowed reaction times (20% slower, p<.01) and BF PV excitation rescued the sleep deprivation induced impairments. BF PV inhibition significantly slowed reaction times (25% slower, p<.02), an effect that resembled the effects of sleep deprivation.

Conclusion: This is the first demonstration of a role for BF PV neurons in attention and in the attention deficits produced by sleep deprivation.

Support: T32 HL007901, I01 BX002774, P01 HL095491, R01 MH039683, I01 BX004500, IK2 BX002130, Stonehill College SURE program, I01 BX001356

0225

DO ASSOCIATIONS BETWEEN DAILY STRESS AND SLEEP VARY BY WORK SHIFT? A WITHIN-PERSON ANALYSIS IN NURSES

Dietch, J. R.¹ Slavish, D. C.² Messman, B.² Wardle-Pinkston, S.³ Kelly, K.² Ruggero, C. J.² Taylor, D. J.³

¹VA Palo Alto Health Care System, Palo Alto, CA, ²University of North Texas, Denton, TX, ³University of Arizona, Tucson, AZ.

Introduction: Longitudinal studies have shown daily stress and sleep are bidirectionally associated. Nurses are particularly likely to experience sleep disturbances and high stress due to demanding work environments. Night shift work may be a unique stressor for nurses that exacerbates associations between stress and sleep. Using a within-person design, we examined the daily bidirectional

associations between stress and sleep and moderation by nightly work shift (day/off shift vs. night shift) in a large sample of nurses. **Methods:** Participants were 393 nurses (91% female; 77% white, mean age = 38.4 years) recruited from two hospitals. Participants completed 14 days of sleep diaries and actigraphy to assess total sleep time (TST) and sleep efficiency (SE). They simultaneously completed assessments of stress on the previous day (0 = "not at all" to 4 = "extremely") and daily work schedule (day/off shift vs. night shift [work between 9pm-6am]).

Results: Results indicated greater daily stress was associated with shorter diary TST (b = -9.49, p<.0001) and actigraphy TST (b = -4.48, p<.01), as well as lower diary SE (b = -0.56, p<.001). When examining reverse pathways of sleep predicting next day stress, both diary TST (b = -0.0004, p<.0001) and actigraphy TST (b = -0.0002, p = .03) predicted higher next-day stress. Lower diary SE predicted higher next-day stress (b = -0.005, p<.001). Only the association between daily stress and nightly diary SE was moderated by daily work shift: only when nurses worked a day or off shift did they have a negative association between daily stress and diary SE (b = -0.68, p<.0001).

Conclusion: Daily stress and sleep disturbances occurred in a bidirectional fashion for night- and day-shift working nurses. Most associations were similar regardless of daily type of work shift. Objective and subjective short TST and low subjective SE may contribute to a cycle of increased stress and are prime targets for a tailored sleep intervention in nurses. More research is needed to develop interventions to address the unique sleep health challenges faced by nurses. **Support:** NIAID R01AI128359-01

0226

SLEEP DURATION AND SYMPTOMS ASSOCIATED WITH RACE/ETHNICITY IN ELITE COLLEGIATE ATHLETES

Ramsey, T.¹ Athey, A.¹ Auerbach, A.¹ Turner, R.² Williams, N.³ Jean-Louis, G.³ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹ ¹University of Arizona, Tucson, AZ, ²George Washington University, Washington, DC, ³New York University, New York, NY.

Introduction: Previous studies have documented sleep disparities in the general population. Given the increased interest in sleep among athletes, and the degree to which demographics and schedules among athletes differ from the general population, this analysis aims to examine the relationship between race/ethnicity and sleep duration and symptoms among elite college athletes.

Methods: Data were obtained from N=189 Division-1 collegiate athletes across a wide range of sports played. Race/ethnicity was self-reported and categorized as Non-Hispanic White, Black/ African-American, Hispanic/Latino, Asian, and American Indian/ Alaskan Native. Outcomes of interest included self-reported typical sleep duration (in hours), CESD depression score, and frequency of sleep symptoms, assessed using items from the Sleep Disorders Symptom Check List (difficulty falling asleep, difficulty staying asleep, early morning awakenings, tiredness, sleepiness, loud snoring, choking/gasping, fragmentation, hypnogogic/pompic hallucinations, sleep paralysis, and nightmares). Sleep duration and depression were evaluated with linear regression, and symptoms were evaluated as ordinal. Covariates included age and sex.

Results: Compared to Non-Hispanic Whites, Blacks/African-Americans reported less sleep (B=-0.80, p<0.0005), more depression (B=2.85, p=0.046), more difficulty maintaining sleep (oOR=2.12, p=0.034), early morning awakenings (oOR=3.15, p=0.001), and sleepiness (oOR=2.11, p=0.048); Hispanic/Latinos reported more hypnogogic/pompic hallucinations (oOR=2.90, p=0.007), sleep paralysis (oOR=2.72, p=0.026), and nightmares (oOR=2.22, p=0.035);

Asians reported more depression (B=4.46, p=0.028), sleepiness (oOR=5.06, p=0.003), loud snoring (oOR=4.71, p=0.018), and sleep paralysis (oOR=3.57, p=0.031); and American Indians/ Alaskan Natives reported less sleep (B=-1.00, p=0.018).

Conclusion: Racial/ethnic differences in sleep duration and sleep symptoms were seen among athletes. Future studies will be needed to replicate and further explain these findings.

Support: The REST study was funded by an NCAA Innovations grant. Dr. Grandner is supported by R01MD011600

0227

THE COMPOUNDING IMPACT OF DAYTIME SLEEPINESS AND BRAIN INJURY ON SUSTAINED VIGILANCE

Dailey, N. S. Raikes, A. C. Wager, M. E. Grandner, M. A. Alkozei, A. Killgore, W. D. University of Arizona, Tucson, AZ.

Introduction: Daytime sleepiness is among the most frequent selfreported complaints by individuals who have sustained a mild traumatic brain injury (mTBI). Previous research demonstrates reduced vigilance and processing speed following mTBI. It has yet to be determined, however, if sustaining a mTBI alone, or the combination of daytime sleepiness and brain injury more greatly impacts cognitive function. The goal of this preliminary analysis was to determine the association between vigilance, daytime sleepiness, and mTBI.

Methods: A total of 137 adults ($M_{age} = 24.89\pm7.2$; 83 females) participated in the study, including 33 healthy controls (HCs) and 104 individuals with a documented mTBI within the preceding 12 months. Daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS), while daytime vigilance was measured using the Psychomotor Vigilance Task (PVT). To assess the effect of mTBI and daytime sleepiness on vigilance, we fit a Poisson regression to the number of lapses on the PVT, with group and ESS scores as predictors.

Results: ESS scores were significantly higher (p<.001) and there were significantly more PVT lapses (p=.03) in those with a recent mTBI, compared to HCs. For those with mTBI, the rate of lapses increased by 7.5% for every 1-point increase in ESS score (p<.001). Furthermore, when compared to HCs, the PVT lapse rate was 1.8x higher for individuals with a history of mTBI (p<0.001), after controlling for ESS scores. **Conclusion:** Daytime sleepiness was negatively associated with sustained vigilance for all participants. However, the magnitude of this association was roughly twice as high in individuals who had sustained a mTBI in the previous year. These findings provide evidence of a significant compounding effect of daytime sleepiness and brain injury on sustained vigilant attention. Clinical evaluation of mTBI would benefit from routine assessment of daytime sleepiness. **Support:** USAMRMC grant (W81XWH-12–0386).

0228

THE IMPACT OF WESTWARD TRAVEL ACROSS 9 TIME ZONES ON SLEEP BEHAVIORS OF FEMALE COLLEGIATE ATHLETES

Frisco, D. J. Goodrich, J. A. Byrnes, W. C. Holliday, M. Wright, K. P. University of Colorado, Boulder, CO.

Introduction: Jet lag can significantly impact an athlete's health and performance. However, the effect of ultra long-haul travel (> 12h flying time) westward across nine time zones on the sleep of female collegiate athletes is limited. We therefore studied the sleep behavior of NCAA Division I female volleyball players on an exhibition tour to China. **Methods:** For this observational study, eleven females were recruited from a NCAA Division I Volleyball team. During the Tour, sleep behavior was monitored using wrist actigraphy (Actiwatch Spectrum, Phillips) before (5 days) outbound travel (PRE-FLIGHT), during air travel to (1) & from (2) China (FLIGHT-DAY 1 & 2) and during the athletes' tour while in China (TOUR-DAY 1–8). Athletes were instructed to wear the actigraphs at all times, except during competition. Standard parameters were collected from the actigraph including sleep durations and sleep efficiency and expressed as mean \pm sd.

Results: Sleep duration and sleep efficiency were similar for PRE-FLIGHT days (~8.3 hrs \pm 1.5, 90.6 \pm 3.2%). Sleep duration and efficiency were significantly lower on FLIGHT-DAY 1 & 2 compared to TOUR-DAY and PRE-FLIGHT, but were not significantly different from each other (~5.2 \pm 2.4h, 80.5 \pm 8.8%). Sleep duration during TOUR-DAY 1–7 followed a quadratic relationship, peaking on TOUR-DAY 1 and reaching a nadir on TOUR-DAY 5, before increasing again through TOUR-DAY 7 (p<0.05). Sleep duration on TOUR-DAY 8 (~5.9 \pm 0.9 h) was significantly lower than PRE-FLIGHT and TOUR-DAY 1,2,6 & 7.

Conclusion: During travel female collegiate volleyball players showed sleep disturbance. Implementation of sleep interventions for jet lag are warranted for athletes traveling across multiple time zones. **Support:** PAC-12 Student-Athlete Health and Wellbeing Initiative, Grant #1554240

0229

EXPLORING THE RELATIONSHIPS BETWEEN SLEEP, STRESS, AND PERFORMANCE IN SIMULATION-BASED LEARNING

McGuire, K.¹ Lorenz, R.²

¹Southern Illinois University Edwardsville, Edwardsville, IL, ²State University of New York at Buffalo, Buffalo, NY.

Introduction: Sleep deprivation and stress may affect performance among students. Simulation-based learning (SBL) in undergraduate nursing programs provides the opportunity for students to practice critical decision-making without fear of patient harm; however, students still report experiencing stress during SBL. Current research is unclear on the effect of sleep deprivation combined with stress on performance in SBL. The purpose of this study was to explore the association between stress, functional outcomes of sleep, and performance in SBL.

Methods: Elements of the Theory of Stress, Appraisal, and Coping and the National League for Nursing Jeffries Nursing Education Simulation Framework guided this study. Baccalaureate nursing students consented to participate in a 1-hour SBL experience that included the collection of one hair and 4 saliva samples for cortisol concentration. Participants completed the Functional Outcomes of Sleep- Short Form and the Perceived Stress Questionnaire. An experienced faculty member evaluated student performance using the Creighton Competency Evaluation Inventory.

Results: Participants (N=35) were mainly female (n=32, 91.4%), white (n=29, 82.9%), with ages ranging between 18–22 years (n=32, 91.4%), and employed outside of nursing school (n=32, 91.4%). Other ethnicities represented include Asian and African American. Kendall's Tau correlations revealed a significant relationship between functional outcomes of sleep and perceived stress (r=-.281, p=.020). Although not significant, a small relationship was observed between functional outcomes of sleep and performance (r=.145, p=.236). No significant relationship between performance and perceived stress (r=-.099, p=.423) was identified.

Conclusion: This study suggests that daytime dysfunction related to sleep is related to perceived stress and performance in undergraduate

nursing students during participation in SBL. Due likely to small sample size, the relationship between sleep and performance was unable to achieve significance. These findings support the need for future research exploring the effects of sleep on stress and performance with larger more heterogeneous samples of students.

Support: This researcher would like to acknowledge and thank the following funding sources for their generous support of this work: Marion Bender Scholarship (Saint Louis University School of Nursing), Dissertation Award from Sigma Theta Tau International-Epsilon Eta Chapter, and Southern Illinois University Edwardsville School of Nursing Faculty Scholar Award.

0230

SLEEP AND INTERROGATION: DOES LOSING SLEEP IMPACT CRIMINAL HISTORY DISCLOSURE?

Krizan, Z. Miller, A. Meissner, C. Iowa State University, Ames, IA.

Introduction: Despite centuries of using sleep deprivation during interrogation, there is virtually no scientific evidence on how sleep shapes behavior in interrogation settings. Moreover, investigative interviews are often conducted at night, or with fatigued subjects. To evaluate the impact of sleeplessness on subjects' behavior during investigative interviews, an experimental study examined the impact of moderate sleep restriction on information disclosure and behavioral reactions during interviews about past illegal acts. Methods: Healthy participants (N=120) were recruited from the university community and randomly assigned to either maintain or curb their sleep (up to 4 hours a night) across two days. Back in the laboratory individuals privately indicated whether they committed various illegal acts. Participants were interviewed while video-recorded about the most severe act they acknowledged. After the initial disclosure, participants listened to a 'model' statement, an unrelated example of a person's detailed event account designed to encourage additional disclosure, after which they again provided information about their offense. Key variables were the severity of the illegal behavior reported and the amount of information provided before and after the model statement (blindly coded from transcripts for quantity and quality).

Results: Sleep-restricted participants slept on average 4.5 hours less (confirmed via actigraphy), reported no differences in perceived treatment by the interviewer, and tended to report less severe offenses. Critically, sleep-restricted participants provided almost 20% less information during their initial disclosure (d = .53, p =.01). After the model statement, however, disclosure was generally higher and similar across conditions (d = .15, p = .35). Sleep-restricted individuals also reported less overall motivation to recall information (d=.27, p = .01).

Conclusion: Results suggest that even moderate sleep loss can inhibit criminal disclosure during interviews, and that reduced motivation could play a role. Also, the use of the model statement could compensate for this effect.

Support: N/A

0231 SLEEP BEHAVIORS OF FEMALE COLLEGIATE ATHLETES

Frisco, D. J.¹ Goodrich, J. A.¹ Holliday, M.¹ Kroeker, K. A.¹ Whiting, C.¹ Byrnes, W. C.¹ Wright, K. P.¹

¹University of Colorado, Boulder, CO, ²University of Colorado, Boulder, CO.

Introduction: Sleep is critical to cognitive and physiologic function. It is likely being a female collegiate student athlete places unique demands upon sleep behavior. Therefore, we aimed to study the

sleep behavior of female collegiate athletes versus a female collegiate control group.

Methods: Full time female students from the University of Colorado Boulder (Altitude = 1,624 m) were recruited from NCAA Division I athletic teams: Cross Country (XC, n=10), Lacrosse (LAX, n =17), Soccer (SOC, n=15), Golf (GOLF, n=6), Tennis (TENN, n=9). 31 female full-time students were recruited as Controls (CONT). Sleep was monitored with wrist actigraphy (Spectrum Actiwatch, Phillips) for a minimum of one week. Subjects were instructed to wear actigraphs at all times except during competition. Outcome variables included nightly sleep duration, total 24h sleep duration, sleep efficiency, sleep midpoint, social jet lag (SJL) and nap duration/frequency.

Results: On weekdays, XC, LAX and SOC had greater nightly sleep durations ~8.5h compared to TENN, GOLF and CONT (p<0.05). Relative to other groups, XC had the earliest sleep midpoint (3:34 AM ± 1:20 vs. 3:53 AM ± 1:15) while SOC and LAX had the lowest sleep efficiency (~87.3 ± 3.2% vs. ~89.6 ± 3.4%) (all p<0.05). There was significant SJL among CONT, LAX and SOC (difference of sleep midpoints on weekdays and weekends; p<0.05). While nightly sleep duration varied significantly between different groups, there were no significant differences in total daily sleep duration when naps are included. A greater percentage of CONT (87 %) napped compared to athletes (64.2 %) (p<0.05); napping duration/frequency of naps per week were not different between groups.

Conclusion: Variations in sleep behavior exists between collegiate student athletes based upon varsity sport with some sports being similar to controls and others being significantly different. Additional research is needed to determine the significance of these findings to academic and athletic performance.

Support: PAC-12 Student-Athlete Health and Wellbeing Initiative, Grant #1554240

0232

IMPACT OF MENTAL HEALTH ON 10-YEAR TRENDS IN HABITUAL SLEEP DURATION

Khader, W. S.¹ Tubbs, A.¹ Fernandez, F.¹ Jean-Louis, G.² Seixas, A. A.² Williams, N. J.² Chakravorty, S.³ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹ ¹University of Arizona, Tucson, AZ, ²New York University School of Medicine, New York City, NY, ³University of

Pennsylvania, Philadelphia, PA.

Introduction: Public health efforts aimed at reducing the decline in habitual sleep duration have not been successful. It is possible that this decline is differentially experienced relative to individuals' mental health status. This would further support the need to focus on mental health as a strategy for improving sleep in the general population.

Methods: We examined 10 years of the National Health Interview Survey data (N=305,555). During all years, habitual sleep duration, age, sex, race/ethnicity, and height and weight (used to compute body mass index) were recorded in the same way. In addition, depressed mood in the past 30 days was evaluated (coded as none, mild, moderate, or severe). Weighted regression analyses examined sleep duration as an outcome, year and depressed mood as predictors, and sociodemographics as covariates. A year-by-depressed mood interaction was computed, and analyses were stratified by group.

Results: There was a significant year-by-depression interaction on linear change in sleep duration over the 10 year period (p=0.0001). Analyses were then stratified by depressed mood. In adjusted analyses, individuals with no depressed mood lost an average of 0.68 minutes of sleep per year (95%CI -0.82,-0.55; p<0.0001). Among those with mild depression, this was 7% higher, at 0.73 minutes (95%CI -1.13,-0.33; p<0.0001). Among those with moderate depressed mood,

this was 154% higher, at 1.73 minutes lost per year (95%CI -2.31,-1.16; p<0.0001). Among those with severe depressed mood, this was 351% higher, at 3.07 minutes per year (95%CI -4.22,-1.92; p<0.0001). **Conclusion:** The 10-year linear decline in habitual sleep duration seems to depend on mental health status. Individuals with better mental health lose less sleep over time, relative to those with worse mental health. This highlights the importance of mental health as a possible avenue for improving sleep health in the population. **Support:** Dr. Grandner is supported by R01MD011600

0233

PAIN, FATIGUE, AND ALTERED REACTIVITY TO A REPEATED PHYSIOLOGICAL STRESSOR IN INSOMNIA PATIENTS: AN EXPLANATORY-DRIVEN ANALYSIS

Goldstein, M. R.^{1,2} Devine, J. K.³ Dang, R.¹ Chatterton, B.¹ Scott-Sutherland, J.¹ Yang, H.^{1,2} Mullington, J. M.^{1,2} Haack, M.^{1,2} ¹Department of Neurology, Beth Israel Deaconess Medical Center, Boston, MA, ²Harvard Medical School, Boston, MA, ³Department of Operational Fatigue and Performance, Institutes for Behavioral Resources, Baltimore, MD.

Introduction: Fatigue and pain are prominent features of functional impairment in insomnia. This study aimed to better understand behavioral and physiological mechanisms of these complex relationships. Methods: 22 participants with insomnia disorder (DSM-5 criteria, 18 female, age 18-49yrs) and 22 good-sleeper controls (19 female, age 18-47yrs) completed two-weeks sleep logs and actigraphy recordings prior to coming to the laboratory for overnight polysomnography and subsequent daytime testing that included questionnaires, three trials of cold pressor test (CPT), and pain testing with blood draws collected throughout. Insomnia diagnosis was determined by a board-certified sleep specialist, and exclusion criteria included psychiatric history within past 6 months, other sleep disorder, significant medical conditions, and any medications within past two weeks with significant effects on inflammation, autonomic function, or other psychotropic effects. For CPT, participants were instructed to immerse hand in ice cold water for at least one minute and rate pain intensity throughout the immersion and 3-minute recovery. Data were analyzed with linear mixed models.

Results: Per inclusion criteria, PSQI scores were differed between groups (insomnia: 10.2 ± 2.7 , range 7–16; control: 1.9 ± 1.3 , range 0–5; p<.001). Insomnia consistently reported higher daily fatigue ratings compared to controls (p<.001), as well as higher spontaneous pain globally and across several specific domains (p's: .007-.03). In response to CPT, groups did not differ in their initial tolerance (i.e. immersion duration, p=.41) or intensity ratings during immersion (p=.88), however insomnia showed blunted recovery in intensity ratings (p<.01). Control participants then showed an ability to habituate to repeated CPT by increasing immersion duration, whereas insomnia slightly decreased in tolerance across trials (Group effect: p<.05).

Conclusion: These data indicate that habituation to and acute recovery from pain is deteriorated in chronic insomnia, which may be a key contributor to maintained pathophysiology over time and mechanism to target with comprehensive treatment.

Support: Merck Inc. MISP# 51971 (investigator-initiated), NIH/ National Center for Research Resources UL1-RR02758 and M01-RR01032 to the Harvard Clinical and Translational Science Center.

0234

SLEEP TIMING AND DURATION PREDICT LEVELS OF REPETITIVE NEGATIVE THINKING THE FOLLOWING DAY

Stewart, E. Acenowr, C. Coles, M. Binghamton University, Binghamton, NY. **Introduction:** Several forms of psychopathology characterized by repetitive negative thinking (RNT) are also associated with problems in sleep timing and sleep duration (Morin & Ware, 1996). These relations have been documented in cross-sectional studies but only a few studies have investigated this relation using a prospective design. This study aimed to: (1) replicate cross-sectional findings linking sleep duration and sleep timing to RNT and (2) use prospective longitudinal methods to extend previous research regarding this relation.

Methods: Participants (N = 127) were undergraduates who completed daily measures of sleep, mood, and RNT for 18 days. Participants mean age was 19.31(SD = 1.41) and 49% were male, and 60% were Caucasian. Measures included the Perseverative Thoughts Questionnaire, the Sleep-50, and a Daily Monitoring Questionnaire (DMQ) comprised of items from the Pittsburgh Sleep Quality Index. Results: Insomnia severity and circadian disruption severity was correlated with RNT, and these relations remained significant after statistically controlling for the influence of negative affect (Insomnia: r(123)=.22, p=.01; Circadian: r(123)=.21, p=.02). When looking longitudinally within person Hierarchical Linear Modeling (HLM) revealed later bedtimes (t(125) = 2.01, p = .05) and shorter sleep durations (t(125) = -3.17, p = .002) were predictors of heightened RNT the next day, even after statistically controlling for negative affect (RNT_{ii}= π_{0i} + π_{1i} (RNT_lag) + π_{2i} (bedtime_lag/hours slept_lag) + π_1 (mood_lag) + e_{ij}). RNT did not predict sleep variables when running the reverse of these models, yet negative affect emerged as a significant predictor of sleep timing (t(125) = 2.41,p = .02) and sleep duration (t(125) = -2.44, p=.02), indicating that mood, not RNT, may influence bedtimes and hours slept.

Conclusion: Results indicate that bedtime and sleep duration may be contributors to RNT, and that sleep disruptions may precede the onset of RNT. If future studies replicate the current study's findings, then sleep variables may serve as an important area of intervention and prevention of excessive RNT. **Support:** N/A

0235

SLEEP DURATION AND TIMING ASSOCIATED WITH EATING BEHAVIORS: DATA FROM NHANES 2015–2016

Bombarda, A.¹ St-Onge, M.² Seixas, A.³ Williams, N.³ Jean-Louis, G.³ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹ ¹University of Arizona, Tucson, AZ, ²Columbia University, New York, NY, ³New York University, New York, NY.

Introduction: Previous studies have shown that, in the laboratory, sleep deprivation leads to unhealthy eating patterns. In real-world samples, lack of sleep is associated with obesity. Few real-world studies of sleep and food intake patterns exist, especially from nationally-representative samples.

Methods: Data from the 2015–2016 National Health and Nutrition Examination Survey (NHANES) were used. NHANES is a national-representative survey collected by the CDC. N=6,291 participants provided data about dietary behaviors and sleep timing. Dietary behaviors included the number of meals not made at home in the past 7 days (NOTHOME), number of fast food/pizza meals in the past 7 days (FASTFOOD), number of pre-made meals in the past 30 days (PREMADE), and number of frozen meals in the past 30 days (FROZEN). Linear regression models examined these as outcomes and predictors including bedtime (minutes), waketime (minutes), sleep duration (hours), and daytime tiredness/fatigue (never, rarely, sometimes, often). Covariates included age, sex, education, income/poverty ratio, race/ethnicity, and body mass index.

Results: Number of meals not made at home (NOTHOME) was associated with a later bedtime (B=2.25, p=0.01) and shorter sleep duration (B=-0.12, p=0.01). FASTFOOD was associated with shorter sleep (B=-0.13,p=0.003) and tiredness/sleepiness sometimes (B=0.77, p=0.007) and often (B=0.55, p=0.03). FROZEN meals were associated with a later waketime (B=3.31, p=0.003) and tiredness/sleepiness sometime (B=1.20, p=0.025) and often (B=1.60, p=0.04). A sleep duration by bedtime interaction was not significant for any outcomes. In models that included overall levels of anxiety, these relationships were maintained.

Conclusion: This is one of the largest studies to show that habitual sleep patterns are associated with real-world food choices. In particular, shorter sleep duration and tiredness/sleepiness are associated with more ready-made and fast food meals. It is possible that lack of sleep leads to worse food choices, or that stress leads to both lack of sleep and easier food options. Given the often poor nutritive value of foods consumed outside the home and pre-prepared foods, these associations may in part explain the influence of sleep on cardiometabolic risk factors. **Support:** Dr. Grandner is supported by R01MD011600

0236

TEAM-BASED ATHLETES SLEEP LESS THAN INDIVIDUAL ATHLETES, BUT DO NOT REPORT MORE INSOMNIA OR FATIGUE

Clay, M. A.¹ Athey, A.¹ Charest, J.² Auerbach, A.¹ Turner, R. W.³ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹ ¹University of Arizona, Tucson, AZ, ²Centre for Sleep & Human Performance, Calgary, AB, CANADA, ³George Washington University, Washington, DC.

Introduction: Collegiate student-athletes face challenges balancing academics and athletics, and getting an adequate amount of sleep is one factor that can assist in sustaining an elite level of play. Teambased sports may present with systematically different sets of demands. **Methods:** Data were obtained at the start of the academic semester from N=189 NCAA Division-1 athletes from a wide range of sports. The sample was 46% female. Individuals were classified as playing in a team sport (e.g., football, basketball, baseball, softball, volleyball) or an individual sport (e.g., swimming, track, golf). Sleeprelated outcomes included self-reported sleep duration and sleep latency, frequency of sleeping pill use (Never, Rarely, Sometimes, Often), Insomnia Severity Index score, and Fatigue Severity Scale score. Regression analyses were adjusted for age and sex.

Results: In adjusted analyses, team-based athletes reported 22.4 minutes less sleep than individual athletes (95%CI -42.8,-1.9; p<0.05). They also reported 5.6 less minutes of sleep latency (95%CI -10.8,-0.3; p<0.05). More frequent sleeping pill use was also reported (oOR=0.96; 95%CI: 0.26,1.67; p=0.007). They did not report any differences in insomnia or daytime fatigue levels.

Conclusion: These results suggest that even though team-based athletes may not report more sleep complaints or daytime complaints, they may be at increased risk for less sleep and more sleep medication. Further work is needed to identify the sources of these differences to guide interventions.

Support: The REST study was funded by an NCAA Innovations grant. Dr. Grandner is supported by R01MD011600

0237

EFFECT OF CHRONIC INTERMITTENT HYPOXIA ON SPATIAL PERFORMANCE IN RATS

Xu, J. Geng, E. Brake, L. Wiemken, A. Keenan, B. Kubin, L. Schwab, R. University of Pennsylvania, Philadelphia, PA.

Introduction: Cognitive and spatial dysfunction is common among patients with obstructive sleep apnea (OSA). The cause of these abnormalities may be related to the effects of hypoxic damage in the brain during sleep. Here we report a rodent model for chronic intermittent hypoxia (CIH) that examines spatial performance tasks via a Barnes Maze paradigm. We hypothesized that increased severity of CIH yields decreased cognitive and spatial performance.

Methods: Three groups of rats were subject to varying levels of hypoxia conditions: sham (21% oxygen; n = 19), moderate (11% oxygen; n = 14), and severe (6% oxygen; n = 21). To deliver hypoxia, rats were exposed to three-minute cycles of oxygen between 21% and condition-specific nadir oxygen for 12 hours daily (during sleep) in specialized chambers. Barnes maze testing was performed at 0, 1, 2, and 3 months. Rats were placed on a circular platform with 19 shallow holes and one deeper target hole to escape the noxious sound. Each month, rats had 3 minutes to find the target hole in four daily trials over four consecutive days. Average maze completion time on day 4 was recorded.

Results: Rats from the three hypoxia groups did not differ significantly in mean maze completion time at baseline (0 months). Throughout the three months of exposure to hypoxic conditions, maze completion time on day 4 did not differ significantly from baseline for sham rats. However, by month 3, rats exposed to severe hypoxic conditions had a significantly larger percent increase from baseline compared to sham rats (p = 0.0358).

Conclusion: Our findings indicate that rats undergoing intermittent hypoxia perform worse than normoxic rats in spatial performance tasks. These data suggest there is a relationship between CIH and cognitive/spatial impairment.

Support: Funded by NIH P01 HL094307

0238

CAMPUS FOOD PANTRY ASSISTANCE IS RELATED TO BETTER PHYSICAL AND MENTAL HEALTH THROUGH ADEQUATE SLEEP AMONG COLLEGE STUDENTS IN A PUBLIC UNIVERSITY SYSTEM

Martinez, S.¹ Kalaydjian, S.² Ritchie, L.³ Nazmi, A.⁴ Prather, A.¹ ¹University of California, San Francisco, San Francisco, CA, ²University of California, Irvine, Irvine, CA, ³University of California Nutrition Policy Institute, Oakland, CA, ⁴California Polytechnic State University San Luis Obispo, San Luis Obispo, CA.

Introduction: Food insecurity is an issue among students in higher education and has been linked to insufficient sleep, and poor mental and general health. College campuses have quickly responded by establishing campus food pantries. However, the extent to which campus food pantries are ameliorating the impacts of food insecurity is unknown.

Methods: Online survey data were collected from a cross-sectional sample of 1,855 students who were food pantry users in the 10-campus UC system. Students were asked to report their number of visits to a food pantry in the past month, and to rate their general health, depressive symptoms, and number of days of enough sleep (in a week) before and after food pantry access. Changes in days of enough sleep, depressive symptoms and general health were computed. Demographic characteristics were obtained from institutional data. Path analysis was used to examine direct and indirect pathways from food pantry use to depressive symptoms and general health through enough sleep days, controlling for workstudy receipt, Pell grant receipt and family income.

Results: Students on average were 21.7 years old (SD= 3.5), and had more days of adequate sleep (25%), and improved depressive symptoms (43%) and general health (31%) after obtaining services from a campus food pantry. An increase in monthly food pantry

use was directly related to a decrease in depressive symptoms (β = 0.08, p<0.001) and an increase in general health (β = 0.07, p=0.001). Additionally, an increase in food pantry use related to an increase in getting more days of enough sleep (β =0.07, p=0.001), which in turn positively related to a decrease in depressive symptoms (β =0.18, p<0.001) and improved general health (β =0.24, p<0.001). **Conclusion:** Food pantry use had a positive relationship with student health outcomes, and enough sleep days played an important mediating role. Findings suggest that emergency food access may have a positive impact on student health outcomes.

Support: This study was funded by the UC Campus Basic Needs Committees.

0239

MINDFULNESS MODERATES THE RELATIONSHIP BETWEEN LUCID DREAMING, NIGHTMARE DISTRESS, AND SLEEP QUALITY

Zendels, P. Barngrover, S. Peach, H. Psychological Sciences department, Charlotte, NC.

Introduction: Nightmares can cause poorer sleep quality. Various mechanisms have been explored as potential treatments for nightmares, including mindfulness practices and lucid dreaming. Presently, little literature has looked at the interaction effects be-

tween mindfulness and lucid dreaming to reduce nightmare distress. Methods: A sample of 275 individuals was recruited from both the United States and Thailand via social media and a student pool of research subjects at UNC Charlotte. Data were recorded on participants' demographic information, lucid dreaming from the Lucidity and Consciousness in Dreams Scale (Voss et al., 2013), Mindfulness using the Five Facet Mindfulness Questionaire (Baer et al., 2006), Nighmares via the Nightmare Distress Questionaire (Belicki, 1992), and sleep quality using the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989). Higher scores on each measure were associated with more lucid dreaming, more mindful behaviors, more severe nightmares, and poorer sleep quality. PROCESS model 8 was run to conduct a moderated mediation analysis (Hayes, 2018). Lucid dreaming was used as the predictor; sleep quality as the outcome variable, nightmare distress as the mediator and mindfulness acted as a moderator on both the direct and indirect pathway of lucid dreaming onto the mediator and outcome.

Results: Mindfulness was a significant predictor at both the mediator and outcome variables. Nightmare distress was a significant predictor of sleep quality. A statistical trend (p=.054) suggests lucid dreaming may have a positive effect on nightmare distress. In the indirect path, lucid dreaming had a positive effect on PSQI scores only for individuals with low mindfulness.

Conclusion: The moderated mediation suggests that individuals with low mindfulness may see a decrease in sleep quality from lucid dreaming. This could be due to lucid dreaming being associated with more severe nightmares. A zero-effect size could not be ruled out of the confidence intervals for individuals of average or high mindfulness, but the data suggest that lucid dreaming alone may not help treat nightmares, but the combination of lucid dreaming and mindfulness therapies could promote lower distress and better sleep quality. **Support:** Psychological Sciences departmental funding

0240

COLLEGE FOOTBALL PLAYERS COMPARED TO OTHER COLLEGIATE ATHLETES: SYMPTOMS OF INSUFFICIENT SLEEP DURATION, INSOMNIA, AND SLEEP APNEA

Abdi, H.¹ Athey, A.¹ Auerbach, A.¹ Turner, R.² Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹ ¹University of Arizona, Tucson, AZ, ²George Washington University, Washington, DC.

Introduction: College athletes experience frequent sleep disturbances. Data from professional football players suggests high rates of sleep apnea symptoms. Little data is available on college football players, especially compared to other athletes.

Methods: Data were obtained from N=189 NCAA Division-I student athletes, including N=45 football players). Outcomes of interest came from the Sleep Disorders Symptom Check List (SDSCL) which rated symptoms on a frequency scale of 0: never and 4: >5 times/ week. Symptoms evaluated were daytime tiredness, any snoring, loud snoring, breathing pauses during sleep, and waking up choking/ gasping sleep apnea), as well as difficulty falling asleep, difficulty with nighttime awakenings, and early morning awakenings (insomnia). Other outcomes include self-reported sleep duration, Insomnia Severity Index, frequency of caffeine use, and frequency of use of medications to help with sleep. Linear and ordinal logistic regression analyses were adjusted for age, sex, year in school, socioeconomic status, and mood. Post-hoc analyses examined men only.

Results: Regarding sleep apnea symptoms, football players reported more snoring (oOR=3.14, p=0.01), loud snoring (oOR=4.38, p=0.008), breathing pauses (oOR=5.42, p=0.0499), and choking/gasping (oOR=8.51), but not daytime tiredness. Regarding insufficient sleep, football players reported no difference in sleep duration but decreased caffeine use (oOR=0.27, p=0.002). Regarding insomnia, football players showed no difference in ISI scores or insomnia symptoms, but increased likelihood of sleeping pill use (oOR=3.01, p=0.03). When analyses were restricted to men only, all of these relationships were maintained.

Conclusion: College football athletes may exhibit different sleep symptoms than other college athletes, as they exhibit more sleep apnearelated symptoms, without the increase in daytime symptoms, such as tiredness.

Support: The REST study was funded by an NCAA Innovations grant. Dr. Grandner is supported by R01MD011600

0241

HABITUAL DAYTIME SLEEPINESS AND THE TIMING OF USE OF ALCOHOL, TOBACCO, AND CAFFEINE

*Tubbs, A.*¹ *Hale, L.*² *Branas, C.*³ *Killgore, W. D.*¹ *Wills, C. C.*¹ *Grandner, M. A.*¹

¹University of Arizona, Tucson, AZ, ²Stony Brook University, Stony Brook, NY, ³Columbia University, New York, NY.

Introduction: Alcohol, caffeine, and tobacco are frequently used in the community, and the timing of use may impact daytime sleepiness. The present analysis examined relationships between daytime sleepiness and timing of alcohol, tobacco, and caffeine use in a real-world sample.

Methods: Data were from the Sleep and Healthy Activity, Diet, Environment, and Socialization (SHADES) study of N=1007 adults age 22–60 from the community. Daytime sleepiness was assessed with the Epworth Sleepiness Scale (ESS). Participants were asked if they had ever used caffeine, alcohol and tobacco. If they reported using a substance, they were then asked which times of day they were likely to use the substance: 5AM-8AM, 8AM-11AM, 11AM-2PM, 2PM-5PM, 5PM-8PM, 8PM-11PM, 11PM-2AM, and 2AM-5AM. Logistic regression analyses examined the relationship between ESS score and likelihood of use of substances at each time.

Results: ESS score was associated with increased odds of ever using alcohol (OR: 1.05, 95% CI: 1.01 to 1.09) or tobacco (OR:

1.04, 95% CI: 1.01 to 1.07). ESS score was associated with an increased likelihood of drinking alcohol in the morning (5AM-8AM, OR: 1.13) and night (11PM-5AM, OR: 1.05). Sleepiness was also associated with increased likelihood of tobacco use in the afternoon (11AM-2PM, OR 1.04) and night (11PM-2AM, OR 1.05). Finally, ESS score was associated with increased likelihood of caffeine use during the midday and afternoon (11AM-5PM, OR: 1.04).

Conclusion: Greater sleepiness is associated with use of alcohol in the morning and at night, and with use of tobacco in the afternoon and at night. Finally, increased sleepiness was associated with caffeine use during the latter part of the workday. Some of these use patterns may be a cause of sleepiness (e.g., morning alcohol use or nighttime smoking) and some a consequence (e.g., daytime caffeine and tobacco use). More research on the impact of real-world sleepiness on real-world substance use is warranted.

Support: This work was supported by a grant from Jazz Pharmaceuticals Dr. Grandner is supported by R01MD011600The SHADES study was funded by R21ES022931

0242

EFFICIENT PERCEPTION-ACTION COUPLING RELATES TO MORE SLOW WAVE SLEEP IN MILITARY PERSONNEL

LaGoy, A. D. Eagle, S. R. Sinnott, A. M. Beckner, M. E. Conkright, W. R. Flanagan, S. D. Martin, B. J. Nindl, B. C. Germain, A. Ferrarelli, F. Connaboy, C. University of Pittsburgh, Pittsburgh, PA.

Introduction: The ability to adapt actions to perceptions of environmental constraints, perception-action coupling, may be compromised by military operational stress (caloric restriction, sleep disruption, physical exertion). Differences in sleep may influence susceptibility to these stressors. We investigated perception-action coupling during simulated military operational stress and the influence of sleep on perception-action coupling.

Methods: During a 5-day simulated military operational stress protocol, thirty-six (6 female) service members (25.8 \pm 4.7 years) completed three trials of a perception-action coupling task (PACT) in the evening after a night of baseline sleep (BASE), two nights of sleep restriction (T1) and a night of recovery sleep (T2). Participants had 8-hr for baseline and recovery sleep (2300-0700) and 4-hr disturbed sleep on sleep restriction nights (0100-0300 and 0500-0700). Polysomnography was used to determine time spent in different sleep stages: stage 2 (N2), slow wave (SWS) and rapid-eye movement (REM). The tablet-based PACT requires participants make quick, accurate perceptual judgments and responses about the ability of virtual balls to fit through virtual apertures. Linear mixed models were used to assess interaction and main effects of study day and prior sleep on PACT response time (RT) and accuracy (ACC). Results: No significant sleep x time interactions or significant main effect of time were found for RT or ACC. A significant main effect of SWS was found for RT ($F_{1,88,307} = 4.331$, p = .04). Higher SWS was related to lower (faster) RT. No significant main effects of other sleep stages were found.

Conclusion: Perception-action coupling was maintained during simulated military operational stress. Participants with more SWS across the study responded faster during the PACT but N2 and REM sleep did not relate to perception-action coupling performance, suggesting a specific effect of SWS on perception-action coupling abilities and behaviors.

Support: Department of Defense Award #W81XWH-17-2-0070 (PI: Nindl)

0243

COMMUNITY-LEVEL DAYTIME SLEEPINESS AND SUBSTANCE USE: IMPLICATIONS OF SLEEP TIME AND MENTAL HEALTH

Khader, W. S.¹ Tubbs, A.¹ Fernandez, F.¹ Chakravorty, S.² Hale, L.³ Branas, C.⁴ Barrett, M.² Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹ ¹University of Arizona, Tucson, AZ, ²University of Pennsylvania, Philadelphia, PA, ³Stony Brook University, Stony Brook, NY, ⁴Columbia University, New York, NY.

Introduction: Daytime sleepiness is associated with impaired functioning and well-being. Those with more sleepiness may turn to illicit substances to overcome these problems. The present study examined whether community-level daytime sleepiness is associated with the likelihood of drug use.

Methods: Data were pulled from the Sleep and Healthy Activity, Diet, Environment, and Socialization (SHADES) study of N=1007 community adults (age 22–60). Daytime sleepiness was assessed with the Epworth Sleepiness Scale (ESS). Use of different substances was assessed with the Alcohol, Smoking, and Substance Involvement Screening Test (ASSIST). The present analyses examined use within the past month of alcohol, tobacco, cannabis, cocaine, amphetamines, inhalants, sedatives, hallucinogens, and illicit opioids. A separate item assessed caffeine. Ordinal logistic regression analyzed ESS score as a predictor of frequency of substance use adjusted for age, sex, education, and race/ethnicity. Additional models included habitual sleep duration and score on the PHQ9 depression scale.

Results: In sociodemographically-adjusted analyses, ESS score was associated with an increased risk of using tobacco (OR=1.04, p=0.015), cannabis (OR=1.04, p=0.014), cocaine (OR=1.07, p=0.009), amphetamines (OR=1.06, p=0.025), inhalants (OR=1.13, p=0.002), sedatives (OR=1.07, p=0.003), hallucinogens (OR=1.12, p=0.001), and opioids (OR=1.12, p=0.0001). Controlling for sleep duration did not significantly affect these relationships, while controlling for depression made every association non-significant except hallucinogens (OR=1.09, p=0.040).

Conclusion: Daytime sleepiness was associated with increased use of nearly all drug categories, but not alcohol or caffeine. Public consumption of alcohol and caffeine might be sufficiently common that the presence of their use cannot be adequately associated with sleepiness. Moreover, the increased frequency of drug use with sleepiness is not linked to sleep deprivation but may reflect emotional distress.

Support: This work was supported by a grant from Jazz Pharmaceuticals Dr. Grandner is supported by R01MD011600

0244

EXAMINING PILOT SAFETY PERFORMANCE INDICATORS AT CRITICAL PHASES OF FLIGHT ACROSS MULTIPLE FLIGHT LEGS DURING COMMERCIAL AIRLINE TRIPS

Lamp, A.¹ Soriano Smith, R. N.¹ Rasmussen, I.¹ Keller, C.¹ Basiarz, E.¹ Belenky, G.¹

¹Washington State University, Spokane, WA, ²Washington State University, Spokane, WA.

Introduction: Prior simulation and operational studies have started to address whether the number of consecutive flight segments negatively affects cognitive performance, fatigue, and sleepiness, without reaching a clear consensus. This study expands this literature by determining whether there are significant changes in cognitive performance, fatigue, and sleepiness at critical phases of flight across multiple flight segments, while accounting for the number of segments, flight direction, trip day, and time-of-day.

Methods: Fifty commercial airline pilots were studied. Each pilot flew two separate short-haul trips, each ranging from 1–4 days and 1–10 flight segments. Cognitive performance, fatigue, and sleepiness were assessed at top-of-climb (TOC) and top-of-descent (TOD) of each flight segment and each trip day. Cognitive performance, fatigue, and sleepiness were assessed using Psychomotor Vigilance Task (PVT) speed, Samn-Perelli (SP) ratings, and Karolinska Sleepiness Scale (KSS) ratings, respectively. Data were analyzed using Wilcoxon t-tests and verified using ANOVAs.

Results: Mean PVT speed (Cohen's d =0.57), SP ratings (Cohen's d = 0.73), and KSS ratings (Cohen's d = 0.63) were significantly worse at TOD than TOC (p < 0.001); and, significantly varied across flight segments (p<0.001). Cognitive performance, fatigue, and sleepiness were consistently and significantly degraded around the fifth flight segment, improved around the sixth to eighth flights segments, and were subsequently degraded around the eighth to tenth flight segments.

Conclusion: The results indicate that cognitive performance, fatigue, and sleepiness vary across flight segments, trip day, and phase of flight. Results suggest that these safety performance indices degrade after five segments, and further degrade after eight flight segments. The results presented could be used to inform future airline scheduling and regulation.

Support: This work has been supported by United Airlines.

0245

RELATIONSHIP OF CHRONOTYPE WITH SLEEP DURATION, DAYTIME SLEEPINESS, AND SUSTAINED ATTENTION IN HIGH SCHOOL STUDENTS: A PILOT STUDY

Morrison, S. W.¹ Scheer, F. A.² Mason, I. C.²

¹Byram Hills High School, 12 Tripp Lane, Armonk, NY, ²Medical Chronobiology Program, Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, Boston, MA 02115; Division of Sleep Medicine,

Department of Medicine, Harvard Medical School, Boston, MA.

Introduction: People with a later chronotype typically sleep later and perform better later in the day. In an early high school schedule, students with later chronotypes earn lower average grades than those with earlier chronotypes. However, other effects of late chronotype on high school students are poorly understood. This study examined the relationship of chronotype with sleep duration, daytime sleepiness and sustained attention in high school students.

Methods: This study was conducted at Byram Hills High School in Armonk, NY, where classes begin at 7:45am. During three class periods, 36 participants (female n=24, n=12, 14-17 years old) completed the Morning-Eveningness Questionnaire (MEQ), Karolinska Sleepiness Scale (KSS), and Psychomotor Vigilance Test (PVT) during their class. Subjects recorded time spent on homework/extracurricular activities and bedtime/waketime from the night prior to the assessment. Results: Later chronotype (lower MEQ score) was significantly associated with later bedtime, shorter sleep duration, and increased sleepiness (r=-0.82, p<0.001; r=0.72, p<0.001; and r=-0.40, p=0.016; respectively). Shorter sleep duration was significantly correlated with increased sleepiness (r=-0.34, p=0.045). Increased workload (time on homework/extracurriculars) was significantly associated with later bedtime (r=0.42, p=0.011). Chronotype, sleep duration, and sleepiness showed no significant association with PVT scores (r=-0.16, p=0.360; r=-0.10, p=0.933; r=0.09, p=0.619, respectively); however, increased workload was significantly associated with increased PVT scores (r=-0.35, p=0.041).

Conclusion: These results are important for the wellbeing of high school students, as they show relationships between late chronotype, short sleep, and increased sleepiness during class in an early school schedule. Further research is needed to determine the best school schedule for high school students based on individual differences in chronotype.

Support: We thank the Authentic Science Research program at Byram Hills High School, specifically teachers Mrs. Stephanie Greenwald, Dr. Caroline Matthew, and Mrs. Megan Salomone.

0246

IMPROVE SLEEP IN COLLEGE STUDENTS THROUGH LIFESTYLE CHANGE ASSIGNMENT

Carter, P.

Capstone College of Nursing, Tuscaloosa, AL.

Introduction: Emerging adults experience a 'new found freedom' in college. Many are making decisions about their sleep-wake cycles leading to ineffective patterns. Poor sleep in college students is influenced by non-modifiable (class time, course assignments) and behavior based (sleep schedule, screen time, exercise and diet) factors. Change is difficult! However, GPA is a great student motivator. This project developed and tested the acceptability of a guided lifestyle change assignment for sleep.

Methods: Undergraduate students at a Southern USA R-1 University who were enrolled in a signature course (Sleep: Are We Getting Enough?) were eligible. Context: Twice weekly lectures presented scientific findings related to sleep science and applied findings to human experiences. Intervention: students completed a guided lifestyle change assignment for sleep. Students indentified a specific sleep related behavior to change (change goal) over a 1 month period (November), submitted an initial lifestyle change plan, three progress reports, and a final reflection.

Results: 800 students participated over 6 years (Fall semesters 2014–2019). Students were primarily female (56%) and nine majors were represented. Goals were grouped into 4 lifestyle foci (Exercise, Screen time, Sleep Schedule, Diet). Progress reports identified barriers and facilitators and plans to address these. Final reflections evaluated overall performance and major takeaways. Qualitative perceptions and quantitative outcomes will be presented in detail. Additionally, discussions of unanticipated outcomes and guidance for incorporating this assignment into existing courses will be presented.

Conclusion: College students can make positive changes to improve their sleep. Guidance to identify and address facilitators and barriers to change is important to create and sustain change. Motivations to change are different for emerging adults vs. older populations. It may be that the most effective way to improve sleep quality in college students is to 'attach a grade' to the activity. **Support:**

0247

EXECUTIVE FUNCTION AND SOCIAL-COGNITIVE PREDICTORS OF BEHAVIORAL SLEEP RESTRICTION

Irish, L. A.^{1,2} Mead, M. P.¹ Veronda, A. C.¹ van Lamsweerde, A. E.³ Gunstad, J.⁴

¹North Dakota State University, Fargo, ND, ²Sanford Center for Biobehavioral Research, Fargo, ND, ³Quantum Improvements Consulting, Orlando, FL, ⁴Kent State University, Kent, OH.

Introduction: Although insufficient sleep is a global public health concern, the causes of insufficient sleep in the general population are variable and complex. A substantial number of individuals

engage in behavioral sleep restriction (BSR), defined as limiting nocturnal time in bed to less than the recommended total sleep time, but little is known about the factors that influence BSR. Notably, the impact of cognitive processes on BSR has not been empirically tested, though processes such as executive function (EF) and social-cognitive beliefs are clearly relevant to other health-related behaviors. This study sought to investigate the extent to which EF and social-cognitive factors predict BSR.

Methods: Participants included 205 healthy adults aged 18–35. EFs (i.e., inhibitory control, working memory, cognitive flexibility) were assessed with a neurocognitive task battery and social-cognitive factors (i.e., attitudes, norms, perceived behavioral control) related to healthy sleep duration were self-reported, followed by 1 week of actigraphy. BSR was represented as the number of nights that actigraphy-estimated time in bed was fewer than 8 hours.

Results: On average, participants engaged in BSR 3 nights per week. Hierarchical linear regressions were tested separately for EFs and socialcognitive factors. Results revealed that, after controlling for general intelligence, EF did not predict BSR. In contrast, social-cognitive factors did predict BSR (F(202,3)=8.71, p<001), with both attitudes (β=.20, p=.005) and perceived behavioral control (β=.15, p<.001) emerging as significant predictors. Interactions between EFs and social-cognitive factors were also explored, suggesting interactions between inhibitory control and perceived behavioral control (p=.03) as well as cognitive flexibility and attitudes (p=.05).

Conclusion: Taken together, these results highlight the high frequency of BSR and the role that social-cognitive factors may play in facilitating BSR among adults who may otherwise obtain healthy sleep. Efforts to promote sleep health in the general population would benefit from greater understanding of modifiable factors that increase BSR.

Support: This research was supported by an Institutional Development Award (IDeA) from the National Institute of General Medical Sciences of the National Institutes of Health under grant number 5P30GM114748.

0248

DOES ATTENTIONAL CONTROL RELATE TO SLEEP DISRUPTION AND REPETITIVE NEGATIVE THINKING?

Acenowr, C. Coles, M. Stewart, E. Binghamton University, Binghamton, NY.

Introduction: Insomnia is associated with increased repetitive negative thinking (RNT) and poor attentional control. There is increasing interest in the relevance of these processes for psychopathology. For example, Cox, Cole, Kramer and Olatunji (2018) proposed that focusing and shifting in attentional control may help explain the link between sleep disturbance and RNT. In support, attentional focus was found to be significant in the relationship between insomnia and RNT. As this study looked at disorder-specific measures of RNT and only insomnia, the current study aimed to replicate and extend the findings by also examining circadian sleep disruption and transdiagnostic RNT.

Methods: The current study included 127 participants. Sleep disruption was assessed by the SLEEP-50 (Spoormaker, Verbeek, van den Bout & Klip, 2005). This measure provides several subscale scores, including disruption in circadian rhythms and insomnia. The Attentional Control Scale (Derryberry & Reed, 2002) is a measure of attentional focus and shifting which was also utilized. Lastly, the Perseverative Thinking Questionnaire (Ehring et al., 2011) is a widely used transdiagnostic measure of RNT.

Results: Pearson's Correlations indicated that both insomnia and circadian disruptions were significantly associated with RNT

(insomnia, r=.27; circadian, r=.24). Mirroring the results of Cox, Cole, Kramer and Olatunji, attentional focus was significant (insomnia, r=-.29; circadian, r=-.28), whereas attentional shift was not (insomnia, r=.02; circadian, r=.06).

Conclusion: The connection between sleep disruption and factors that contribute to psychopathology needs to be better understood. This study differentiates types of attention and their relation to insomnia and circadian sleep disruption, and RNT. If attentional focus can link sleep disruption and RNT, clinicians can move one step closer to understanding the development of risk factors that may jeopardize an individual.

Support: n/a

0249

EFFECTS OF ALCOHOL CONSUMPTION ON SLEEP-WAKE PATTERN OF A RAT MODEL OF ANXIETY

Fierro, A.¹ Cortés, C.¹ Eguibar, J.^{2,1}

¹Institute of Physiology, Puebla, MEXICO, ²Research Office, VIEP, Puebla, MEXICO.

Introduction: Anxiety is an important factor for self-administered alcohol as a tool to reduce its symptoms. However, alcohol is capable to disrupt sleep-wake patterns in subjects with medium- to high-alcohol consumption. We have selectively bred two sublines from Sprague-Dawley rats that differ on its yawning frequency. High-yawning (HY) rats have a mean of 20 yawns/h, whereas the Low-yawning (LY) rats have only 2 yawns/hour. LY rats also showed anxious responses when evaluated on standardized tests. The aim of this study was to assess the changes on sleep-wake patterns after chronic alcohol consumption.

Methods: We used 8 males from HY and LY sublines at 3 months of age, they lived in acrylic cages with water and food pellets available *ad libitum* under a 12:12 light-dark cycle (lights on at 0700) and temperature of 21 ± 1 °C. All subjects were implanted to record EEG, EMG and EOG to characterize sleep-wake phases. A baseline sleep-wake recording was obtained for 24 h. A solution of 9.6% alcohol was administered as a single source of hydration for seven days and then a second sleep-wake recording was obtained. After that period, an additional bottle containing purified water was available. Position of the bottles was randomly changed daily. Water and alcohol consumption were measured daily for a period of 3 weeks and then a third sleep-wake recording was obtained.

Results: LY rats consumed more alcohol than HY rats (P<0.05), and they had an increase of bouts and duration of slow wave sleep and REM sleep on their active phase after alcohol administration (P<0.05). **Conclusion:** LY rats display an anxious behavior and therefore consumed more alcohol compared to HY rats, and only LY rats were susceptible to alcohol effects on sleep on their active phase. **Support:** Partially supported by CONACYT grants 243333 and 243247 to CC and JRE, respectively. Grants from VIEP-BUAP 2018 and CA in Neuroendocrinología BUAP-CA-288.

0250

THE DAY-TO-DAY ASSOCIATIONS BETWEEN SLEEP CHARACTERISTICS, AFFECT, AND AFFECT REACTIVITY

Wong, P. M.¹ Hasler, B. P.² Kamarck, T.¹ Wright, A.¹ Hall, M.² Carskadon, M. A.³ Manuck, S. B.¹

¹Department of Psychology, University of Pittsburgh, Pittsburgh, PA, ²Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, PA, ³Department of Psychiatry & Human Behavior, Alpert Medical School of Brown University, Providence, RI. **Introduction:** Despite the high co-occurrence of sleep and mood disturbances, day-to-day associations between sleep characteristics (sleep duration, continuity, timing) and dimensions of mood (positive affect, PA, and negative affect, NA) remain unclear. Few field studies have tested whether sleep changes may affect mood by altering people's emotional response to daily experiences outside the laboratory. The current study aimed to examine how sleep characteristics influence affective states and a measure of emotional response to daily experiences—affect reactivity.

Methods: Participants were healthy, midlife adults (30-54 yrs old, N = 462, 47% male) drawn from the Adult Health and Behavior Project-Phase 2 study. Sleep was measured with actigraphy across a 4-day monitoring period; hourly self-report measures of affect, work demand, and social interactions were collected via ecological momentary assessment. Affect reactivity was quantified as hour-to-hour changes in affect following these daily experiences. We used hierarchical linear modeling to examine whether sleep characteristics on a given night predicted average daily affect and moderated affect reactivity the following day.

Results: When participants slept later than their average sleep time on a given night, they reported greater NA the following morning (before 12pm) and afternoon (12-6pm; B's =.03, p's<.05). When participants slept longer than their average sleep duration, they subsequently reported greater NA throughout the following day (B's =.02, p's<.05). Sleep efficiency was unrelated to affect report (p's > .05). While episodes of greater work demand and social conflict predicted lower PA and higher NA (p's<.05), participants' sleep characteristics did not moderate these associations (p's>.05). Conclusion: Overall, our findings suggest proximal associations between sleep characteristics and next-day mood. While previous studies have shown effects of sleep disruptions on affect reactivity, we did not observe such associations in the context of small, day-to-day fluctuations in sleep characteristics among healthy individuals. Support: National Institutes of Health Grant PO1 HL040962 (to S.B.M.).

0251

ASSOCIATION BETWEEN SLEEP DISRUPTION AND WORKPLACE PRODUCTIVITY: THE HIDDEN COST OF CAREGIVING

Robbins, R.¹ Weaver, M. D.¹ Barger, L. K.¹ Quan, S. F.¹ Zhivotovsky, S.² Czeisler, C. A.¹

¹Division of Sleep and Circadian Disorders, Boston, MA, ²Harvard College, Cambridge, MA.

Introduction: Caregiving for older spouses or family members is common with an aging population. The responsibilities caregivers maintain can disrupt sleep, which may present workplace productivity consequences. We sought to test whether caregiving-related sleep disturbance was associated with workplace productivity among adults caring for older spouses and family members.

Methods: Cross-sectional analysis of the 2011 National Study of Caregiving dataset was conducted. The dataset is comprised of family members and unpaid caregivers to older adults in the U.S. (age 65 and above) receiving assistance with self-care, mobility, or household activities. Caregivers reported the frequency of sleep disturbance ("In the last month, how often did caregiving cause your sleep to be interrupted") from 0 ("never" or "rarely") to 1 ("some nights") and 2 ("most nights" or "every night"). Workplace measures included: 1) presenteeism, or the degree to which caregiving affected productivity at work, measured from 1 "not at all" to 10 "very much"; 2) absenteeism, or the proportion of hours missed from work due to caregiving to total hours worked, and 3) productivity loss, or

the sum of absenteeism and presenteeism. Generalized linear models examined the relationship between workplace productivity and sleep disturbance, while controlling for relevant covariates, including age, income, education, and self-reported health.

Results: Participants comprised 2,007 caregivers. Mean (SD) age was 63.5 (12.9), and 66.4% of the participants were women (n=1,334). The most common relationship to the older adult was daughter (n=704, 35.1%), followed by spouse/partner (n=422,21.0%), and son (n=310,15.5%). Compared to no "rarely" or "never" experiencing sleep disruption, reporting disruption "most nights" or "every night" was associated with presenteeism (OR=1.2, 95%CI:1.1–1.2), absenteeism (OR=1.1, 95%CI: 1.0–1.1), and total productivity loss (OR=1.22, 95%CI: 1.1 to 1.3).

Conclusion: Caregiving-related sleep disruption is a risk factor for workplace productivity losses, hindering caregivers from professional advancement and further degrading their caregiver capacity. Future study of means for improving sleep among caregivers is warranted. **Support:** T32HL007901

0252

INFLUENCE OF SLEEP ON NEGATIVE SOCIAL JUDGMENT

Corona, F. E.¹ Mednick, S. C.¹

¹University of California, Irvine, Irvine, CA, ²University of California, Irvine, Irvine, CA.

Introduction: Sleep plays an important role in emotional regulation. Emotional regulation can be disrupted by psychosocial stress, including social judgment. It is not known whether sleep can regulate emotions arising from social judgment. The aim of this study is to determine if sleep can reduce emotional reactivity associated with negative social judgment.

Methods: On day one, subjects participated in a social judgment task (SJT) in the morning, followed by a nap or wake condition. Subjects' physiological responses to social judgment task (skin conductance and heart rate) and mood were measured before and after the SJT, after the nap/wake condition, and once on day three. **Results:** Mood ratings decreased after social judgment for all subjects, but increased following the nap intervention. Mood ratings improved after the 2 day delay. There were no changes in social status. Skin conductance during judgment decreased for subjects in nap condition, but not wake. After a 2 day delay, skin conductance decreased across subjects, with those in nap condition experiencing greater decrease. Heart rate response during social judgment exposure decreased only for those in nap group, with a 2 day delay showing decreased heart rate response across all subjects.

Conclusion: A nap directly following social judgment buffers negative response to the experience by decreasing emotional reactivity as measured by mood, skin conductance, and heart rate response. No differences were found between nappers and non-nappers on day three, suggesting that sleep benefits emotional reactivity associated with the stress of social judgment and that these benefits can happen with a nap or a night of sleep, more than a period of wake. **Support:** University of California, Irvine.

0253

EATING BEHAVIORS, PHYSICAL ACTIVITY, AND SLEEP IN SHIFT WORKERS: RESULTS FROM A COMBINED FIELD AND LABORATORY STUDY

Chen, Y. Lauren, S. Shechter, A.

Columbia University Medical Center, New York, NY.

Introduction: In shift workers, short sleep duration combined with circadian misalignment may affect behaviors that impact regulation of energy balance and metabolism. We conducted a combined fieldand-laboratory study to determine how real-life shift work affects diet, physical activity, and sleep via objective and self-report measures. **Methods:** Participants were day (n=12) and night (n=12) shift workers from an urban hospital setting (nurses and technicians, all female). During the field portion of the study, participants wore a wrist-mounted accelerometer to track sleep and physical activity during their series of shifts, and completed a computer-assisted 24-hour dietary recall. After awakening from the sleep episode following the final work shift, participants entered the laboratory in the fasted state and underwent an ad libitum 14-item test-meal buffet to objectively quantify food choice and intake.

Results: Sleep duration was significantly shorter and worse quality in night vs. day workers. Physical activity levels were not different between groups. Based on 24-h dietary recall, night vs. day workers consumed less protein (65.9 \pm 39.0 vs. 87.2 \pm 40.7 g, p=0.01) and fiber (12.5 \pm 6.0 vs. 16.9 \pm 6.2 g, p=0.01), but did not differ in daily intakes of calories, fat, or carbohydrate. Night vs. day workers reported a longer daily window of eating duration (14.2 \pm 3.8 vs. 12.0 \pm 1.5 h, p=0.02). In the lab test-meal, there were no group differences in total calories consumed. When expressed as percent of calories consumed, night vs. day workers had lower protein intake (11.82 \pm 4.05 vs. 16.03 \pm 5.69 %; p=0.05).

Conclusion: To our knowledge, this was the first study to include a laboratory-based behavioral assessment of food choice/intake in real-life night and day shift workers using objective measures. We did not assess measures of circadian phase so can only assume that circadian misalignment, in addition to the disturbances in sleep duration and quality, contributes to findings. Changes to dietary patterns in night vs. day workers (namely, reduced protein intake which may affect satiety, and prolonged daily eating duration window) may present potential pathways by which night shift work contributes to risk for overweight and obesity. **Support:** UL1TR000040

0254

THE EFFECTS OF CHRONIC SLEEP RESTRICTION ON MULTIPLE OBJECT TRACKING

*Costedoat, G.*¹ *Feria, C. S.*² *Pradhan, S.*^{3,1} *Stone, L. S.*⁴ *Flynn-Evans, E. E.*⁴

¹San Jose State University Research Foundation, Moffett Field, CA, ²San Jose State University, San Jose, CA, ³Department of Management, Menlo College, Atherton, CA, ⁴NASA Ames Research Center, Moffett Field, CA.

Introduction: The ability to simultaneously track numerous moving objects in the presence of irrelevant stimuli is essential for successfully carrying out a variety of tasks. Sleep loss impairs neurocognitive functioning and, as a result, attentional processing capacity is reduced. The objective of the current study was to determine if performance on the multiple object tracking (MOT) task was adversely impacted by a week of chronic sleep restriction (CSR).

Methods: Twelve healthy participants (6 males, 6 females) kept a fixed sleep-wake schedule, with a constant waketime, at home for four weeks (actigraphy confirmed compliance). During weeks one and three, participants maintained 9 hours in bed. During weeks two and four, participants were randomly assigned to 5 and 9 hours of sleep. Following weeks two and four, participants completed a 13-hour laboratory visit under dim light (< 15 lux), where they maintained a constant posture and were provided with hourly isocaloric snacks. MOT was presented

at approximately 6 and 8 hours after waking. Participants were required to track four, five, or six moving targets in the presence of identical distractors (always 12 total objects).

Results: Participants slept significantly less when assigned to 5 (M = 4.43 hours, SD = 0.33 hours), compared to 9 hours of sleep (M = 7.42 hours, SD = 0.42 hours; F(1, 22) = 206.89, p = 0.00). The proportion of correct MOT responses was significantly lower following 5 (M = 0.70, SD = 0.15) compared to 9 hours of sleep (M = 0.77, SD = 0.12; F(1, 22) = 10.29, p < .05).

Conclusion: A week of CSR adversely impacted MOT performance compared to a week of sleep satiation. These findings have implications for individuals, such as air traffic controllers and truck drivers, who must visually track multiple moving objects, often while chronically sleep deprived.

Support: Supported by the Force Health Protection Program of the Office of Naval Research (SAA2402925-1, Contract Award no. N0001418IP00050).

0255

COMPARING PERFORMANCE, FATIGUE AND SLEEPINESS BETWEEN SHORT-HAUL AND ULTRA LONG-HAUL COMMERCIAL AIRLINE OPERATIONS

Lamp, A. Rasmussen, I. Soriano-Smith, R. Keller, C. Basiarz, E. Belenky, G.

Washington State University - Spokane, Spokane, WA.

Introduction: Safety performance indicators (SPIs) are used in aviation to determine if a trip that is non-compliant with federal regulations is safe to fly. Exemptions to regulations can be granted if a safety case demonstrates that the SPIs for an alternative means of compliance (AMOC; i.e., a trip outside regulations) are noninferior to SPIs for a safety standard operation (SSO; i.e. a trip compliant with regulations). Through this process, it has previously been suggested that ultra-long-range flights are non-inferior to long-range flights due to increased sleep opportunity. We determined whether SPIs for non-compliant ultra-long-range (ULR) trips are non-inferior to those for compliant short-haul (SH) trips. Methods: Performance, fatigue, and sleepiness were assessed at the top of descent (TOD) of flight segments using the Psychomotor Vigilance Task (PVT), Samn-Perelli (SP) fatigue scale, and Karolinska Sleepiness Scale (KSS), respectively. Data were analyzed using non-inferiority testing. Two different ULR trips with different TOD times (ULR trip 1: n=81; ULR trip 2: n=22) were compared to two types of SH trips, including one trip that contained one or more all-night flights (SH trip 1: n=48) and one trip with zero all-night flights (SH trip 2: n=47).

Results: Non-inferiority was found for the SPIs at most comparison points. For example, comparing the SPIs for ULR trip 2 and SH trip 1 at final TOD, non-inferiority was found for all SPIs. In contrast, comparing the SPIs for ULR trip 1 and SH trip 1 at final TOD, non-inferiority was found for SP and KSS, while non-inferiority for PVT was only suggested.

Conclusion: The findings suggest that the AMOC trips are as safe as or safer than the compliant SH trips. This raises questions regarding the structure of SH trips and how differences in the structures play a role in performance, fatigue and sleepiness. **Support:** United Airlines

0256

CAN A SINGLE NIGHT'S SLEEP ARCHITECTURE PREDICT NEXT-DAY AFFECT AND AFFECT REGULATION?

ten Brink, M. Zhang, J. Manber, R. Kreibig, S. Gross, J. J. Stanford University, Stanford, CA.

Introduction: Disrupted sleep has been shown to alter next-day affective functioning by decreasing positive mood, increase negative reactivity, and impairing people's ability to regulate their affect. However, few studies have examined how particular aspects of sleep timing and architecture influence typical daytime affect. Based on clinical and laboratory research on reactivity to emotional stimuli, we hypothesized a particularly important role for REM sleep in next-day affective functioning.

Methods: We analyzed a subset of N = 64 from a larger study of healthy adult community members who had complete data from a single night of ambulatory polysomnography (PSG) at home as well as morning and evening diaries. We tested whether PSG-derived total sleep time, sleep efficiency, REM percentage, REM latency, and SWS latency predicted self-reported negative mood and use of affect regulation the following morning and day using linear regression models.

Results: Surprisingly, there were no significant associations (p > 0.05) between any of the five PSG sleep architecture measures with self-reported negative morning mood, daytime mood, or daytime affective regulation.

Conclusion: This finding indicates that objective sleep measures from a single night of at home PSG in a healthy adult population are not necessarily predictive of an individual's subsequent emotional well-being on the following day.

Support: National Science Foundation Graduate Fellowship

0257

PRELIMINARY FINDINGS: ATTENTIONAL BIAS FOR FOOD CUES UNRELATED TO TIME AWAKE OR CIRCADIAN PHASE DURING FORCED DESYNCHRONY IN ADOLESCENTS

Wong, P. M.¹ Barker, D. H.² Raynor, H. A.³ Hart, C.⁴ Carskadon, M. A.²

¹Department of Psychology, University of Pittsburgh, Pittsburgh, PA, ²Department of Psychiatry & Human Behavior, Alpert Medical School of Brown University, Providence, RI, ³Department of Nutrition, University of Tennessee, Knoxville, TN, ⁴Department of Public Health, Temple University, Philadelphia, PA.

Introduction: Sleep quantity and quality influence attentional bias, and attentional bias for food cues has been associated with body weight. Whether the endogenous circadian cycle and/or time from awakening to bedtime contribute to attentional bias for food and differences in weight remain unclear. Using a 28-h forced desynchrony (FD) design, we hypothesized that adolescents with overweight (OW) and obesity (O) would have more attentional bias for food cues later in the wake episode and at a later circadian phase compared to adolescents with a healthy weight (HW).

Methods: 50 (28 male) adolescents (12-15yr) completed 7 FD cycles. Participants completed an attentional bias task that included three food word categories (savory, sweet, fruits/vegetables) and one non-food word category (school supplies). The task was completed at 6 fixed times each cycle: Task 1 was 1.3h after scheduled awaking, Task 2 was 2h after Task 1, and Tasks 3–6 followed at 3-h intervals. Weight categorization used body mass index (BMI) percentiles (CDC): HW (>5th and <85th; n=24), OW (85th and <95th; n=13), or O (\geq 95th; n=14). Endogenous circadian period was determined using salivary melatonin onsets (Mean: HW=23.88h; OW=24.01h; O=23.86h). Effect of circadian phase and time since scheduled awakening was assessed by mixed effects modeling using 6 circadian and 6 time-awake bins.

Results: We found no significant differences between weight groups in attentional bias for any of the food categories (p's >.05). We also

saw no significant time awake effects or circadian influence on attentional bias, nor did time awake or circadian phase moderate the associations between weight category and attentional bias (p's >.05). **Conclusion:** Weight groups did not impact food-related attentional bias across the wake episode or circadian phase. Future directions will explore whether attentional bias for food types predicts food choice and food consumption in this study. **Support:** DK101046

0258

EARLY SEMESTER SLEEP VARIABILITY PREDICTS DEPRESSION AMONG COLLEGE STUDENTS

Price, S.¹ Chikersal, P.¹ Doryab, A.¹ Villalba, D.¹ Dutcher, J.¹ Tumminia, M.² Cohen, S.¹ Creswell, K.¹ Mankoff, J.³ Dey, A.³ Creswell, D.¹

¹Carnegie Mellon University, Pittsburgh, PA, ²University of Pittsburgh, Pittsburgh, PA, ³University of Washington, Seattle, WA.

Introduction: Sleep is a critical behavior predicting mental health and depressive symptomatology in young adults. The extant scientific literature generally focuses on self-reported sleep measures over relatively short time frames. Here, we examine whether actigraphy-measured sleep variables early in the academic semester predict depressive symptomatology at the end of the semester among first and second year college students. There is currently debate in the sleep literature about which sleep variables are the most robust predictors of depression among young adults. In this study, we evaluate total sleep time, midpoint sleep time, and sleep variability where variability is defined by the mean-squared successive difference (MSSD) of midpoint sleep as predictors of depression.

Methods: The sample consisted of 160 first and second year college students at a private American university. The students completed a beginning and end of semester assessment of depressive symptomatology using the Center for Epidemiologic Studies Depression Scale (CES-D), and wore a Fitbit throughout the semester to capture sleep features of interest: total sleep time (TST), midpoint sleep, and midpoint MSSD.

Results: When controlling for beginning of semester CES-D, early semester (weeks 3–6) midpoint sleep MSSD significantly (p < 0.05) predicted increased end of semester CES-D. These effects were specific to the sleep variability measure (MSSD). Total sleep time and sleep chronotype (i.e. midpoint sleep) were not significant predictors of end of semester depressive symptomatology.

Conclusion: Early semester sleep window variability among college freshmen, particularly during stressful midterm exams, is a robust risk factor for depression among college students. This work contributes to initial actigraphy studies suggesting that MSSD measures of sleep window variability foster increased mental health risks among young people. This work calls for further investigation to understand possible causal relationships between sleep variability and mental health.

Support: This work was supported by the Life@CMU project funded by the Carnegie Mellon University Provost's Office.

0259

SHIFTWORKERS ARE AT INCREASED RISK OF DEVELOPING CHRONIC PAIN AND OPIOID USE DISORDERS: A STUDY OF 116,000 UK BIOBANK PARTICIPANTS OVER A DECADE

Gao, L.^{1,2} Li, P.^{2,3} Cui, L.² Luo, Y.^{1,4} Vetter, C.⁵ Saxena, R.^{1,6,7} Scheer, F. A.^{2,3,6} Johnson-Akeju, O.¹ Hu, K.^{2,3}

¹Department of Anesthesia, Critical Care and Pain Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, MA, ²Division of Sleep and Circadian Disorders, Brigham and Women's Hospital, Boston, MA, ³Division of Sleep Medicine, Harvard Medical School, Boston, MA, ⁴Center for Pain Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, MA, ⁵Department of Integrative Physiology, University of Colorado, Boulder, CO, ⁶Broad Institute of MIT and Harvard, Cambridge, MA, ⁷Center for Genomic Medicine, Massachusetts General Hospital, Boston, MA.

Introduction: In the current epidemic of opioid-related deaths, and widespread use of opioids to treat chronic pain, there is a pressing need to understand the underlying risk factors that contribute to such devastating conditions. Shiftwork has been associated with adverse health outcomes. We tested whether shiftwork during middle age is linked to the development of chronic pain and opioid misuse. Methods: We studied 116,474 participants in active employment between 2006–2010 (mean age 57 ± 8 ; range 37-71) from the UK Biobank, who have been followed for up to 10 years until 2017. We included participants who were free from all forms of selfreported pain, and were not taking opioid medications at baseline. Chronic pain and opioid use disorder diagnoses were determined using hospitalization records and diagnostic coding from ICD-10. Multivariate logistic regression models were performed to examine the associations of shiftwork status (yes/no) and nightshift frequency (none/occasional/permanent) and with incident chronic pain and/or opioid use disorder during follow-up. Models were adjusted for demographics, education, Townsend deprivation index, major confounders (BMI, diabetes, bone fractures/injuries, operations, peripheral vascular disease, joint/inflammatory diseases, cancer, standing/manual labor at work) and covariates (smoking, alcohol, high cholesterol, depression/anxiety, and cardiovascular diseases).

Results: In total, 190 (1.6/1,000) developed chronic pain or opioid use disorders. Shiftworkers (n=17,673) saw a 1.5-fold increased risk (OR 1.56, 95% CI: 1.08–2.24, p=0.01) relative to day workers. Within shiftworkers, those who reported occasional nightshift work (n=3,966) were most vulnerable (OR 1.57, 95% CI: 1.06–2.34, p=0.02). Results remained similar after adjusting for baseline sleep duration, chronotype and insomnia.

Conclusion: Shiftwork, and in particular rotating nightshift work is associated with increased risk for developing chronic pain and opioid use disorders. Replication is required to confirm the findings and to examine underlying mechanisms.

Support: This work was supported by NIH grants T32GM007592, RF1AG064312, and RF1AG059867.

0260

ASSOCIATION BETWEEN FREE-LIVING PHYSICAL ACTIVITY AND SLEEP IN ICELANDIC ADOLESCENTS

Rognvaldsdottir, V.¹ Johannsson, E.¹ Soffia, H. M.¹ Stefansdottir, R. S.¹ Arngrimsson, S. A.¹ Cheng, K. Y.² Brychta, R.² Gudmundsdottir, S. L.¹ ¹University of Iceland, Reykjavik, ICELAND, ²National Institutes of Health, Bethesda, MD.

Introduction: Sleep and physical activity are both important to health, but the demands of our modern schedule often require individuals to choose one over the other. In adolescents, the association between objectively measured sleep and physical activity is not well established in the literature. The aim of current study was to assess associations between free-living and physical activity and sleep among 15-year-old adolescents.

Methods: Free-living physical activity and sleep were assessed with wrist-worn accelerometers, sleep diary, and questionnaires during a 7-day period including school days and non-school days in 270 (161 girls) adolescents (mean age 15.8±0.3y) in Reykjavik, Iceland. Linear regression analysis was used to explore the associations between objectively measured physical activity and sleep. T-test was used to determine if there is a significant difference in objectively measured sleep between those who reported sports or exercising <6 versus ≥ 6 h/week. **Results:** Weekly mean physical activity (2040±466 counts/min of wear/day) was negatively associated with total sleep time $(6.6 \pm 0.64 \text{ h/night})$ ($\beta \pm SE = -3.5 \pm 0.7$, p<0.001). However, physical activity was also negatively associated with minutes of wake after sleep onset on non-school days (p=0.047) and standard deviation (i.e. night-to-night variability) of total sleep time over the week (p=0.028). Subjects who reported exercising ≥ 6 h/week (n=116) had lower night-to-night variability in bedtime (41.2±27.9 min) than those who did not (49.8 ± 37.5 min), p=0.033.

Conclusion: The negative association between physical activity and sleep duration suggests that in more active individuals' physical activity may be displacing sleep. However, greater physical activity is also associated with fewer minutes of awakening and a less variable sleep schedule, indicating better sleep quality. These findings suggest that physical activity is important for good sleep quality, but students should more closely consider sleep guidelines when designing an exercise schedule. Future studies should test how change in sleep patterns might influence physical activity. **Support:** Icelandic Centre for Research, National Institute of Diabetes and Digestive and Kidney Diseases.

0261

A RANDOMIZED TRIAL ON THE EFFECTS OF STANDARD AND FLEXIBLE DUTY-HOUR RULES ON INTERN SLEEP AND ALERTNESS

Dinges, D. F.¹ Asch, D. A.¹ Shea, J. A.¹ Bellini, L. M.¹ Carlin, M.¹ Malone, S. K.¹ Desai, S. V.² Sternberg, A. L.² Tonascia, J.² Katz, J. T.³ Silber, J. H.⁴ Volpp, K. G.¹ Mott, C. G.⁵ Mollicone, D. J.⁵ Basner, M.¹ ¹Perelman School of Medicine University of Pennsylvania, Philadelphia, PA, ²John's Hopkins University, Baltimore, MD, ³Brigham and Women's Hospital, Boston, MA, ⁴Children's Hospital of Philadelphia, Philadelphia, PA, ⁵Pulsar Informatics, Inc, Philadelphia, PA.

Introduction: Duty hour regulations affect resident sleep, education, and patient care in complex ways. We performed a national clusterrandomized trial (iCOMPARE) in 63 internal medicine residency programs comparing the effects of the 2011 duty-hour standards to a more flexible set of duty hour rules characterized by maintaining an 80-hour workweek but without limits on shift length or mandatory time off between shifts, relative to patient mortality, intern educational outcomes, and intern sleep and alertness.

Methods: In the sleep and alertness sub-study, sleep duration and morning sleepiness and alertness were assessed with actigraphy, the Karolinska Sleepiness Scale, and a 3-minute Psychomotor Vigilance Test (PVT-B) for 14 days in 193 interns from 6 standard programs and 205 interns from 6 flexible programs.

Results: During the 14-day study periods, interns in standard and flexible programs averaged 7.03h sleep/24h (95% confidence interval [CI] 6.78h, 7.27h) and 6.85h sleep/24h (95% CI 6.61h, 7.10h), respectively. Sleep duration (difference between arms of -0.17h/24h; 1-sided lower 95% confidence limit -0.45h; NIM -0.5h; P=0.02 for noninferiority) and KSS sleepiness (difference 0.12 points; 1-sided upper 95% confidence limit 0.31 points; NIM 1 point; P<0.001)

were noninferior in flexible versus standard programs. We could not establish noninferiority for PVT-B alertness (difference -0.3 lapses; 1-sided upper 95% confidence limit 1.6 lapses; NIM 1 lapse; P=0.10). Based on analyses by shift type, sleep duration was 1.77h shorter on days when interns in flexible programs finished an overnight shift relative to a regular day shift (p<.001), with significant decreases in subjective and objective alertness, and frequent reports of excessive sleepiness, especially between 12am and 6am.

Conclusion: There were no signs of relevant chronic sleep loss across shifts in interns in flexible programs relative to their standard program counterparts. Interns were able to compensate for the sleep lost during extended overnight shifts by increasing sleep duration on nights prior to day shifts, night shifts, and days off. Increased sleepiness and reduced alertness of interns following extended overnight shifts need to be mitigated and suggest a role for fatigue-risk management programs.

Support: Supported by NHLBI grants U01HL125388 and U01HL126088 and grants from the ACGME.

0262

AN EXPLORATORY EXAMINATION OF THE RELATIONSHIP BETWEEN NEGATIVE AFFECT, STRESS AND EMOTIONAL REACTIVITY, MINDFULNESS, AND SLEEP INCOMPATIBLE BEHAVIORS

Nagy, S. Pickett, S. M. Sosa, J. Garcell, A.

Florida State University College of Medicine, Tallahassee, FL.

Introduction: Stress has been identified as a barrier to engaging in positive health behaviors. Sleep interventions, including sleep hygiene recommendations, highlight stress management as an important treatment component. However, the relationship between negative emotion, stress management or emotion regulation, and positive sleep behaviors has largely been unexamined. Therefore, the current study, through secondary analyses, examined the relationships between negative affect, stress and emotional reactivity and sleep incompatible behaviors. Lastly, the indirect effect that dispositional mindfulness, as a possible self-regulatory mechanism, may have on the relationship was also examined. It was hypothesized that greater levels of stress, emotional reactivity, and negative arousal along with lower levels of dispositional mindfulness would predict higher engagement in sleep incompatible behaviors.

Methods: Participants (n=308) identified mostly as female (55.8%) and White/Caucasian (83.2%) and with an average age of 36.76 (SD = 12.20). Participants completed the Perceived Stress and Reactivity Scale (PSRS), the Positive and Negative Affect Scale (PANAS), the Sleep Behaviors Self-Rating Scale, and the Five Facet Mindfulness Questionnaire (FFMQ). A multiple regression analysis was conducted using the relevant subscales from the PSRS, PANAS, and FFMQ to predict the occurrence of behaviors incompatible with healthy sleep.

Results: The hypothesis was partially supported. Results indicated that the model significantly predicted sleep incompatible behavior (R^2 = .108, F(9, 299) = 4.042, p < .001), with only negative affect (β = .163, t(299) = 2.555, p = .011) and nonreactivity (β = -.219, t(299) = -2.484, p = .014) remaining significant when all variables were entered in the model.

Conclusion: The findings demonstrate that negative affect and reactivity are significant predictors of engagement in poor sleep hygiene practices. They also suggest that certain facets of dispositional mindfulness has an indirect relationship with sleep incompatible behaviors. The results may contribute to the development of sleep health interventions and highlight the need for future research. **Support:** N/A

0263

LATER BEDTIME IS ASSOCIATED WITH DIFFERENCES IN PREFRONTAL GRAY MATTER VOLUME AND EXECUTIVE FUNCTION DEFICITS

Taylor, E. Grandner, M. A. Killgore, W. D. University of Arizona, Tucson, AZ.

Introduction: Evening chronotypes tend to have reduced white matter integrity within their axonal tracts and reduced functional connectivity compared to morning types, a pattern that has been attributed to chronic "social jet lag" and its attendant disrupted sleep patterns. It is not clear whether brain differences are due to chronotype, total sleep time (TST), or whether they are associated with actual bedtime exclusive of these other factors. Here we examined morphometric gray matter volume (GMV) and its correlation with bedtime, after controlling for confounding factors.

Methods: Forty-five healthy adults (22 female; aged 20 to 43), completed the Horne-Ostberg Morningness-Eveningness Questionnaire (MEQ) and the Tower of London (TOL), an executive function task of planning and sequencing. Participants wore an actigraph for 7 days and completed structural MRI imaging during the period of actigraphy. Morphometric GMV data were processed using standard procedures in SPM12. After controlling for age, sex, and intracranial volume, actigraphic bedtime was entered into a regression analysis to predict orbitofrontal GMV. Regional GMV was extracted and correlated with TOL performance. Effects of TST and MEQ were also examined during regression.

Results: Later bedtime was associated with increased GMV in the left superior orbitofrontal gyrus (p=.044, FWE corrected). This association remained significant after controlling for TST and MEQ. Moreover, greater GMV in this cluster was associated with poorer planning and sequencing on TOL in terms of excess moves (r=.357, p=.017), and faster response times (r=-.356, p=.018), suggesting impulsivity.

Conclusion: Later bedtime was associated with greater GMV of the orbitofrontal cortex, even after controlling for TST and chronotype, and this pattern was associated with greater impulsiveness and poorer planning. The causal direction of the relationship between bedtime and executive deficits remains unclear and will require further research. However, it is clear that sleep patterns, brain structure, and behavior are intimately related.

Support: Defense Advanced Research Projects Agency Young Faculty Award: DARPA-12-12-11-YFA11-FP-029

XI. Sleep Deprivation, Loss and Disruption

0264

THE EFFECTS OF PREVIOUS NIGHT'S SLEEP, 12-HOUR SHIFT, AND WORKLOAD ON SLEEPINESS IN EMERGENCY MEDICAL SERVICE PROVIDERS

Pavuluri, H. Schmidt, W. M. Amalean, A. Fowler, L. University of South Carolina School of Medicine Greenville, Greenville, SC.

Introduction: Shift-work has been shown to be detrimental to workers in many ways, including having negative effects on sleep. Many factors related to shift-work can contribute to these negative effects, including increased workload and less sleep the previous night. The effects of shift-work on sleep have been studied extensively in nurses and physicians, but this research is lacking in paramedics and emergency medical technicians (EMTs). This study assessed the effects of previous night's sleep, day- versus night-shift, and workload on sleepiness in paramedics and EMTs. **Methods:** Thirty-three EMTs and paramedics were tested before and after their 12-hour (either day- or night-) shift. Testing consisted of questions about previous night's sleep, pupillometry to assess pupillary response for physiological sleepiness, and the Stanford Sleepiness Scale for subjective sleepiness. The number of calls made per shift was used to quantify workload/call volume.

Results: An analysis of variance assessed the effect of shift (day/ night), previous night's sleep, and workload on post-shift sleepiness. Pupillometry demonstrated that participants were sleepier after 12-hour shifts, but those who slept 8–9 hours the night before were less affected. Pupillary response indicated higher levels of sleepiness following the night-shift compared to the day-shift. This was contrary to perceived sleepiness, which was higher after the day-shift than the night-shift. Higher call volume resulted in a sleepier physiological response, and this effect was shown to be dependent upon the shift.

Conclusion: Performing shift-work results in increased sleepiness in workers, especially after a night shift. However, physiological sleepiness does not always correspond to perceived sleepiness. In addition, increased workload results in increased sleepiness, especially during the day shift. This highlights the importance of obtaining 8–9 hours of sleep before a 12-hour shift to protect against the effects of sleepiness on patient care and EMS workers themselves.

Support: This was funded in part by a UofSC Provost Grant.

0265

CORTISOL AND C-REACTIVE PROTEIN FAIL TO PREDICT INDIVIDUAL DIFFERENCES IN NEUROBEHAVIORAL PERFORMANCE RESPONSES TO TOTAL SLEEP DEPRIVATION AND PSYCHOLOGICAL STRESS

Goel, N.¹ Yamazaki, E. M.¹ MacMullen, L. E.² Ecker, A. J.² ¹Biological Rhythms Research Laboratory, Department of Psychiatry and Behavioral Sciences, Rush University Medical Center, Chicago, IL, ²Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA.

Introduction: Individuals show marked differential vulnerability in neurobehavioral deficits from psychosocial stress and sleep deprivation. Although changes in salivary cortisol and C-reactive protein (CRP) typically occur across total sleep deprivation (TSD) and recovery sleep, whether these biological markers during fully rested conditions predict individual differences in cognitive performance during TSD and stress remains unknown.

Methods: Thirty-one healthy adults (ages 27–53; mean \pm SD, 35.4 \pm 7.1y; 14 females) participated in a five-day experiment consisting of

two 8h time-in-bed (TIB) baseline nights, followed by 39h TSD, and two 8h-10h TIB recovery nights. A modified Trier Social Stress Test (TSST) was conducted on the day of TSD to induce psychological stress. Salivary cortisol and CRP from blood were obtained at six time points during the study (pre-study, baseline, during TSD, during TSD after the TSST, after recovery, and post-study). A median split of TSD performance [total lapses (>500 ms response time) and errors] on the 10-minute Psychomotor Vigilance Test (PVT) defined cognitively resilient (n=15) and cognitively vulnerable (n=16) groups. Repeated measures ANOVA and post-hoc comparisons corrected for multiple testing, examined cortisol and CRP across time points between groups. Results: In both cognitively resilient and vulnerable individuals, cortisol increased with TSD compared to baseline in the morning and decreased with TSD + psychological stress in the afternoon compared to TSD alone. By contrast, there were no significant changes in CRP levels throughout the experiment. In addition, there were no significant time*group interactions in cortisol or CRP levels.

Conclusion: Salivary cortisol increased with TSD compared to baseline and showed a time-of-day effect with stress during TSD. Notably, cortisol and CRP did not differ between cognitively resilient and vulnerable individuals across TSD, psychological stress or recovery sleep and thus are not reliable biomarkers for predicting performance under these conditions.

Support: NASA NNX14AN49G.

0266

A BRIEF NAP DURING A PERIOD OF SLEEP DEPRIVATION DOES NOT MITIGATE COGNITIVE DEFICITS

Stepan, M. E. Altmann, E. M. Fenn, K. M. Michigan State University, Lansing, MI.

Introduction: Sleep deprivation consistently impairs vigilant attention and placekeeping, which is the ability to maintain place in a sequence of steps without skipping or repeating steps. Placekeeping is a broadly important component of higher-order cognition. Previously, we found that caffeine benefitted vigilant attention but had no effect on placekeeping for most individuals. Here, we investigated the extent to which another intervention, brief naps, mitigated deficits in vigilant attention and placekeeping during a period of sleep deprivation.

Methods: In the evening, participants completed assessments of placekeeping and vigilant attention, the UNRAVEL task and Psychomotor Vigilance Task (PVT), respectively. Participants were then randomly assigned to either stay awake in the laboratory overnight (Sleep-deprived) or sleep at home (Rested). Sleep-deprived participants were also randomly given a 0, 30, or 60 minute nap opportunity. During the naps, participants were setup with partial polysomnography. In the morning, Rested participants returned and everyone completed UNRAVEL and PVT again.

Results: Sleep deprivation increased placekeeping errors, particularly following interrupted performance, and increased attentional lapses. A brief nap opportunity did not mitigate placekeeping or vigilant attention deficits. Polysomnography data showed that total sleep time was negatively related to placekeeping errors following an interruption; participants who slept more made fewer post-interruption errors. Slow wave sleep (SWS) was negatively related to attentional lapses and placekeeping errors on non-interruption trials; participants who obtained more SWS made fewer lapses and non-interruption errors. Sleep latency was also negatively related to attentional lapses, such that participants who fell asleep quickly, an indication of greater sleepiness, had more attentional lapses.

Conclusion: A brief nap during a period of sleep deprivation is not a viable intervention and longer naps may be required before

observable performance benefits emerge. However, specific aspects of sleep architecture were related to performance on the two tasks, suggesting domain-specific deficits due to sleep deprivation. **Support:** Funding received by the Office of Naval Research

N00014-16-1-2841.

0267

SCHOOL NIGHT SLEEP DURATION EFFECT ON RISK FOR IN-AND-OUT OF SCHOOL SUSPENSIONS: AN INVESTIGATION IN A MIDDLE-SCHOOL AGED SAMPLE

Cook, J. D.¹ Peppard, P. E.² Blair, E. E.³ Tran, K. M.¹ Plante, D. T.¹ ¹University of Wisconsin School of Medicine and Public Health, Department of Psychiatry, Madison, WI, ²University of Wisconsin School of Medicine and Public Health, Department of Population Health Sciences, Madison, WI, ³University of Wisconsin-Whitewater, Department of Educational Foundationscat, Whitewater, WY.

Introduction: Sleep plays an important role in adolescent education and development. Sleep impacts student school attendance, academic performance, and daytime behaviors. There has been limited investigation into the impact on sleep duration (SD) on school suspension risk. Given the growing public health and policy focus on altering school start times to increase SD, this study assessed SD association with school suspension risk using a middle-school aged sample from the Madison (Wisconsin) Metropolitan School District (MMSD), prior to implementation of a planned district-wide delay in middle school start times. Methods: 4,175 middle-school aged students from 12 MMSD schools completed a sleep survey, which included school-night SD (SNSD). Self-reported SNSD between 4-and-12 hours served as criterion for inclusion in final sample. Mixed effects modeling was employed with students nested within school. Logistic regression determined SNSD association with in-school (ISS) and out-of-school (OSS) suspensions. ISS and OSS were dichotomized (No ISS/OSS = 0; nonzero ISS/OSS = 1) to serve as outcome variables. Full model covariates included age, sex, race, circadian preference, parent educational level, homelessness, free and reduced lunch, and special education status.

Results: Final sample included 3,860 students. Shorter SNSD associated with greater likelihood of OSS [OR = 0.83, 95% CI (-0.28, -0.09), $X^2 = 16.1$, p < 0.0001], but not ISS [OR = 0.97, 95% CI (-0.14, -0.070), $X^2 = 0.44$, p = 0.51]. Significance between SNSD and OSS was maintained in the full model [OR = 0.84, 95% CI (-0.27, -0.08), $X^2 = 13.2$, p = 0.0003]. Each additional hour of sleep associated with 16% lower risk of OSS.

Conclusion: These results suggest that students with shorter SD are at increased risk for OSS, which further highlights the potential deleterious impact of short SD on adolescent educational experience. **Support:** This research was generously supported by a grant from the Madison Education Partnership (MEP).

0268

DEVELOPMENT OF AN INTEGRATED MODEL OF SLEEP DEPRIVATION IN ADOLESCENCE

Kwon, M. Park, E. Livingston, J. A. Dean, G. E. Suzanne, D. S. School of Nursing, University at Buffalo, SUNY, Buffalo, NY.

Introduction: Sleep deprivation is a consistently and widely concerning problem among adolescents. Although a few models have been proposed to explain the relationships and pathways through which factors influence sleep in adolescents, there are lack of theoretical models that apply both biological and behavioral factors that contribute to sleep deprivation across the trajectory of adolescent development. The current study proposes an integrated model that draws upon constructs from several influential theories with an aim to re-conceptualize factors associated with sleep deprivation as a chronic and cyclic problem that emerges from biological and behavioral changes in youth.

Methods: The Two Process Model of Sleep Regulation, Spielman's 3p model, the theory of planned behavior, dual systems model, and sleep health framework are used to develop an integrated model of factors that lead to sleep deprivation in adolescents.

Results: The resulting integrated model highlights the importance of adolescent's inherent nature of delayed sleep phase at pubertal onset (*two process model*); increased reward-seeking that precedes the structural maturation of their cognitive control and emotions (*dual systems model*); and their attitudes/perceptions towards sleep (*theory of planned behavior*), which is often geared toward not prioritizing sleep. *Sleep health framework* adequately frames the pattern of sleep-wakefulness in adolescents using a multidimensional approach of sleep. Moreover, the new model presents contextual factors (*Spielman's 3p model*) and the way that these constructs interact in order to maintain a vicious cycle of insufficient sleep which leads to chronic sleep deprivation.

Conclusion: The current model portrays a wide-ranging view of mechanisms underlying sleep deprivation among adolescence by integrating both biological and behavioral aspects. The model is proposed to encourage researchers to explore these conceptual elements of biological and neurobiological changes, and behavioral problems in order to operationalize relevant measures to relate the concepts to sleep deprivation and subsequent health outcomes in adolescents. **Support:** None

0269

RESTRICTING SLEEP INCREASES TEENS' SEDENTARY BEHAVIOR WITHOUT IMPACTING MODERATE TO VIGOROUS PHYSICAL ACTIVITY

Krietsch, K. Duraccio, K. Zang, N. Beebe, D. Cincinnati Children's Hospital and Medical Center, Cincinnati, OH.

Introduction: Short sleep duration has been linked to obesity risk in adolescence. However, most research has focused on potential changes in appetite/intake, rather than physical activity or sedentary behaviors. It remains unknown if, in the daily lives of adolescents, sleep restriction increases moderate- to-vigorous physical activity (e.g., by providing more time for it) or discourages such activity (in favor of sedentary behaviors). This was the first study to use gold-standard objective measures to assess cause-and-effect relationships between sleep duration and the resulting activity levels of adolescents in the naturalistic environment.

Methods: N=104 healthy teens (ages 14–18) completed the 3-week within-subjects crossover sleep manipulation experiment during the summer. Following a 7-night a sleep stabilization week, teens were randomly assigned to 5 nights in Short Sleep (6.5hrs sleep opportunity) or Healthy Sleep (9.5hrs sleep opportunity). Following a 2-night "washout" period, they crossed over to the alternate sleep condition. Throughout the study, they wore validated waist-worn accelerometers to objectively measure sedentary and physical activity levels, and wrist-worn actigraphs to confirm adherence to their sleep condition.

Results: When in Short Sleep (vs. Healthy Sleep), teens on average slept 112 minutes less (p<.0001, d=1.72) per wrist actigraphy. Waistworn accelerometers reflected 99 more minutes in sedentary behavior (p<.0001, d=.97), and 16 more minutes in light physical activity (p=.002, d=.31) during short sleep. Teens did not differ in moderate-to-vigorous physical activity between conditions (p=.95, d=.03).

Conclusion: Among healthy adolescents, a realistic dose of sleep restriction did not affect moderate-to-vigorous physical activity

levels, but did sharply increase time in sedentary behavior. Given the negative weight and health consequences of sedentary behavior, these results have practical implications for obesity prevention/intervention efforts. They suggest that extending teen sleep may neither encourage nor discourage healthy physical activity, but may help curb unhealthy behaviors (e.g., sedentary behavior). **Support:** R01 HL120879

0270

SOMNOLENCE PROFILES IN MICE SUBMITTED TO ACUTE AND CHRONIC SLEEP DEPRIVATION

Fernandes, G. L.¹ Araujo, P.² Tufik, S.¹ Andersen, M.¹ ¹Universidade Federal de Sao Paulo, São Paulo, BRAZIL, ²Escola de Ciências Médicas da Santa Casa de São Paulo, Sao Paulo, BRAZIL.

Introduction: Sleepiness is a behavioral marker of homeostatic sleep regulation and is related to several negative outcomes with interindividual variation, which may amount to central sleep mechanisms. However, there is a lack of evidence linking progressive sleep need and sleepiness with factors of individual variability, which could be tested by acute and chronic sleep deprivation. Thus, the study objective was to investigate the development of sleepiness in sleep deprived mice. Methods: C57BL/6J male mice (n=340) were distributed in 5 sleep deprivation groups, 5 sleep rebound groups and 10 control groups. Animals underwent acute total sleep deprivation for 3, 6, 9 or 12 hours or chronic sleep deprivation for 6 hours for 5 consecutive days. Sleep rebound groups had the opportunity to sleep for 1, 2, 3, 4 hours after acute sleep deprivation or 24 hours after chronic sleep deprivation. During the protocol, sleep attempts were counted as a sleepiness index. After euthanasia, blood was collected for corticosterone assessment.

Results: Using the average group sleep attempts, it was possible to differentiate between sleepy (mean>group average) and resistant to sleepiness animals (mean<group average). Frequency of resistant mice was 65%, 56% and 53% for 3, 6, 9 and 12 hours of acute sleep deprivation, respectively, and 74% in chronic sleep deprivation. 52% of the sleepiness variance might be explained by individual variation during chronic sleep deprivation and 68% of sleepiness variance during acute sleep deprivation was attributed to extended wakefulness. A normal corticosterone zenith was observed at the start of the dark phase, independent of sleep deprivation.

Conclusion: Different degrees of sleepiness in sleep deprived mice were verified. Sleep deprivation per se was the main factor explaining sleepiness during acute sleep deprivation whereas in chronic deprivation individual variation was more relevant.

Support: This work was financially supported by Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP) (#2017/18455-5), Coordenação de Aperfeiçoamento de Pessoal Nível Superior (CAPES) - grant code 001, ConselhoNacional de Desenvolvimento Científico e Tecnológico (CNPq) (#169040/2017–8)and Associação Fundo de Incentivo à Pesquisa (AFIP).

0271

EFFECTS OF EXPERIMENTALLY SHORTENED SLEEP ON DIETARY OUTCOMES IN ADOLESCENTS

Duraccio, K. M. Krietsch, K. N. Beebe, D. W. Cincinnati Children's Hospital Medical Center, Cincinnati, OH.

Introduction: Adolescents who sleep less may consume more calories during the day; however, shortened sleep may have greater impact on the dietary quality of foods, particularly foods high in sugar content. This study examined the impact of shortened sleep on total caloric

intake, macronutrients of food (fats, proteins, carbohydrates), added sugars consumed, and glycemic load/index of foods consumed.

Methods: 110 adolescents (ages 14–17, M = 15.73(1.80); 63.6% female) underwent a within-subject counterbalanced experimental sleep manipulation, spending either 6.5 or 9.5 hours/night in bed for five nights. During each sleep period, adolescents completed three dietary recalls (reporting amounts and types of foods consumed over a 24-hour period) administered by dietary core study staff. We conducted a series of repeated-measure general linear models comparing averaged weekly dietary outcomes of interest by sleep condition. We also explored whether experimental order of the sleep conditions, family income, or adolescent gender, body mass index (BMI), or race moderated the main effects of sleep condition on dietary outcomes. Alpha was set at .05 for primary analyses and .01 for exploratory analyses.

Results: We observed a main effect of sleep on carbohydrates (p=.038) and added sugars (p=.009) consumed, as well as the glycemic index (p=.013) and glycemic load (p=.009) of foods consumed. We did not observe a main effect of sleep on total calories or total grams of fat or protein consumed. Exploratory analysis found no significant interactions of the moderators with sleep condition on dietary outcomes.

Conclusion: Adolescents are consuming more carbohydrates, added sugars, and foods higher in glycemic index and load when sleep restricted, compared to well-rested, despite eating comparable amounts of calories. Sleep restricted adolescents may be drawn to foods that provide quick releases of energy to counteract sleepiness experienced during the day.

Support: R01 HL120879

0272

EFFECTS OF GESTATIONAL SLEEP RESTRICTION ON MATERNAL BEHAVIOR IN RODENTS: A SYSTEMATIC REVIEW AND META-ANALYSIS

Pires, G. N.^{1,2} Baenninger, T.² Mello, V.² Andersen, M. L.¹ Tufik, S.¹ ¹Universidade Federal de São Paulo, São Paulo, BRAZIL, ²Santa Casa de Sao Paulo School of Medical Sciences, Sao Paulo, BRAZIL.

Introduction: Sleep restriction during pregnancy is related with postpartum depression in clinical settings. Preclinical studies have been conducted in rodent models of maternal behavior, intending to evaluate the mechanisms behind this relationship, but have raised inconsistent data so far. Our aims were to perform a preclinical meta-analysis, evaluating the actual effects or prenatal sleep restriction on maternal behavior in rodents.

Methods: A bibliographic search was conducted in Pubmed, Scopus, Web of Science, Psychinfo and Lilacs. Search strategy encompassed three domains: sleep restriction during pregnancy (as intervention), maternal behavior (as outcome) and experimentation animals (as population). Studies were first selected based on titles and abstracts, followed by full text analysis and data extraction. Individual effect size for each articles was calculated using standardized mean difference and meta-analysis was conducted using a DerSimonian and Laird random effects model.

Results: 144 articles were included in our initial data screening. Sample was reduced to six records after screening. A meta-analysis was performed, including data from two maternal behavior tests (pup retrieval test and ethogram-based analyses). A total of 115 animals were included. Meta-analysis showed that sleep restriction during pregnancy have no significant effects on maternal behavior. **Conclusion:** Clinical studies and meta-analysis have shown that sleep restriction and disorders during pregnancy increase risk for postpartum depression. However, preclinical studies fail to corroborate these results, as sleep restriction during

pregnancy does not reduce maternal behavior in rodents. The presence of negative effects in women and the maintenance of normal maternal behavior levels in rodents under comparable intervention demonstrate that other factors might mediate this relationship (among which, sociocultural factors might play a role). The maintenance of maternal behavior seems to be an adaptive behavior, assuring the suitability and survival of the litter, even in face of environmental stresses. **Support:** AFIP, FAVC, CNPq, FAPESP

0273

RESIDUAL SLEEP DIFFICULTIES DURING RESET OPERATIONS PREDICT GREATER POST-DEPLOYMENT MENTAL HEALTH DIFFICULTIES IN U.S. SOLDIERS: A CROSS-LAGGED ANALYSIS

So, C. J.¹ Alfano, C. A.¹ Riviere, L. A.² Quartana, P. J.² ¹University of Houston, Houston, TX, ²Walter Reed Army Institute of Research, Silver Spring, MD.

Introduction: Military service is associated with a number of occupational stressors, including non-conducive sleeping environments, shift schedules, and extended deployments overseas. Service members who undergo combat deployments are at increased risk for mental health and sleep difficulties. Bidirectional associations between sleep and mental health difficulties are routinely observed, but the directional association of these difficulties from one deployment to the next has not been addressed. The purpose of this study was to examine whether residual sleep problems or mental health difficulties after a 12-month period of reset operations following an initial deployment were associated with changes in sleep and mental health following a subsequent deployment.

Methods: Data from 74 U.S. Soldiers were case-matched across three time points. Participants were assessed 6 months (T1) and 12 months (T2) following an initial deployment. Participants were then assessed 3 months (T3) following a subsequent deployment. Symptoms of PTSD, anxiety, depression, and sleep difficulties were assessed at all three time points.

Results: Cross-lagged hierarchical regression models revealed that residual sleep difficulties across the time points uniquely predicted later changes in PTSD and anxiety symptoms, but not depressive symptoms, following a subsequent deployment. Conversely, residual mental health difficulties were not unique predictors of later changes in sleep difficulties.

Conclusion: These findings suggest that higher levels of residual sleep difficulties 12 months following a prior deployment are associated with larger increases in mental health problems following a subsequent deployment. Moreover, and importantly, the converse association was not supported. Residual mental health difficulties prior to deployment were not associated with changes in sleep difficulties. These data provide a viable target for intervention during reset operations to mitigate mental health difficulties associated with combat deployments. They might also help inform return-to-duty decisions. **Support:** N/A.

0274

ASSOCIATIONS OF TNFA GENE POLYMORPHISM WITH RESILIENCE TO SLEEP DEPRIVATION AND CAFFEINE SENSITIVITY

Skeiky, L.^{1,2} Hansen, D. A.^{1,2} Satterfield, B. C.^{1,2,3} Petrovick, M.⁴ Balkin, T. J.⁵ Capaldi, V. F.⁵ Ratcliffe, R. H.⁵ Brager, A. J.⁵ Van Dongen, H.^{1,2} ¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ³Department of Psychiatry, College of Medicine, University of Arizona, Tucson, AZ,
 ⁴Group 49 - Biological and Chemical Technologies, MIT Lincoln Laboratories, Lexington, MA, ⁵Sleep Research Center, Behavioral Biology Branch, Center for Military Psychiatry and Neuroscience, Walter Reed Army Institute of Research, Silver Spring, MD.

Introduction: Sleep deprivation degrades the fidelity of human brain information processing, leading to cognitive impairment. Carriers of the A allele of a single nucleotide polymorphism of the TNF α gene (G308A, rs1800629) have been found to be resilient to cognitive impairment due to sleep deprivation as compared to individuals homozygous for the G allele. Caffeine mitigates the cognitive impairment associated with sleep deprivation. We investigated whether the effects of caffeine and TNF α genotype interact.

Methods: In an 18-day, controlled, in-laboratory study, 12 healthy adults (age 27.4 \pm 6.9; 6 females) underwent three sessions of 48-hour total sleep deprivation (TSD), with each TSD session preceded and followed by three nights of baseline and/or recovery sleep (10 hours time in bed). In randomized, counterbalanced, double-blind, placebo-controlled fashion, during each TSD session a specific dose of caffeine (0, 200, or 300 mg) was administered four times at 12-hour intervals. Vigilant attention was measured every 2 hours during each TSD session with a psychomotor vigilance test (PVT), for which the log of the signal-to-noise ratio (LSNR) derived from the RT distribution was determined as a measure of the fidelity of information processing. Each subject's TNF α genotype was assessed from a blood sample.

Results: Subjects homozygous for the TNF α G allele showed greater PVT impairment during sleep deprivation in the 0 mg caffeine (i.e., placebo) condition as compared to carriers of the A allele and as compared to the 200 and 300 mg caffeine conditions (mixed-effects ANOVA, genotype by dose interaction: $F_{2,566}$ =5.23, p=0.005). There was no appreciable caffeine-related difference in performance for carriers of the A allele, who were relatively resilient to TSD regardless of caffeine dose.

Conclusion: These results suggest non-additive, interacting effects of $TNF\alpha$ genotype and caffeine and a potentially shared mechanism of action with regard to the fidelity of information processing during sleep deprivation.

Support: This research was supported by ONR. AJB, TJB, VFC, RHR were supported by DoD MOMRP-USAMRDC. The views expressed here are those of the authors and do not represent the official policy or position of the DoD.

0275

EXPOSURE TO EXPERIMENTALLY INDUCED SLEEP DISTURBANCE AFFECTS THE INFLAMMATORY RESOLUTION PATHWAYS IN HEALTHY HUMANS

Engert, L. C.^{1,2} Dubourdeau, M.³ Mullington, J. M.^{1,2} Haack, M.^{1,2} ¹Department of Neurology, Beth Israel Deaconess Medical Center, Boston, MA, ²Harvard Medical School, Boston, MA, ³Ambiotis SAS, Toulouse, FRANCE.

Introduction: Sleep disturbances are assumed to impair health through induction of low-grade systemic inflammation. Experimental studies have shown that such inflammatory upregulation does not normalize even after a couple of nights of recovery sleep. We hypothesized that sleep disturbances do not only affect inflammatory pathways, but also the recently detected inflammatory resolution pathways, which actively terminate

inflammation. Mediators of inflammatory resolution mainly derive from omega-3 fatty acids converted to specialized pro-resolving mediators (SPMs), such as resolvins. We investigated SPMs in healthy humans exposed to a novel model of experimental insomnia.

Methods: Twenty-four individuals (age 18-42 years, 12 women) participated in a study consisting of two 19-day in-hospital protocols (insomnia/control). After three nights of baseline sleep (8h/night, 23:00-07:00), participants in the experimental insomnia condition were exposed to three cycles of three nights of disturbed sleep (delayed sleep-onset, hourly sleep disruption, advanced sleep-offset) followed by one night of 8h-recovery sleep. The protocol ended with three additional nights of recovery sleep. In the control condition, participants had an uninterrupted sleep opportunity (8h/night) across the 19-day protocol. Blood samples were taken at 11:00 at baseline, during experimental insomnia exposure, and after recovery sleep. Several SPMs were measured in plasma using liquid chromatography-tandem mass spectrometry (LC-MS/MS). Data were analyzed using linear mixed models. Results: Exposure to experimental insomnia affected several SPMs compared to control sleep, including a decrease of resolvin D4 and E2 concentrations, which was still evident after the third recovery night (p<.05).

Conclusion: This is the first investigation on the effects of experimentally induced sleep disturbance on inflammatory resolution pathways. The results support that SPMs, particularly resolvin D4 and E2, are decreased by sleep disturbances, and do not normalize after a couple of nights of recovery sleep. Targeting these pathways by pharmacologically increasing SPMs may help to limit inflammatory consequences of sleep disturbances.

Support: NIH/NINDS R01-NS091177; NIH/National Center for Research Resources UL1-RR02758 and M01-RR01032 to the Harvard Clinical and Translational Science Center.

0276

DOES LOSING SLEEP UNLEASH ANGER?

Krizan, Z.¹ Miller, A.¹ Hisler, G.³

¹Iowa State University, Ames, IA, ²Iowa State University, Ames, IA, ³University of Pittsburgh School of Medicine, Pittsburgh, PA.

Introduction: Sleeping is understood as essential to affective function, yet little is known about how sleep shapes more specific and contextualized emotional responses besides anxiety and depression, such as anger. Anger itself involves arousal and can disrupt sleep. To examine the causal role of sleep in anger, a daily-diary study and an experimental study tested whether shortened sleep amplifies angry feelings, while exploring mediating mechanisms of this influence.

Methods: The daily-diary study (N = 202) collected daily reports of last-night's sleep, daily stressors, and state anger across one month from college students, examining sleep and anger within everyday life. The experimental laboratory study (N = 147 community residents) examined changes in anger experienced during aversive noise following random assignment to either at-home sleep restriction (by about 5 hours across 2 nights), or to individuals' regular schedule.

Results: In the daily-diary study, individuals experienced more anger on days following less sleep than their usual, with half of this effect attributed to the increased frequency of stressors experienced on such days, and somewhat independently from the effect of sleep duration on negative affect more generally. In the experimental study, well-slept individuals adapted to noise and reported less anger and negative affect after 2 days. In contrast, sleep-restricted individuals exhibited higher and increased anger responses. The impact of sleep restriction on anger held even after accounting for negative emotions more generally. Subjective sleepiness accounted for most of the experimental effect of sleep loss on anger.

Conclusion: Together, these results provide compelling evidence that lost sleep amplifies anger in both the laboratory and everyday life, while also pointing to short-term (subjective sleepiness) and mid-term (stress) mediators of these influences. The findings also point to the value of examining specific emotional reactions (and their regulation) in the context of sleep disruption, alongside affect more broadly. **Support:** N/A

0277

SLEEP RESTRICTION DOES NOT ALTER THE TRANSCRIPTION OF ADIPOSE TISSUE INSULIN-SIGNALING REGULATORS

Singh, P.¹ Covassin, N.² Bukartyk, J.² Davison, D.² Svatikova, A.² St Louis, E. K.² Somers, V. K.² ¹Pennington Biomedical Research Center, Baton Rouge, LA,

²Mayo Clinic, Rochester, MN.

Introduction: Voluntary sleep curtailment decreases insulin sensitivity. However, molecular mechanisms underlying impaired insulin signaling are not completely understood. To gain molecular insights, we examined the transcription of known cellular insulinsignaling regulators in adipose tissue obtained from subjects undergoing experimental sleep restriction.

Methods: Nineteen healthy subjects (males: 11; age: 19 - 36 years; BMI: $24.5 \pm 3.6 \text{ kg/m}^2$) underwent a normal (9 hours/night) and a restricted sleep (4 hours/night) sequence in a random order. Each inpatient stay included a screen visit followed by 4 days of acclimation and 9 days of experimental phase consisting of a controlled sleep opportunity. Eucaloric diet was provided to ensure weight maintenance. Abdominal fat sample was obtained at the screen and end of the experimental phase. mRNA was quantified by RT-PCR. Fasting morning blood draws were obtained at the end of acclimation and experimental phases. Mixed models were used for analysis.

Results: mRNA expression did not differ in normal and restricted sleep condition for SOCS3 [changes in normal Vs. restricted sleep, 0.02 (0.11, -0.01) Vs. 0.01 (0.12, -0.07), p=0.33], PTEN [-0.22 (0.18, -0.39) Vs. -0.10 (0.21, -0.20), p=0.22], PTB1B [-0.003 (0.04, -0.07) Vs. -0.01 (0.06, -0.06), p=0.74] and Cav-1 [-2.45 (0.78, -8.39) Vs. -6.31 (1.68, -8.29), p=0.34]. Further, transcription of insulin-receptor also did not change [0.01 (0.07, -0.06) Vs. -0.03 (0.09, -0.07), p=0.92]. However, within group analysis show significant decreases in Cav-1 mRNA only during sleep restriction (normal: p=0.09; restricted: p=0.003). Importantly, restricting sleep duration was associated with lowering of insulin (0.6 \pm 1.9 Vs. -1.6 \pm 1.7 uIU/ml; p=0.003), no change in glucose (-1.5 \pm 3.8 Vs. -3.7 \pm 4.12 mg/dl, p=0.06) and improvement in HOMA-IR index (0.12 \pm 0.44 Vs -0.42 \pm 0.43, p=0.002).

Conclusion: Chronic sleep restriction does not alter the transcription of cell-signaling regulators in abdominal adipose tissue. Moreover, restricting sleep duration without increasing calorie intake did not seem to decrease insulin sensitivity as determined by HOMA-IR.

Support: NIH grants HL114676, Mayo Clinic CCaTS UL1 TR002377; AHA grant 13POST16420009 to NC

0278

CHANGES IN SLEEP ARCHITECTURE DURING LONG-DURATION SPACEFLIGHT

Piltch, O.¹ Flynn-Evans, E.² Stickgold, R.³

¹Harvard College, Cambridge, MA, ²NASA Ames Research Center, Mountain View, CA, ³Harvard Medical School, Boston, MA.

Introduction: Previous projects have shown that astronauts sleep significantly worse in mission than on Earth. However, it is unclear

how sleep architecture is influenced by microgravity. Such information could inform our understanding of the adaptive mechanisms NREM and REM sleep on Earth. We investigated how sleep architecture is affected during spaceflight relative to on Earth.

Methods: Sleep architecture was assessed using the Nightcap monitor before (pre-flight, n=113 nights), during (in-flight, n=68 night), and after (post-flight, n=61 nights) missions aboard the Mir space station for four cosmonauts and one astronaut. We compared hand-scored REM/NREM/wake staging in/post-flight to a pre-flight baseline using mixed-effects regression to account for subject variability. We also used mixed-effects modeling to assess changes over time in different phases of the mission.

Results: Participants averaged an hour less sleep in space (5.4 \pm 0.66) compared to pre-flight (6.6 \pm 0.70; p < .0001) and spent significantly more time awake in bed, leading to a 20.8% reduction in sleep efficiency. Sleep architecture was also affected by spaceflight: percentages of time in bed for NREM and REM decreased significantly by 9.9% and 26.6% respectively. REM latency nearly doubled during spaceflight to 88 ± 3 minutes. All metrics were stable across the in-flight phase, with the exception of an increase in sleep latency (β : 0.47; p = 0.0009) and a decrease in time in bed (β = 0.85; p < .0001). Conclusion: These data substantiate previous findings focused on sleep continuity in microgravity. A variety of metrics demonstrate worse sleep in space. NREM and REM time significantly decreased alongside an increase in wakefulness, but the relative proportion of these stages also changed significantly: REM sleep suffered more than NREM in spaceflight conditions. These longitudinal data add value to our nebulous understanding of how sleep functions in microgravity. Support: Mary Gordon Roberts Fellowship, NAS 9-19406, NIMH #MH-48,832, The MacArthur Foundation Mind-Body Network, and Healthdyne Technologies

0279

FATIGUE AND PAIN RESPONSES ACROSS REPEATED EXPOSURE TO EXPERIMENTALLY INDUCED SLEEP DISTURBANCE AND INTERMITTENT RECOVERY SLEEP: SEX DIFFERENCES

Goldstein, M. R.^{1,2} Simpson, N. S.³ Devine, J. K.⁴ Dang, R.¹ Connors, C.¹ Engert, L. C.^{1,2} Chatterton, B.¹ Scott-Sutherland, J.¹ Yang, H.^{1,2} Mullington, J. M.^{1,2} Haack, M.^{1,2}

¹Department of Neurology, Beth Israel Deaconess Medical Center, Boston, MA, ²Harvard Medical School, Boston, MA, ³Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Palo Alto, CA, ⁴Department of Operational Fatigue and Performance, Institutes for Behavioral Resources, Baltimore, MD.

Introduction: Sleep disturbances are more common in women than in men, as are many chronic pain disorders characterized by inflammation and fatigue. This study investigated sex differences in fatigue and pain responses to sleep disruption and whether such responses recover with uninterrupted sleep.

Methods: 24 healthy young individuals (12 women; ages 18–42 yrs) participated in a study consisting of two counterbalanced 19-day experimental in-hospital stays, separated by two months. Following 3 baseline nights, participants were exposed to 3 nights of sleep disruption (SD) involving delayed sleep onset, hourly awakenings, and early-morning awakenings without return to sleep, followed by 1 night of recovery sleep. This 4-day cycle was repeated three times and finished with 3 additional nights of recovery sleep. Total sleep opportunity on SD nights was 4 hrs, and on recovery/sleep control (SC) nights was 8 hrs. Light exposure, ambient temperature, food and fluid intake, and physical activity were controlled. Self-reported

fatigue and pain, pain sensitivity, and habituation were collected throughout. Data were analyzed with linear mixed models. **Results:** For women but not men, fatigue in response to SD recovered incompletely starting after the 2^{nd} sleep disruption-recovery cycle and remained elevated after the final 3 recovery nights in women (*p*<.05).

Additionally, women became more sensitive to pressure pain in response to SD (p<.001) with incomplete return to baseline after the final 3 recovery nights. Whereas men habituated to cold pain across SC and even more so across SD (p=.045 Day, p=.021), women did not habituate. **Conclusion:** These results indicate that incomplete recovery in both fatigue and pressure pain, alongside a lack of habituation to cold pain, in response to show discrution may explain the acompton of pain.

in response to sleep disruption may explain the common co-occurrence of insomnia, fatigue, and pain observed as more prevalent in women. **Support:** NIH/NINDS R01-NS091177; NIH/National Center for Research Resources UL1-RR02758 and M01-RR01032 to the Harvard Clinical and Translational Science Center.

0280

CHANGE IN SLEEP DEPTH ACROSS THE NIGHT AS A MEASURE OF SLEEP ADEQUACY

Schweitzer, P. K.¹ Griffin, K.¹ Younes, M.² Walsh, J. K.¹ ¹Sleep Medicine & Research Center, St. Luke's Hospital, Chesterfield, MO, ²Sleep Disorders Centre, University of Manitoba, Winnipeg, MB, CANADA.

Introduction: It is well known that sleep becomes lighter towards the end of the night reflecting the reduction in homeostatic sleep pressure. We hypothesized that more adequate nocturnal sleep (i.e. sufficient quantity and quality for the individual) would result in a greater reduction in sleep depth across the night and would be reflected in decreased next-day sleep tendency.

Methods: In a secondary analysis of data from a study in which sleep depth was altered by sleep restriction combined with either placebo or gaboxadol (a delta-promoting drug) we correlated change across the night in two measures of sleep depth with next-day Multiple Sleep Latency Test (MSLT) latencies. Forty-one healthy subjects underwent 8 consecutive sleep studies; two baseline, four sleep restriction (5 hours) and two recovery nights. MSLT was performed following each baseline night and the last two restriction nights. Sleep depth in the first and last hours of NREM sleep was determined by two

methods: 1) Log delta spectral power; 2) The odds-ratio-product (ORP), a recently introduced continuous measure of sleep depth. The difference between initial and final values was calculated (Δ Delta, Δ ORP). Post-restriction MSLT latency was correlated with baseline MSLT latency, Δ Delta, Δ ORP, log delta power and ORP in the last hour, lost total sleep time and lost REM time.

Results: $\Delta Delta$ was -0.27 ±0.13 and ΔORP was 0.17 ±0.13, both changes reflecting lightening of sleep across the night. In both univariate and multivariate analysis only baseline MSLT latency (p < 0.001) and ΔORP (p < 0.01) were significantly and positively correlated with post-restriction MSLT latency.

Conclusion: The reduction in sleep depth across the night as measured by ORP, but not by delta power, is significantly correlated with reduced objective sleepiness following sleep restriction. $\triangle ORP$ may be a useful index that reflects sleep adequacy during the night. **Support:** None

0281

SLEEP DEPRIVATION IMPAIRS COOPERATIVE BEHAVIOR SELECTIVELY: EVIDENCE FROM PRISONER'S AND CHICKEN DILEMMAS

Ma, N. Lin, Y. Mo, L. South China Normal University, Guangzhou, CHINA. **Introduction:** Cooperation, the cornerstone of human interaction, has attracted much attention since it was indispensable in the contemporary world. However, little research has been done on whether sleep deprivation altered human cooperative behavior. In the present work, we investigated cooperation and sleep deprivation directly, aiming to evaluate the influences of acute sleep deprivation on cooperation with two classical social dilemmas, the Prisoner's dilemma (PD) and the chicken dilemma (CD).

Methods: All participants (N=24) were required to come for the experiments twice, one time for normal sleep condition, the other time for sleep deprivation condition, with a counter-balanced sequence. In the following afternoon, participants completed the psychomotor vigilance task (PVT) and two social dilemmas tasks, as well as the Karolinska Sleepiness Scale (KSS), the Risk Orientation Questionnaire (ROQ) and the Positive and Negative Affect Schedule (PANAS).

Results: Our results demonstrated that sleep deprivation significantly impaired cooperative behaviors in the CD but not in the PD. In addition, this detrimental effect was not related with the alteration in the risk-seeking, objective alertness, subjective sleepiness, and mood.

Conclusion: The current findings revealed that sleep deprivation impairs human cooperative behaviors selectively. However, the underlying mechanism remains to further explore with neuroimaging studies and focus on the mediating role of general trust and sensitivity of loss.

Support: This research was supported by the National Natural Science Foundation of China (31500906), Guangdong Natural Science Funding - General Program (2019A1515012182).

0282

CORRELATION BETWEEN SLEEP DEPTH IN THE RIGHT AND LEFT CEREBRAL HEMISPHERES FOLLOWING SLEEP DEPRIVATION, RESTRICTION OR NOISE EXPOSURE

Younes, M.¹ Kuna, S. T.² Pack, A. I.² Schweitzer, P. K.³ Walsh, J. K.³ Smith, M. G.⁴ Basner, M.⁴ Aeschbach, D.⁵ ¹University of Manitoba, Winnipeg, MB, CANADA, ²Division of Sleep Medicine/Department of Medicine, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, ³Sleep Medicine & Research Center, St. Luke's Hospital,, Chesterfield, MO, ⁴Unit for Experimental Psychiatry, Division of Sleep and Chronobiology, Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania,, Philadelphia, PA, ⁵Dept. of Sleep and Human Factors Research, Institute of Aerospace Medicine, German Aerospace Center, GERMANY.

Introduction: The Odds-Ratio-Product (ORP) is a highly-validated continuous index of sleep depth (range 0=deep sleep; 2.5=full wakefulness). ORP values fluctuate within this range as sleep state changes between wake and different sleep stages. In healthy non-sleep deprived adults, intra-class correlation coefficient of concurrent right vs. left ORP values (R / L coefficient) is typically >0.80. In a recent study R / L coefficient was markedly reduced in many critically-ill patients and these patients failed to be weaned from mechanical ventilation. Given the high prevalence of sleep loss in such patients we hypothesized that reduction in R/L coefficient might result from sleep loss. This retrospective EEG analysis of data from 3 independent research studies investigated if R / L coefficient decreases in pure models of sleep deprivation, restriction or noise exposure during sleep in healthy subjects. Methods: Polysomnograms were obtained from three studies: A) 200 subjects who underwent 36 hours of total sleep deprivation; B) 21 subjects who underwent 4 consecutive nights of sleep restriction (5 hrs. / night); C) 72 subjects who were exposed to intermittent traffic noise events with maximum sound pressure levels ranging

from 45–65 dB(A) for 10 consecutive nights. For study A, R / L coefficient was calculated from pre- and post-deprivation sleep studies and the two values were compared. For study B, coefficient was calculated at baseline and in each restriction night. For study C, the coefficient was calculated in each of the 10 exposure nights and the slope of the change was calculated.

Results: In study A, the coefficient decreased from 0.82 ± 0.12 at baseline to 0.74 ± 0.16 after sleep deprivation (p < 0.0001). In study B, the coefficient decreased from 0.83 ± 0.11 at baseline to 0.75 ± 0.15 on the 4th restriction night (p < 0.01). In study C, coefficient decreased at a rate of 0.003 ± 0.001 per exposure night (p < 0.001). **Conclusion:** The correlation between sleep depth in the right and left hemispheres deteriorates following sleep deprivation, restriction.

tion or noise-induced sleep fragmentation.

Support: NIH P50 HL060287

0283

DIFFERENTIAL IMPACT OF CHRONOTYPE ON POSITIVE AND NEGATIVE AFFECT FOLLOWING SLEEP RESTRICTION

Cox, R. C. Olatunji, B. O. Vanderbilt University, Nashville, TN.

Introduction: Previous research indicates sleep deprivation significantly decreases positive affect, while negative affect is often unaffected (Reddy et al., 2017; Schwarz et al., 2019). However, it is unclear whether individual difference factors predict a differential emotional response to sleep loss. Though one study found chronotype predicts decreased positive affect following sleep loss in adolescents (Dagys et al., 2012), the effect of chronotype on emotional response to sleep loss in adults remains unknown.

Methods: The present study addressed this limitation by examining whether chronotype predicts changes in positive and negative affect following partial sleep restriction. Healthy sleeping adults (N=113; Insomnia Severity Index<8) completed the Morningness-Eveningness Questionnaire (Horne & Ostberg, 1976) on day 1, and were instructed to sleep normally on night 1. On the evening of day 2, participants completed the Positive and Negative Affect Schedule (PANAS; MacKinnon et al., 1999) to sample baseline daily emotions. On night 2, sleep was restricted to 4-8am. On day 3, participants completed the PANAS post-sleep restriction. Compliance was verified with actigraphy (n=22 excluded for noncompliance), and response to the sleep restriction procedure was checked with the Stanford Sleepiness Scale (Hoddes, Dement, & Zarcone, 1972; n=18 excluded for nonresponse).

Results: Results suggest a significant decrease in positive affect from pre-sleep restriction (M=15.63, SD=4.09) to post-sleep restriction (M=11.08, SD=3.93), t(71)=10.33, p<.01, and a trend decrease in negative affect from pre-sleep restriction (M=7.68, SD=3.62) to post-sleep restriction (M=6.90, SD=2.41), t(71)=1.60, p=07. Chronotype was significantly associated with post-sleep restriction negative affect, controlling for baseline negative affect (β =-.42, p<.01), such that eveningness was associated with increased negative affect following sleep restriction. In contrast, chronotype was unrelated to post-sleep restriction positive affect.

Conclusion: These findings indicate that evening types may be more likely to experience emotions implicated in anxiety-related disorders following sleep loss, such as fear and nervousness.

Support: This work was supported by the National Institute of Mental Health of the National Institutes of Health [F31MH113271] and a Graduate Student Summer Research Award from Vanderbilt University. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

0284

THE EFFECTS OF A CIRCADIAN-ALIGNED WATCHBILL SHIFT WORK SCHEDULE ON SLEEP QUALITY AND QUANTITY IN U.S. NAVY SUBMARINE PERSONNEL

*Dahlquist, D. T.^{1,2} Chinoy, E. D.^{1,2} Markwald, R. R.¹ Chabal, S. A.*³ ¹Naval Health Research Center, San Diego, CA, ²Leidos Inc., San Diego, CA, ³Naval Submarine Medical Research Laboratory, Groton, CT.

Introduction: Prior to 2014, the U.S. Navy Submarine Force operated on a non-circadian-aligned watchbill shift work schedule (18hour day-length) that resulted in insufficient sleep. For instance, prior research reported that submariners received insufficient sleep on this schedule, and modest sleep restrictions can adversely affect performance, alertness, and, ultimately, negatively impact mission outcomes. Thus, the present study characterized sleep patterns of submariners operating on a newer, circadian-aligned 24-hour day-length watchbill. Methods: Submariners (n=58; 27.8±5.9 years) of various ranks volunteered from a U.S. Navy submarine. Submariners wore a research-grade actigraphy watch over a 30-day underway mission, for objective sleep measurement of time in bed (TIB), total sleep time (TST), and sleep efficiency (SE). Subjective sleep was measured from questionnaires (Pittsburgh Sleep Quality Index [PSQI], Insomnia Severity Index [ISI], Profile of Mood States [POMSfatigue subscale]) that were taken pre- and post-underway.

Results: Compared with pre-underway, at post-underway submariners reported significantly higher scores on the ISI, PSQI global sleep quality, and POMS-fatigue (all p<0.05, indicating worse sleep and fatigue ratings). According to actigraphy, submariners acquired on average 6.7 ± 1.0 hours TST, 7.5 ± 1.1 hours TIB, and 88.9 ± 3.9 % SE per day throughout the underway mission. Actigraphy-determined TIB and TST were variable compared with PSQI self-reported TIB and TST.

Conclusion: Study results indicate that submariners experience modest sleep restriction on a newly implemented 24-hour watchbill, which is an improvement in sleep relative to prior assessments of the former standard 18-hour watchbill. However, submariners endorsed lower sleep quality and higher fatigue levels from a month-long underway mission. This study is one of the first examinations of sleep under the 24-hour watchbill mandate that was instated in 2014. Future studies should further evaluate sleep and test fatigue mitigation strategies in different shift configurations of the 24-hour watchbill.

Support: Joint Program Committee-5 Fatigue Mechanisms and Countermeasures Working Group

0285

PREVENTATIVE VOLUNTARY EXERCISE AMELIORATE SYNAPTIC-PRUNING DEFECTS ON SLEEP-DEPRIVED ADOLESCENT

Tuan, L. Tsao, C. Lee, L. National Taiwan University, Taipei, TAIWAN.

Introduction: Since adolescent is a critical period for brain development, adequate sleep during this period is essential to cognitive performance as well as the psychological health in later life. Emerging evidence on sleep-deprived animal models has detailed the impacts of sleep loss on the developing brain. For instance, our previous study demonstrated that 72 h sleep deprivation (SD) disrupted microglia-mediated synaptic refinement in the dentate gyrus (DG). Physical exercise is proved to counteract the harmful consequences of various stress or neurodegenerative models by modulating microglial function. Therefore, we hypothesized that physical

exercise might be a preventive intervention to rescue the failure of synaptic pruning and microglial function after SD in adolescent mice.

Methods: C57/BL6 male mice 3 weeks were assigned to the home cage (HC), home cage with voluntary exercise (HC+VE), sleep deprivation (SD), or sleep deprivation with voluntary exercise (SD+VE) group. In the groups with VE, a running wheel was placed in the cage 11 days before the SD paradigm. Following 72 h SD, animals were subjected to a short-term memory test or sacrificed directly for further examination.

Results: Our results showed that impaired memory performance was reversed in sleep-deprived mice after VE. Also, the SD+VE group exhibited less dendritic spine density compared to the SD group, implying VE rescue the synaptic pruning defect after SD. Greater microglia phagocytic ability, characterized by increased internalized postsynaptic materials and lysosomal structure within individual microglia, were observed in the SD+VE compared to the SD group. We also investigated the mRNA expression of microglia-specific receptors critical to developmental synaptic refinement and found an upregulation of CX3CR1 expression in both HC+VE and SD+VE compared to sedentary groups.

Conclusion: Here we provided evidence featuring that developmental VE significantly alleviated the SD-induced memory defects. Moreover, the SD-induced synaptic pruning impairment and microglial maladaptation were also prevented by the VE regimen, suggesting that physical exercise is a possible therapeutic interventions to the cognitive performance as well as the developmental trajectories to the adolescent brain under the influence of sleep insufficiency. **Support:** Supported by the Ministry of Science and Technology of the Republic of China (Grant number: 104-2314-B-002-129-MY4).

0286

SCHEDULE CHARACTERISTICS OF HEAVY VEHICLE DRIVERS ARE ASSOCIATED WITH EYE-BLINK INDICATORS OF REAL-TIME DROWSINESS ON THE ROAD

Shekari Soleimanloo, S.^{1,2,3} Sletten, T. L.^{4,3} Clark, A.^{4,3} Cori, J. M.^{2,3}
Wolkow, A. P.^{4,3} Beatty, C.^{4,3} Shiferaw, B.^{5,3} Barnes, M.^{2,6}
Tucker, A. J.^{4,3} Anderson, C.^{4,3} Rajaratnam, S. M.^{4,3} Howard, M. E.^{2,3,4,6}
¹Institute for Social Science Research, The University of Queensland, Queensland, Brisbane, AUSTRALIA, ²Institute for Breathing and Sleep, Department of Respiratory and Sleep Medicine, Austin Health, Victoria, Melbourne, AUSTRALIA, ³CRC for Alertness, Safety and Productivity, Victoria, Melbourne, AUSTRALIA, ⁴Turner Institute for Brain and Mental Health, School of Psychological Sciences, Monash University, Victoria, Melbourne, AUSTRALIA, ⁶Faculty of Technology, Victoria, Melbourne, AUSTRALIA, ⁶Faculty of Medicine, Dentistry and Health Sciences, University of Melbourne, Victoria, Melbourne, AUSTRALIA.

Introduction: While up to 52% of heavy vehicle crashes are drowsiness-related, the contributions of schedule factors to realtime objective drowsiness in heavy vehicle drivers (HVDs) have not been studied. Eye-blink parameters are a reliable indicator of driver drowsiness. This study aimed to examine the relationship between work-related factors and objective drowsiness in HVDs.

Methods: HVDs (all males, aged 49.5 \pm 8 years) undertook 5weeks of sleep-wake monitoring (Philips Actiwatch, N=15), and 4-weeks of infrared oculography (Optalert, Melbourne, Australia) to monitor their eye-blink parameters (averaged each minute) while driving their own vehicle (N=12). Participants slept for 5.75 \pm 1.4 hours before the drives. Drowsiness events were defined as any Johns

SLEEP, Volume 43, Abstract Supplement, 2020

Drowsiness Scores (JDS) scores larger than 2.6 based on prior research. The relationships of schedule factors and drowsiness events per hour were assessed via mixed linear regression models.

Results: Drowsiness event rates were 3-5 times greater between 22:00 and 03:00 hours compared to between 16:00 and 17:00 hours (17- 25 events/h vs 5 events/h, P= 0.0001 to 0.007). The frequency of drowsiness events at night varied with shift start time and time into shift (P= 0.0001 to 0.001). Compared to the first hour of driving, drowsiness event rates rose significantly during the 13^{th} to the 21^{st} hours into the shift (13- 59 events/h vs 5.5 events/h, P= 0.0001 to 0.007). During sequential night shifts drowsiness events were 1.8 times more compared to 1–3 sequential day shifts (9 events/h vs 5 events/h, P= 0.012 to 0.019).

Conclusion: Driving at night, for more than 12 hours and sequential night shifts increase real-time drowsiness in HVDs, with these factors interacting resulting in even higher rates of drowsiness events. Longitudinal studies in larger populations will further define how these factors interact to inform the work scheduling of HVDs to reduce the risk of drowsiness.

Support: This research was supported by the CRC for Alertness, Safety and Productivity.

0287

MOTIVATED PERFORMANCE WHILE SLEEP DEPRIVED: NEUROBEHAVIORAL CORRELATES OF ATTENTIONAL EFFORT DEPLOYMENT

Massar, S. A. Lim, J. Sasmita, K. Chee, M. W.

Centre for Sleep and Cognition, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, SINGAPORE.

Introduction: Sleep deprivation (SD) has a negative impact on the motivation to exert effort. This may contribute to the decline in attentional performance observed under SD. In this study we examined how SD affects motivated performance and effort-based decision making. Particularly, we used functional magnetic resonance imaging (fMRI) to uncover the neural mechanisms underlying the interplay between SD and motivated behavior.

Methods: Twenty-seven healthy subjects were tested once after a night of sleep in the lab (9h Time in Bed; Rested Wakefulness = RW), and once after a night of total sleep deprivation (SD). Participants performed an effortful attention task with different incentive levels (0, 10, or 50 cents/fast and correct response). Behavioral performance and fMRI data were collected during task performance. Subsequently, participants performed an effort-based choice task, during which they could choose to earn additional rewards for performing the attention task for a longer duration.

Results: As expected, attentional performance was worse in the SD session compared to the RW session. In addition, performance improved as a function of incentive level both in the RW and the SD session. This reward-effect was accompanied by increased activation in attention-related brain areas, and increased arousal-related thalamus activation. This reward-modulation was more extensive during RW than SD, particularly in the anterior cingulate cortex (ACC) and anterior insula (aIns; both areas involved in effort regulation). Results from the decision making task were less willing to perform the attention task for a further duration after SD compared to RW.

Conclusion: Results show that performance decline after SD is reward-dependent and willingness to perform is reduced. Reward modulation of attention-related brain activation is reduced, particularly in areas that are associated with effort regulation (i.e. ACC and aIns). These findings indicate that motivational factors contribute to decline of vigilance following sleep deprivation.

Support: This work was supported by grants awarded to Dr. Michael Chee from the National Medical Research Council (NMRC/STaR/0015/2013), and the Far East Organization.

0288

WITHDRAWN

0289

WHAT IS THE OPTIMAL DURATION TO SLEEP IN ON WEEKENDS?

Stone, J. E. Cheong, F. Phillips, A. J.

Turner Institute for Brain and Mental Health, School of Psychological Sciences, Monash University, Melbourne, AUSTRALIA.

Introduction: Most individuals in the workforce exhibit differing sleep/wake patterns between work days and weekends. Work days are typically characterized by shorter and earlier sleep. On weekends, sleep debt is repaid by sleeping later and longer, often due to evening events. While social jet-lag (the mismatch in work vs. free sleep timing) is associated with poor health outcomes, repaying sleep debt is beneficial to health. The degree to which individuals should sleep in on weekends is currently unknown.

Methods: We used a mathematical model of human sleep/wake timing, which has been validated for predicting sleep/wake patterns in a variety of field/lab conditions. Sleep timing constraints are inputs, and the model generates predicted sleep/wake patterns and alertness levels. We simulated a traditional 7-day work week, with 7am rise times on week days. Inter-individual differences in chronotype were modeled by varying intrinsic circadian period. The model was applied to two conditions: (i) free choice of sleep onset times on weekends; or (ii) late nights on weekends (2am bedtime). Weekend rise time was systematically varied to optimize predicted daytime alertness.

Results: Optimal weekend rise times varied as a function of chronotype. With free choice sleep onset times, the model predicted optimal rise time was later for late types than early types, ranging from 7:20 to 8:40am across individuals. Sleeping later than optimal was associated with poorer performance due to misaligned circadian phase. The same trend was observed in the late-night condition, but with later optimal rise times, ranging from 8:30 to 9:50am. **Conclusion:** Although individuals should maintain a consistent sleep/wake pattern on all days of the week, they often do not, due to work or social commitments. Within real-world constraints, we provided the first objective recommendations for sleep timing on the weekend, finding a compromise between repaying sleep debt and avoiding circadian misalignment. **Support:** N/A

0290

CAFFEINE EFFICACY VARIES AS A FUNCTION OF INDIVIDUAL VULNERABILITY TO SLEEP RESTRICTION

Demiral, S. B. Doty, T. J. Ratcliffe, R. H. Hughes, J. D. Balkin, T. J. Capaldi, V. F.

Walter Reed Army Institute of Research, Silver Spring, MD.

Introduction: We previously showed that relative to placebo (PL), caffeine (CAF) significantly improved psychomotor vigilance task (PVT) reaction time (RT) during the first 2 days (ACUTE phase), but not during the last 3 days (CHRONIC phase) of sleep restriction (SR) (Doty et al., 2018). However, while individual differences in RT during sleep deprivation have been previously documented, the interaction between CAF and individual vulnerability (VUL) during SR on PVT-RT is not well-known.

Methods: For statistical analysis, we computed trends in RTs (SLOPE) as follows; baseline, 1st and the 2nd SR days to represent ACUTE phase, and the 2nd, 3rd, 4th and 5th SR days for CHRONIC phase. Participants in each GROUP (CAF or PL) were split into 2 for VUL; high vulnerable (HIGHVUL), and low vulnerable (LOWVUL), depending on the number of minor lapses made during SR. We used 3-way ANOVA model with independent measures (2x2x2; GROUPxVULxPHASE) and a dependent measure (SLOPE).

Results: We found a main effect of VUL (F=12.69, p<0.001), an interaction between GROUP and PHASE (F=12.95, p<0.001) and an interaction between VUL, GROUP, and PHASE (F=8.04, p<.01). Resolving this 3-way interaction for *ACUTE* revealed a main effect of VUL (F=9.34, p<.005), a main effect of GROUP (F=5.96, p<.05). Although the interaction between VUL and GROUP failed to achieve significance (F=3.46, p=0.073), only for the LOWVUL, PL participants were significantly higher than CAF, p<0.01). Resolving the 3-way interaction for *CHRONIC* revealed a main effect of GROUP (F=8.95, p<0.01), no significant of VUL (F=3.36, p=0.077) and an interaction between VUL and GROUP (F=6.11, p<0.05). Resolving this interaction showed that only for the LOWVUL participants in CAF, the slope was higher than PL (p<.001).

Conclusion: Performance enhancing effects of caffeine were only evident for low vulnerability participants, and for only the first few days of sleep restriction. At the tested dose level, caffeine did not result in meaningful improvements in performance in highly vulnerable participants during the sleep restriction period.

Support: Department of Defense Military Operational Medicine Research Program (MOMRP)

0291

RECOVERY SLEEP ALLEVIATES MOOD DISTURBANCE FOLLOWING CHRONIC SLEEP RESTRICTION, ALBEIT TRANSIENTLY AND IN A DOSE-DEPENDENT MANNER

Mange, A.¹ Jones, C. W.¹ Kaizi-Lutu, M.¹ Basner, M.¹ Dinges, D. F.¹ ¹Perelman School of Medicine University of Pennsylvania, Philadelphia, PA, ²Perelman School of Medicine University of Pennsylvania, Philadelphia, PA.

Introduction: Fatigue is one contributor to mood disturbance observed following sleep restriction; however, the contribution of other factors remains unclear. This study examined contributions to mood disturbance resulting from sleep restriction beyond that of fatigue, evaluated the benefit of recovery sleep, and assessed whether recovery sleep buffered the re-emergence of mood disturbance upon re-exposure to sleep restriction.

Methods: N=223 healthy participants (48% female; n=108) approximately 30-years-old (SD=6.89, range=22-45 years) completed two baseline nights of 8h time in bed (TIB), followed by five nights of 4h TIB, and were then then randomized to one of 7 sleep recovery opportunities (i.e., 0, 2, 4, 6, 8, 10, or 12 hours TIB). Following the sleep period, a subset of participants (n=72) were re-exposed to five consecutive nights of 4h TIB. The profile of mood states (POMS) was completed every 2h during wakefulness and daily averages were calculated. The POMS total mood disturbance (TMD) score without the fatigue subscale (i.e., mood disturbance = TMD - fatigue) was the primary outcome to isolate changes in mood disturbance beyond fatigue. Individual growth curve models were applied to the trajectory of mood disturbance. General linear models were used to evaluate the dose-response function of mood disturbance after recovery sleep.

Results: Mood disturbance (absent the POMS fatigue scale) increased with each day of sleep restriction (β =1.550 per day;

P<0.0001), and decreased with longer recovery sleep durations in a dose-dependent manner (β =-1.614 for every 2h increase; P<0.0001). The benefits of recovery sleep were abated by the second night of 4h sleep during re-exposure, where mood disturbance was slightly higher than that observed before recovery, but this difference was not statistically significant (β =0.046; P=0.85).

Conclusion: The study findings suggest that fatigue is not the only contributor to mood disturbance following sleep restriction. Recovery sleep attenuates mood disturbance in a dose-dependent manner, albeit transiently. Candidate pathways linking sleep restriction and mood include the immune system and the dynamics of sleep physiology. **Support:** This work was funded by National Institute of Health NIH R01NR004281 and National Space and Biomedical Research

0292

Institute NSRBI NCC 5-98.

BASELINE PRO-INFLAMMATORY CYTOKINE AND CORTISOL LEVELS DIFFERENTIALLY PREDICT MOOD DISTURBANCE AND WORKING MEMORY DEFICITS INDUCED BY CHRONIC SLEEP RESTRICTION

Jones, C. W. Kaizi-Lutu, M. Mange, A. Basner, M. Dinges, D. F. Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA.

Introduction: Sleep restriction disturbs mood, impairs neurocognitive performance, and elevates proinflammatory cytokine levels. However, whether basal inflammation influences mood and neurocognitive performance across sleep restriction is unknown. This study examines whether baseline IL-6, cortisol, and TNF- α levels predict the deterioration of mood and working memory performance across sleep restriction.

Methods: N=124 healthy participants (52% female; n=64), 22–45 years of age, had valid protein, mood, and working memory data. The study included two baseline nights (8h time in bed [TIB]) followed by five nights of 4h TIB. Venous blood was collected on the second baseline day and IL-6, cortisol, and TNF- α levels were measured via commercially available ELISA assays. The profile of mood states (POMS) and the digit span test (DST) were completed every 2h during wakefulness and daily averages were computed. Mixed-effects multi-level models, adjusted for baseline, evaluated the main effect of IL-6, cortisol, and TNF- α levels on the POMS and DST independently and examined the trajectory of POMS and DST by the interaction of protein levels and day of sleep restriction.

Results: At baseline, IL-6, cortisol, and TNF- α levels were not associated with POMS or DST. There was a main effect of IL-6, but not cortisol or TNF- α , levels on mood disturbance (β =3.811; P=0.015); IL-6 levels did not predict the trajectory of mood across sleep restriction (β =0.187; P=0.57). Higher baseline cortisol levels predicted increasing mood disturbance across days (β =-1.329; P<0.0001). Higher baseline TNF- α levels predicted degrading DST performance across days (β =-0.313; P=0.020). Higher IL-6 (β =-0.246; P=0.010) and lower cortisol (β =0.185; P=0.037) levels also predicted degrading DST performance across days.

Conclusion: The study findings suggest that basal inflammatory cytokine and cortisol levels are implicated in the individual risk of mood disturbance and working memory deficits resulting from chronic sleep restriction and highlight the need to consider biological processes and phenotypes together.

Support: This work was supported by National Institute of Health NIH R01NR004281 and National Space and Biomedical Research Institute NSRBI NCC 5–98.

0293

SLEEP DEPRIVATION AFFECTS THE ACOUSTIC PROPERTIES OF HUMAN SPEECH

THORET, E.¹ GAURIAU, C.² ANDRILLON, T.³ PRESSNITZER, D.¹ LEGER, D.⁴

¹Laboratoire des systèmes perceptifs, Département d'études cognitives, École normale supérieure, PSL University, CNRS, Paris, FRANCE, ²Université de Paris, Paris Descartes, EA 7330 VIFASOM, Paris, FRANCE, ³Université de Paris, Paris Descartes EA 7330 VIFASOM, Paris, FRANCE, ⁴Université de Paris, Paris Descartes, PARIS, FRANCE.

Introduction: Lack of sleep drastically affects many aspects of human behavior. The early detection of sleepiness is thus a major challenge for health and security reasons. Here we investigated the effect of sleep deprivation on the acoustic properties of human speech.

Methods: Twenty-four participants were sleep deprived for two days (two successive nights with only 3 hours of sleep). They were recorded reading a short text aloud before and after sleep deprivation. An auditory model, based on spectro-temporal modulations, was used to analyse the acoustic properties of their speech and served as a front-end to machine-learning classifiers.

Results: Results showed that sleepiness could be accurately detected with individually-trained classifiers. However,we were not able to fit a generic classifier for all participants. As we relied on an auditory-inspired model,we could identify and interpret the acoustic features impacted by sleep deprivation. Again,no simple diagnostic feature could be easily identified in the group- level analyses of the speech signals. We therefore developed a novel probing method, combining signal detection theory and noise activation of the classifier, to understand what made the classifier successful for each participant. This led to a diagnostic map for each participant, specifying which frequency region and modulation rates were impacted by sleep deprivation for this particular individual

Conclusion: In addition to suggesting a practical machine learning algorithm to detect sleep deprivation, combining our probing method with considerations about voice production could help uncover the physiological impact of sleep deprivation at the level of each individual. **Support:**

0294

BASELINE RESPONSE SPEED PREDICTS LOCUS COERULEUS INTEGRITY CHANGE AFTER SLEEP DEPRIVATION

Quan, P.¹ Lei, H.¹ Wang, J.¹ Liu, W.¹ Zhang, X.¹ Dinges, D.² Rao, H.¹ ¹Center for Functional Neuroimaging, Department of Neurology, University of Pennsylvania, Philadlephia, PA, ²Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania, Philadlephia, PA.

Introduction: Locus coeruleus (LC) is the major source of norepinephrine (NE) in the brain, which plays a key role in maintaining arousal and alertness. Sleep loss significantly impairs arousal and alertness. However, it is unknown whether sleep loss disrupts LC integrity, which can be measured non-invasively by diffusion tensor imaging (DTI). In the current study, we used DTI to examine the effects of one night of acute total sleep deprivation (TSD) on fractional anisotropy (FA), an index reflecting fiber density, axonal diameter and myelination.

Methods: We analyzed DTI and psychomotor vigilance test (PVT) data from N=54 health adults (23 females, age range 21–50 years) from a well controlled in-laboratory sleep deprivation study. Participants

were randomized to either a TSD condition (n=40) without sleep on night 2, or a control condition (n=14) with no sleep loss. Standard DTI scans were conducted on the morning of day 2 and day 3 between 0700h-1000h. The PVT reciprocal response time (RRT) was used to measure individual's response speed at baseline without sleep loss. LC regions-of-interest (ROI) were defined by standard templates from Keren et al. (2009). Imaging data were analyzed using FSL toolbox.

Results: For the whole TSD group, no differences were found in the LC FA values before and after sleep deprivation (p > .2). However, when dividing the TSD group to a slow group and a fast group based on their baseline PVT response speed, significantly increased LC FA were found in the slow group (p = .007) but not in the fast group (p > .4). The PVT RRT negatively correlated with LC FA value changes after TSD (r = .44, p = .004). No correlations were found between the PVT RRT and LC FA changes in the control group.

Conclusion: Our results showed that baseline vigilance response speed correlated with LC integrity change after sleep deprivation, with slower response exhibiting greater changes in LC integrity. These findings support the key role of LC-NE system in the regulation of alertness and arousal.

Support: Supported in part by NIH grants R01-HL102119, R01-MH107571, R21-AG051981. CTRC UL1RR024134, and P30-NS045839.

0295

SKELETAL MUSCLE DIACYLGLYCEROL ACCUMULATION AND IMPAIRED INSULIN SENSITIVITY DURING INSUFFICIENT SLEEP

Morton, S. J.¹ Bergman, B. C.² Zemski-Berry, K. A.² Harrison, K. A.² Schauer, I. E.^{2,3} Wright, K. P.^{4,2} Broussard, J. L.^{1,2} ¹Sleep and Metabolism Laboratory, Department of Health and Exercise Science, Colorado State University, Fort Collins, CO, ²Division of Endocrinology, Metabolism and Diabetes, University of Colorado Anschutz Medical Campus, Aurora, CO, ³Rocky Mountain Region Veterans Affairs Medical Center, Aurora, CO, ⁴Sleep and Chronobiology Laboratory, University of Colorado Boulder, Boulder, CO.

Introduction: Insufficient sleep impairs insulin sensitivity; however, the mechanism(s) by which this occurs are unknown. We previously reported an elevation in plasma free fatty acid concentration during insufficient sleep, suggesting dysregulated lipid metabolism. Lipid accumulation in muscle—specifically certain species of diacylglycerol (DAG)—is associated with impaired insulin sensitivity. We therefore tested the hypothesis that insufficient sleep leads to skeletal muscle DAG accumulation.

Methods: As part of an ongoing study, thirteen sedentary, healthy, lean adults ($25.8\pm3.2y$; $22.7\pm1.9kg/m^2$; 3F; mean \pm SD) participated in a controlled 6-day in-laboratory protocol with 9h in bed (habitual sleep) followed by 4 nights of 5h in bed (insufficient sleep), achieved by delaying bedtime by 4 hours. For one week prior to the study, participants maintained a 9h sleep schedule. Participants consumed energy balanced diets 3 days prior to and throughout the laboratory protocol. Insulin sensitivity was assessed using a hyperinsulinemic euglycemic clamp before and after insufficient sleep. Skeletal muscle biopsies of the vastus lateralis were taken immediately before each clamp. In a subset of subjects (n=10), quantitative lipidomic analyses using LC/MS/MS were performed on biopsied muscle tissue.

Results: Insulin sensitivity was impaired following insufficient sleep $(10.7\pm1.5 \text{ vs } 9.6\pm1.2 \text{ mg/kg/min}, \text{p}<0.05, \text{mean}\pm\text{SEM})$. There were no changes in skeletal muscle concentration of total triglycerides (TAGs), nor specific TAG species. However, insufficient sleep tended to increase

skeletal muscle accumulation of total 1,2-DAGs (p=0.13) and significantly increased specific saturated species of 1,2-DAG, including Di-C18:0 DAG (p<0.05), previously implicated in insulin resistance. In contrast, 1,3-DAGs are not thought to impair insulin sensitivity and specific species were decreased or unchanged during insufficient sleep. **Conclusion:** Preliminary findings suggest that skeletal muscle lipid accumulation of diacylglycerol species during insufficient sleep may be a contributing mechanism by which insufficient sleep dysregulates metabolic physiology.

Support: NIH K01DK110138, R03 DK118309, UL1 TR002535, and GCRC RR-00036

0296

LESS SELF-REPORTED ALERTNESS AND MOTIVATION DURING SLEEP RESTRICTION ARE ASSOCIATED WITH DECREASED ATTENTIONAL PERFORMANCE

Mathew, G. M.¹ Strayer, S. M.¹ Ness, K.² Bailey, D. S.³ Buxton, O. M.^{1,4,5} Chang, A.^{1,6}

¹Department of Biobehavioral Health, College of Health and Human Development, Pennsylvania State University, University Park, PA, ²Department of Medicine, Division of Metabolism, Endocrinology, and Nutrition, University of Washington, Seattle, WA, ³Penn State College of Medicine, Hershey, PA, ⁴Division of Sleep Medicine, Harvard Medical School, Boston, MA, ⁵Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, Boston, MA, ⁶College of Nursing, Pennsylvania State University, University Park, PA.

Introduction: Some individuals demonstrate more performance decrements on the psychomotor vigilance task (PVT) after sleep restriction (SR). We investigated whether individuals who reported less alertness and/or less motivation after SR demonstrated poorer performance on the PVT.

Methods: Fifteen healthy men (22.3±2.8 years) participated in a 10-night inpatient protocol with three nights of 10-hour baseline time in bed (TIB), five nights of SR (5-hour TIB), then two recovery (10-hour TIB) nights. Participants completed the 10-minute PVT (Joggle Research® battery) approximately every two hours during wake. Outcomes included number of false starts (<100 ms reaction time, RT) and number of lapses (≥500 ms RT). Participants reported alertness and motivation levels after each PVT. Median splits were used to characterize changes in alertness ("sleepy," n=8, versus "alert," n=7) and motivation ("unmotivated," n=7, versus "motivated," n=8) from the last day of baseline to the last day of SR. Outcomes were analyzed in mixed models with the predictor day*alertness or day*motivation, excluding the first three baseline days to preclude practice effects.

Results: There were significant interactions between day and alertness (p=.025) and day and motivation (p=.043) for false starts. False starts followed a quadratic inverted-U shape across days in sleepy (b=-0.16, p=.003) and unmotivated (b=-0.16, p=.004) participants, but not in alert or motivated participants (p>.05). There was a significant interaction between day and alertness for lapses (p=.008); lapses followed a quadratic inverted-U shape across days with a stronger effect in sleepy (b=-0.43, p<.001) versus alert (b=-0.15, p=.031) participants. There was no interaction between day and motivation for lapses.

Conclusion: Participants reporting less alertness were more likely to make both false starts and lapses after SR; those reporting less motivation were more likely to make false starts, but not lapses. Findings suggest greater motivation is sufficient to preserve inhibitory control but not vigilance after sleep restriction. In contrast, greater alertness despite sleep restriction was sufficient to preserve inhibitory control and resulted in lower vigilance decrements.

Support: This study was funded by grant UL1TR000127 from the Clinical and Translational Science Institute and the College of Health and Human Development at the Pennsylvania State University (Chang PI).

0297

RELATIONSHIP BETWEEN SLEEPINESS SYMPTOMS QUESTIONNAIRE RATINGS AND PSYCHOMOTOR VIGILANCE PERFORMANCE IN A TOTAL SLEEP DEPRIVATION STUDY

Skwara, A. K.^{1,2,3} Skeiky, L.^{1,2} Van Dongen, H.^{1,2} Hansen, D. A.^{1,2} ¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ³School of Biosciences and Medicine, University of Surrey, Guildford, UNITED KINGDOM.

Introduction: While measuring the neurobehavioral consequences of sleepiness is arguably best done with performance tasks providing objective assessments of impairment, this often proves challenging in real-world operational settings. Self-report instruments measuring subjective sleepiness provide a practical alternative, but field studies generally fail to show high correlation between objective and subjective assessments of impairment. The Sleepiness Symptoms Questionnaire (SSQ) is a self-report instrument developed to address this issue by asking about observable symptoms of sleepiness and (motor vehicle driving) performance impairment. In a laboratory sleep deprivation study, we compared SSQ sleepiness ratings to performance impairment on a 10min psychomotor vigilance test (PVT). Methods: N=12 healthy normal sleepers (ages 21-39v, 6 females) participated in a 4-day in-laboratory study. After a baseline day (9h time in bed; 22:00-07:00), subjects underwent 38h total sleep deprivation (TSD) followed by a recovery day (9h time in bed; 22:00-07:00). As part of neurobehavioral testing throughout the experiment, subjects completed the SSQ and PVT back to back at 6.5h, 14.5h, 22.5h, and 30.5h TSD, and 6.5h after recovery sleep. Data from one subject were incomplete and discarded prior to analysis.

Results: As TSD progressed, the SSQ sleepiness ratings and the number of lapses (RTs>500ms) on the PVT were elevated, with sleepiness and performance impairment peaking at 22.5h TSD. Both measures returned to baseline levels after recovery sleep. Mixed-effects analysis of covariance showed moderate correlation between SSQ ratings and PVT lapses (r=0.44, $F_{1,43}$ =24.1, p<0.001). **Conclusion:** Self-reported sleepiness on the SSQ reflected the expected homeostatic and circadian changes in sleep pressure during TSD and following recovery sleep, as did PVT performance impairment. The moderate correlation between subjective ratings on the SSQ, with its emphasis on observable sleepiness symptoms, and objective impairment on the PVT suggests that the SSQ may be a reasonably reliable instrument for measuring sleepiness under conditions of acute sleep deprivation. **Support:** Jazz Pharmaceuticals

0298

SLEEP AT SEA: A QUALITATIVE EXAMINATION OF BARRIERS TO SLEEP AND MITIGATION STRATEGIES AMONG SHIPBOARD SAILORS

Schmied, E. A.¹ Harrison, E. M.² Dell'Acqua, R.¹ Glickman, G. L.³ Hurtado, S. L.⁴

¹Leidos, Inc, San Diego, CA, ²University of California San Diego, San Diego, CA, ³Uniformed Services University of the Health Sciences, Bethesda, MD, ⁴Naval Health Research Center, San Diego, CA.

Introduction: Sleep disturbance is prevalent among service members; however, little is known about factors that impede sleep in unique operational environments, such as aboard naval ships. Given the importance of sleep to health and performance, identifying both causes and potential solutions to sleep disturbance is critical. The objective of this qualitative study was to elucidate barriers to sleep and the strategies U.S. Navy sailors use to improve their sleep and combat fatigue while underway.

Methods: Interviews were conducted with 22 active duty service members assigned to sea duty. The semi-structured interview guide assessed the experiences of service members sleeping in shipboard environments, including the strategies used to improve sleep and combat fatigue. Interview transcripts were analyzed using applied thematic content analysis by two independent coders.

Results: Most participants were male (91.8%) and enlisted (95.5%). The most commonly reported barrier to sleep was stress (e.g., job-related stress), followed by rotating schedules, and environmental factors (e.g., noise, light). Many reported prioritizing other activities over sleep when off duty. Though only a few reported specific strategies to improve sleep while underway, strategies that were described primarily included mitigation of environmental barriers (e.g., noise-canceling headphones or sleep masks). However, some participants acknowledged these strategies are not always feasible (e.g., cost, reduced responsiveness to alarms or commands). Notably, few sailors reported using stress mitigation or relaxation strategies to help sleep. Caffeine intake was the only reported strategy for alerting when fatigued.

Conclusion: Sailors reported many barriers to sleep that are unique to the shipboard environment, yet most did not report using any mitigation strategies. Further, few used alerting techniques (other than caffeine) when fatigued. This at-risk population could benefit from targeted educational interventions covering sleep-promoting behaviors, prioritization of sleep, and fatigue mitigation.

Support: This work was supported by the Military Operational Medicine Research Program, Early Assessment and Intervention Working Group, under work unit no. N1702.

0299

TOTAL SLEEP DEPRIVATION AND TIME ON TASK: NOT THE SAME FOR SUSTAINED ATTENTION AND EXECUTIVE PROCESSES AND POOR BENEFIT OF CAFFEINE

Erblang, M.^{1,2} Quiquempoix, M.^{1,2} Vergez, A.¹ Van Beers, P.^{1,2} Guillard, M.^{1,2} Elbaz, M.^{3,2} Drogou, C.^{1,2} Léger, D.^{3,2} Chennaoui, M.^{1,2} Sauvet, F.^{1,2} Rabat, A.^{1,2}

¹IRBA, Brétigny sur Orge, FRANCE, ²EA 7330 VIFASOM, Paris, FRANCE, ³Centre du sommeil et de la vigilance, Hôtel Dieu, Paris, FRANCE.

Introduction: Mental Fatigue is commonly questioned regarding time on task or sleep debt effect (Hockey, 2013; Pattyn et al., 2018) or sleep debt effect (Krause et al., 2017). No studies have neither investigated contributions of these two factors for different cognitive processes nor benefit of caffeine.

Methods: 24 right-handed and healthy subjects (18–50 years old), with a median chronotype and sleep need participated in a 2-experimental counter-balanced (placebo: PBO and caffeine: COFFEE - 2.5 mg/kg) total sleep deprivation protocol (TSD = 27 hours of continuous wakefulness). Subjective sleepiness (KSS), sustained attention (PC-PVT), inhibition (Go-NoGo) and working memory (2N-Back) capabilities were tested each morning during BASE and TSD (10 min. test session from 9:15 am to 10:15 am). Caffeine was ingested with a decaffeinated drink at 8:30 am.

Results: KSS score (5.6 \pm 0.4 vs 3.2 \pm 0.3; p<0.001), number of Lapses (9.8 \pm 1.7 vs 0.4 \pm 0.2; p<0.001), mean response time (RT: 308 \pm 4.9 vs 260 \pm 9.4; p<0.001) in PVT, errors of commission (6.4 \pm 0.4 vs 3.1 \pm 0.3; p<0.01) and mean response time (RT: 336 \pm 24 vs 301 \pm 13; p<0.01) in Go-NoGo were significantly higher after TSD compared to BASE. Neither significant difference in the proportion of correct responses (CR: 0.92 \pm 0.015 vs 0.90 \pm 0.014; p>0.15) nor RT (592 \pm 49 vs 640 \pm 28 ms, p > 0,11), were observed in the 2N-Back task. Further analyses showed different TOT x TSD interaction for PVT (after 3 min of task engagement), Go-NoGo (after 6 min) and 2N-Back (after 8 min). Number of Lapses was significantly but partially reduced (5.8 \pm 0.4 vs 9.8 \pm 1.7; p<0.01) in COFFEE condition compared with PBO with more aversive effects for Go-NoGo errors and 2N-Back BR.

Conclusion: Our results are in accordance with previous studies showing differential kinetic of cognitive deficits under TSD, limited benefit of sleep banking and regular physical activity (Arnal et al., 2015; Rabat et al., 2019; Sauvet et al., 2019) and no benefit of caffeine on executive processes (Gottsellig et coll., 2006; Killgore et coll., 2007, 2012).

Keywords: TSD, TOT, Attention, Executive, Caffeine. **Support:** Grants from the *French General Directorate for Armament* (Contract Number: SAN-1–509).

0300

THE CIRCADIAN TIMING OF SLEEP AFFECTS THE RATE OF ACCUMULATION OF NEUROBEHAVIORAL IMPAIRMENT ACROSS DAYS OF SLEEP RESTRICTION *McCauley, M. E.*^{1,2} Van Dongen, H.^{1,2} Banks, S.³ Dinges, D. F.⁴

¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ³Sleep and Chronobiology Laboratory, Behaviour-Brain-Body Research Centre, University of South Australia, Adelaide, AUSTRALIA, ⁴Division of Sleep and Chronobiology, Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA.

Introduction: Chronic restriction of nighttime sleep to less than \sim 8h/day leads to build-up of neurobehavioral impairment across days. Although it is known that sleep loss effects depend on the circadian timing of sleep, it is not known how the timing of restricted sleep influences the accumulation of neurobehavioral impairment over days. Here we studied the accumulation of impairment across days of restricted sleep placed in the morning or afternoon.

Methods: N=71 healthy young adults (39% female; ages 21-45y, mean \pm SD: 27.9 \pm 6.6y) completed a 14-day laboratory study. After two baseline days with nighttime sleep (8h TIB: 23:30-07:30), subjects were randomized to 10 consecutive days of A) morning sleep at 4h, 6h, or 8h TIB ending at 11:30 each day (n=18, 8, 8, respectively), or B) afternoon sleep at 4h, 6h, or 8h TIB ending at 19:30 each day (n=13, 17, 7, respectively). Subjects were tested on the 10min psychomotor vigilance test (PVT) every ~2 hours during scheduled wakefulness. Daily averages for PVT lapses (RTs>500ms) observed between 2h and 14h after awakening were analyzed with non-linear mixed-effects regression to investigate differences in the neurobehavioral impairment build-up rate between sleep restriction conditions.

Results: Afternoon sleep conditions showed a significant sleep doseresponse effect (p<0.001), with the fastest accrual of PVT performance deficits across days in the 4h condition, and slow-to-negligible accumulation (p=0.36) of PVT performance deficits in the 8h condition. However, morning sleep resulted in no significant sleep dose-response effect (p=0.96). All 3 morning sleep doses displayed negligible ($p\ge0.12$) accumulation of impairment across days.

Conclusion: In this sample of young adults, sleep dosages ending in the morning (at 11:30) appear to provide considerable protection against cumulative performance deficits from sleep restricted to 4h-6h/day over 10 days, suggesting that the afternoon circadian promotion of wakefulness can sustain behavioral alertness even over multiple days of repeated sleep restriction.

Support: NIH grants R01-NR04281 and M01-RR00040

0301

DIFFERENT INDICES OF VIGILANT ATTENTION DURING SLEEP DEPRIVATION: EVIDENCE OF MULTIPLE VIGILANCE CONSTRUCTS?

Lawrence-Sidebottom, D.^{1,2,3} Hinson, J. M.^{1,4} Whitney, P.^{1,4} Honn, K. A.^{1,2} Van Dongen, H.^{1,2}

¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ³Neuroscience graduate program, Washington State University, Pullman, WA, ⁴Department of Psychology, Washington State University, Pullman, WA.

Introduction: Total sleep deprivation (TSD) causes profound vigilant attention deficits, with large, trait-like individual differences, as evidenced convincingly by response lapses on the psychomotor vigilance test (PVT). There is debate, however, about the role of vigilant attention deficits in the effects of TSD on other speeded performance tasks besides the PVT. We addressed this issue by testing whether PVT response lapses are related to delays in responding to stimuli under strict deadlines in two decision making tasks.

Methods: N=54 healthy adults (aged 21-38y; 31 females) completed an in-laboratory TSD study. Following a 10h baseline sleep opportunity, cognitive testing occurred after 25h and 29h of TSD (09:00 and 13:00). Testing included an AX continuous performance task with switch (AX-CPTs), which is a dynamic decision making task requiring subjects to respond to a frequently occurring cue-probe combination; an identical pairs continuous performance task (CPT-IP), which is a 1-back go/ no-go task; and a 10min PVT. Lapses (RTs>500ms) on the PVT and target accuracy on the AX-CPTs and CPT-IP were calculated as indices of vigilant attention. Intraclass correlation coefficients (ICCs) were used to quantify the stability of individual differences, and absolute rank-order correlation ($|\rho|$) was used to compare the three indices.

Results: The stability of individual differences ranged from fair to substantial (PVT: ICC=0.44; AX-CPTs: ICC=0.73; CPT-IP: ICC=0.31). The rank-order correlation between the AX-CPTs and CPT-IP vigilant attention indices was relatively high ($|\rho|$ =0.44), whereas correlations with PVT lapses were much lower (AX-CPTs: $|\rho|$ =0.14; CPT-IP: $|\rho|$ =0.04).

Conclusion: Individual differences during TSD were moderately stable for each index of vigilant attention, but the relationships between PVT lapses and the other indices were weak. This suggests that any or all of the indices considered here are not pure measures of vigilant attention, or that vigilant attention may constitute multiple, distinct constructs. **Support:** CDMRP grant W81XWH-16-1-0319

0302

ONE WEEK OF RECOVERY SLEEP IS INSUFFICIENT TO RESTORE SUSTAINED ATTENTION PERFORMANCE FOLLOWING THREE WEEKS OF CHRONIC SLEEP RESTRICTION

Yuan, R. K.^{1,2} Zitting, K.^{1,2} Vujovic, N.^{1,2} Wang, W.^{1,2} Buxton, O.^{1,2,3} Williams, J. S.^{1,2} Czeisler, C. A.^{1,2} Duffy, J. F.^{1,2} ¹Brigham and Women's Hospital, Boston, MA, ²Harvard Medical School, Boston, MA, ³Pennsylvania State University, University Park, PA.

Introduction: Sleep loss negatively impacts many aspects of neurobehavioral performance, including sustained attention and reaction times. However, the time course of recovery from chronic sleep restriction (CSR) is not well understood. To explore this, we assessed the effects of 3 weeks of CSR followed by 1 week of recovery on psychomotor vigilance task (PVT) performance in healthy adults.

Methods: 8 healthy adults (27–71; 4f) participated in a 37-day inpatient study. The study consisted of 6 baseline (BL) days with 8–16 h time-in-bed, followed by 3 weeks of CSR (5-5.6h time-inbed at night), and 1 week of recovery (RC; 8-10h time-in-bed). Sustained attention was assessed by 10-minute visual PVTs administered every 2h starting ~5h after wake (~4/day). Linear and generalized linear mixed models were used to compare average reaction times (RT) and number of lapses, respectively, from the last 3 days of baseline, CSR, and recovery.

Results: Average RT was almost twice as long at the end of CSR compared to baseline (p<0.0001). Moreover, it remained significantly slower than baseline by roughly 173ms, even after 1 week of recovery (p<0.0001). Similarly, there was a threefold increase in the number of lapses at the end of CSR compared to baseline (p<0.0001) which remained elevated after one week of recovery (p<0.0001).

Conclusion: One week of recovery sleep of 8-10 h/night following 3 weeks of chronic sleep restriction was insufficient for full recovery of sustained attention as assessed by PVT reaction time and number of lapses. This suggests that chronic sleep restriction has consequences on neurobehavioral performance that do not fully dissipate within one week.

Support: Study supported by P01AG009975 and conducted in the Brigham and Women's Hospital Center for Clinical Investigation, part of Harvard Clinical and Translational Science Center supported by UL1TR001102. KMZ supported by a fellowship from the Finnish Cultural Foundation. RKY supported by T32HL007901 and F32HL143893. NV supported by T32HL007901 and F32AG051325.

0303

HEART RATE AND SYSTOLIC BLOOD PRESSURE INCREASE DURING EXPERIMENTAL SLEEP RESTRICTION

Reichenberger, D. A. Strayer, S. M. Mathew, G. M. Buxton, O. M. Chang, A.

Pennsylvania State University, State College, PA.

Introduction: Experimental sleep restriction is associated with elevated daytime cardiac activity, including heart rate (HR) and blood pressure (BP). However, some studies have found changes in systolic (SBP) but not diastolic blood pressure (DBP) or found changes in neither. Although findings are mixed, there may be a dose-response effect of cumulative sleep loss on daytime cardiac activity, such that HR and BP increase above basal levels with additional nights of insufficient sleep. This study examined changes in cardiac activity during experimental sleep restriction.

Methods: We used multilevel models with random effects for individuals to analyze data from 15 healthy males (M=22.3 years old, SD=2.8) in an 11-day inpatient protocol consisting of three nights of 10-hour/night baseline sleep opportunity, five nights of sleep restriction (5-hour/night sleep opportunity), and then two recovery nights (10-hour/night sleep opportunity). HR and BP were measured approximately every two hours during wake.

SLEEP, Volume 43, Abstract Supplement, 2020

Results: HR increased 0.75 beats/minute with each successive night of sleep restriction (SE=0.18, p<0.001). HR was 5.13 beats/minute higher during the recovery condition than during baseline or sleep restriction (SE=1.05, p<0.001). During sleep restriction only, HR was lower in the later morning and evening compared to the earliest morning timepoint of the day, F(10, 743)=10.44, p<0.001. SBP increased 0.33 mmHg following each successive night of sleep restriction (SE=0.16, p=0.041); however, SBP was only marginally higher during the sleep restriction condition than during baseline (b=1.90, SE=1.09, p=0.082).

Conclusion: Our findings suggest that HR and SBP increase with each additional day of experimental sleep restriction, even after accounting for diurnal effects on HR and SBP. HR did not recover to baseline levels following a night of recovery sleep, suggesting that longer recovery sleep may be necessary to recover from a week of sleep restriction.

Support: Grant UL1TR000127 (Chang PI), Clinical and Translational Science Institute; College of Health and Human Development at Pennsylvania State University.

0304

GREATER SLOW-WAVE ACTIVITY IS ASSOCIATED WITH DETERIORATING MOOD ACROSS SLEEP RESTRICTION

Larson, O. R.¹ Jones, C. W.² Basner, M.² Dinges, D. F.²

¹Department of Psychology, University of Pennsylvania,

Philadelphia, PA, ²Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA.

Introduction: Mood progressively deteriorates over consecutive days of sleep restriction. The neurobiological processes active during sleep that influence the risk of mood disturbance are unknown. This study investigated the relationships between physiological sleep parameters (i.e., slow-wave activity (SWA), slow-wave energy (SWE), rapid eye-movement (REM) sleep duration and latency), and self-reported measures of mood across sleep restriction. Methods: N=181 healthy participants (48.1% female; 30±6.8 yrs) had valid polysomnography (PSG) and mood data. The study design included two baseline nights (8h time in bed [TIB]) followed by five nights of 4h TIB. PSG (EEG derivations C3-A2, Fz-A1, O2-A1) was collected on the second baseline night (B2), first night of 4h TIB (SR1), and the fifth night of 4h TIB (SR5). The Profile of Mood States was assayed on days following PSG. Power spectral analysis for SWE and SWA was conducted (delta power; band: 0.5-4.5 Hz). General linear regression models were used to independently assess the slope of SWE, SWA, percent REM of total sleep time (TST), and REM latency on mood disturbance across sleep restriction.

Results: At baseline, higher SWE (unadjusted; r=0.21; P=0.004) and SWA (unadjusted; r=0.19; P=0.007) were associated with greater mood disturbance; these relations were attenuated when adjusted for age and sex. No relation was found between mood and REM latency or REM percent of TST. The slope of mood disturbance from B2 to SR5 was associated with greater percentage increases in C3 SWA on SR5 relative to B2 (β =0.039; P=0.008); this association was not observed for SWE (β =-0.016; P=0.48). The slope of REM latency and REM percent of TST were not associated with the slope of mood disturbance.

Conclusion: Our results indicate that greater SWA due to sleep restriction was associated with greater mood disturbance, suggesting that less SWA may confer resilience to mood disturbances resulting from sleep restriction.

Support: This work was supported by National Institute of Health NIH R01NR004281 and National Space and Biomedical Research Institute NSRBI NCC 5-98.

0305

GREY MATTER VOLUMETRIC DIFFERENCES ARE PREDICTIVE OF ATTENTIONAL LAPSES DURING SLEEP DEPRIVATION

Vanuk, J. R.¹ Raikes, A. C.² Dailey, N. S.² Grandner, M. A.² Killgore, W. D.² ¹University of Arizona Psychiatry/Psychology, Tucson, AZ, ²University of Arizona Psychiatry, Tucson, AZ.

Introduction: Inter-individual differences in resistance to cognitive effects of sleep loss are well established and extend from basic vigilance capacities to more nuanced emotional processing. Neurobiological markers related to gray matter volumetric differences associated with resilience to sleep deprivation (SD) have yet to be explored. We collected anatomical magnetic resonance imaging on well-rested healthy adults and correlated gray matter volume (GMV) with the number of lapses on a psychomotor vigilance test (PVT) subsequently occurring over 29-hours of SD.

Methods: 45 individuals (23 males; mean age: $25.36 \pm 5.62y$) completed a baseline neuroimaging session while well-rested and returned 2-4 days later to complete 29h of SD. The PVT was administered at one-hour intervals across SD. High-resolution T1 structural scans were used for a volume-based morphometric analysis (CAT12). Images were segmented and normalized following automated procedures and smoothed at 8 mm FWHM. Regions of interest were constrained to the anterior cingulate and ventral frontal areas of the cortex. GM volume was correlated with the total number of lapses across all PVT administrations, after controlling for age, sex, and total intracranial volume.

Results: Total number of lapses positively correlated with GMV in two clusters comprised of areas in the anterior cingulate cortex (FWE corrected, p = 0.046), as well as the opercular and triangular parts of the inferior frontal gyrus (FWE corrected, p = 0.006).

Conclusion: Susceptibility to attentional lapses was predicted by greater gray matter volume in the ventrolateral prefrontal and anterior cingulate cortices. Current findings support that individual differences in attentional resiliency during SD may be, in part, due to differences in gray matter volume within cortical areas previously shown to be functionally affected by sleep loss. **Support:** DARPA (12-12-11-YFA11-FP-029)

0306

CORTICOTROPIN-RELEASING HORMONE RECEPTOR 1 GENE POLYMORPHISM MODULATES COGNITIVE FLEXIBILITY FOLLOWING ACUTE STRESS AND TOTAL SLEEP DEPRIVATION

Satterfield, B. C.^{1,2,3} Anlap, I.¹ Esbit, S. L.¹ Killgore, W. D.¹ ¹Social, Cognitive, and Affective Neuroscience Lab, University of Arizona, Tucson, AZ, ²Sleep and Performance Research Center, Washington State University, Spokane, WA, ³Elson S. Floyd College of Medicine, Washington State University, Spokane, WA.

Introduction: Dynamic decision processes requiring flexible updating of information are impaired by stress and sleep loss, both of which activate the hypothalamic-pituitary-adrenal (HPA) stress response. Corticotropin-releasing hormone (CRH) initiates the HPA pathway. The CRH receptor (CRHR1) gene contains a single nucleotide polymorphism that modulates this response. We investigated whether cognitive flexibility is affected by CRHR1 polymorphism following a night of acute stress and total sleep deprivation (TSD). **Methods:** N=46 healthy, young adults ($21.8\pm3.4y$; 21 females) participated in an in-laboratory 31h sleep deprivation study. Beginning at 19:30 until 07:30, the Maastricht Acute Stress Test (MAST) was

administered every 4h. The MAST alternates a cold pressor task with an oral subtraction task five times in a single bout. At 29h wakefulness, subjects performed a novel go/no-go reversal learning task. Stimulus-response rules were presented at the beginning of the task, and subjects were asked to either respond or withhold a response to the presented stimuli while receiving accuracy feedback. Halfway through the task, the stimulus-response rules were reversed. Performance was assessed by discriminability index (d'), hit rate (HR), and false alarm rate (FAR). Saliva samples were collected immediately prior, immediately after, and 30min after each MAST and assayed for cortisol. One saliva sample from each subject was assayed for CRHR1 genotype.

Results: CRHR1 genotypes were in Hardy-Weinberg equilibrium (χ^2 =2.97, p=0.08). Mixed effects ANOVA with fixed effects of CRHR1 genotype, pre/post-reversal, and their interaction found a significant CRHR1 by reversal interaction for d' ($F_{2,319}$ =3.88, p=0.022) and HR ($F_{2,319}$ =3.16, p=0.044) following a night of stress and TSD. No such interaction was found at well-rested baseline (d': $F_{2,319}$ =2.51, p=0.083; HR: $F_{2,319}$ =1.55, p=0.213). Subjects homozygous for the T allele had higher mean post-MAST cortisol levels (0.40±0.06 µg/dL) with better pre-reversal performance, but worse post-reversal performance compared to heterozygous and homozygous G allele carriers.

Conclusion: CRHR1 genotype modulates dynamic decision making following a night of acute stress and TSD. A higher cortisol stress response (T/T genotype) is beneficial to maintaining task relevant information (stability), but significantly impairs the ability to update task-relevant information following a change in situational demands (flexibility).

Support: CDMRP grant W81XWH-17-C-0088

0307

GRAY MATTER VOLUME CORRELATES OF PSYCHOMOTOR VIGILANCE SPEED DURING SLEEP DEPRIVATION

Meinhausen, C. E. Vanuk, J. R. Grandner, M. A. Killgore, W. D. Department of Psychiatry, The University of Arizona College of Medicine, Tucson, AZ.

Introduction: Sleep deprivation has often been associated with decreased cognitive control, including deficits in the ability to sustain attention. Psychomotor vigilance speed slows following a period of fatigue, and can lead to disastrous results in daily life. In order to determine the brain areas correlated with reduced psychomotor vigilance speed, as a result of diminished sleep, a voxel-based morphometry analysis was performed prior to a period of monitored sleep deprivation. The mean speed of response time during the final 17 hours of a 29-hour sleep deprivation was then measured with the Psychomotor Vigilance Test (PVT), a reaction-timed task that measures the speed participants respond to a visual stimulus. Methods: 45 healthy individuals (male=23 female=22) between the ages of 20-43 years (M=25.4 SD=5.6) participated in the study. Structural neuroimaging data were collected using a T3 magnetic resonance imaging scanner following a typical night's sleep. Mean PVT speed was monitored with an hourly 10-minute PVT assessment during a monitored overnight sleep deprivation session. Speed was defined as the reciprocal of reaction time (1/RT).

Results: PVT speed was negatively correlated with grey matter volume (P<.05 FWE-corrected) in the prefrontal cortex, specifically the right posterior inferior frontal gyrus (p=.030; MNI coordinates = 36, 12, 26). **Conclusion:** Our findings indicate that gray matter within the right posterior inferior frontal gyrus is greater in individuals who are more vulnerable to slowing of PVT responses during an overnight period of sleep deprivation. These findings suggest that inter-individual

differences in the ability to sustain psychomotor vigilance during sleep loss may be related to increased gray matter in the right lateral prefrontal cortex and could have implications for understanding the neurobiological substrates of vulnerability and resilience to sleep loss. **Support:**

0308

DRD2 C957T GENOTYPE INFLUENCES VIGILANT ATTENTION PERFORMANCE IMPAIRMENT DURING TOTAL SLEEP DEPRIVATION

Muck, R. A.^{1,2} Skeiky, L.^{1,2} Schmidt, M. A.^{1,2} Satterfield, B. C.^{1,2} Wisor, J. P.^{1,2} Honn, K. A.^{1,2} Van Dongen, H.^{1,2}

¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA.

Introduction: There are substantial, phenotypical individual differences in the adverse impact of total sleep deprivation (TSD) on vigilant attention performance. Dopaminergic genotypes have been found to contribute to these phenotypical differences. Here we investigated the association between a single nucleotide polymorphism (SNP) of the dopamine receptor D2 (DRD2) gene, C957T (rs6277), on vigilant attention performance measured with the psychomotor vigilance test (PVT) in a laboratory TSD study.

Methods: N=46 healthy adults (ages $26.0\pm5.3y$; 25 females) completed a 4-day in-laboratory study with a baseline day (10h time in bed: 22:00-08:00), a 38h TSD period, and a recovery day (10h time in bed: 22:00-08:00). DNA isolated from whole blood was assayed for DRD2 C957T genotype using real-time polymerase chain reaction. PVT performance was measured during TSD at 2-4h intervals, and analyzed for genotype using mixed-effects analysis of covariance of lapses of attention (RTs>500ms).

Results: The genotype distribution in this sample - 28.3% C/C, 50.0% C/T, 21.7% T/T - was found to be in Hardy-Weinberg Equilibrium (X^2_1 =0.0008, p=0.98). As expected, there was a significant effect of time awake on PVT performance ($F_{14.602}$ =26.67, p < 0.001). There was a significant main effect of DRD2 genotype $(F_{2,602}=3.24, p=0.040)$ and a significant interaction of time awake by DRD2 genotype ($F_{28,602}$ =1.96, p=0.003). Subjects homozygous for the T allele showed greater impairment during extended wakefulness than carriers of the C allele. Genotype explained 7.6% of the variance in the PVT data observed during the 38h TSD period. Conclusion: Subjects homozygous for the T allele of DRD2 C957T were considerably more vulnerable to TSD-induced PVT performance impairment than carriers of the C allele. A recent study showed that DRD2 C957T influences PVT performance in interaction with a SNP of the DAT1 gene. Here, DRD2 genotype was by itself also associated with PVT performance impairment during TSD.

Support: CDMRP awards W81XWH-16-1-0319 and W81XWH-18-1-0100.

0309

STRESS INDUCED NITRIC OXIDE SYNTHASE ACTIVATION IN THE BASAL FOREBRAIN IS A RESULT OF SLEEP LOSS

Chiem, E. Nichols, I. Paul, K.

University of California, Los Angeles, Los Angeles, CA.

Introduction: The mechanisms underlying the reciprocal relationship between stress and sleep are unclear. Nitric oxide, a diffusible signaling molecule, plays an important role in physiological stress responses and sleep. The medial septum (MS) and vertical diagonal band (VDB) are sleep regulatory regions of the basal forebrain whose cells express nitric oxide synthase (NOS). In this study, we examined the effects of sleep loss and restraint stress on NOS activation in the MS and VDB. **Methods:** Adult male and female C57BL/J6 mice were randomly assigned to a control, total sleep deprivation (TSD), or restraint stress group. TSD was performed for 6h using gentle handling, and restraint was performed for 6h by immobilizing the mice in a plastic restraint device. Both procedures began at light onset in a 12:12 light:dark cycle. Immediately following the procedures, mice were sacrificed, and NADPH-diaphorase (NADPH-d) was measured in the MS and VDB to determine NOS activity.

Results: A multivariate ANOVA revealed main effects of TSD and restraint stress on NADPH-d in the MS (F(2,13) = 7.0921, p = 0.011) and VDB (F(2,13)=6.416, p = 0.014) in females. A *post hoc* analysis showed a significant difference between control and TSD (p = 0.011 (MS), p = 0.014 (VDB)), and between control and restraint (p = 0.032 (MS), p = 0.048 (VDB)), but no significant difference between TSD and restraint. There is a sex difference in NADPH-d in these regions (p < 0.005) that reverses direction following TSD and restraint stress. **Conclusion:** Our findings provide evidence that NOS activity in the basal forebrain may underlie sex differences in stress responses. Since there is no significant difference between the TSD and restraint stress, this suggests that the effect of restraint stress on NOS activation is a result of sleep loss, and not due to induction of a stress mechanism. **Support:** This study was partly funded by R01-NS078410 and UCLA start-up funds.

0310

OCULOMETRICS TRACK PERFORMANCE ON THE PSYCHOMOTOR VIGILANCE TASK DURING ACUTE SLEEP DEPRIVATION

*Feick, N. H.*¹ *Tyson, T. L.*² *Arsintescu, L.*¹ *Cravalho, P. F.*¹ *Stone, L. S.*² *Flynn-Evans, E. E.*²

¹San Jose State Research Foundation, San Jose, CA, ²NASA Ames Research Foundation, Moffett Field, CA.

Introduction: Sleep deprivation and circadian misalignment impairs human sensorimotor performance and reduces vigilant attention, which increases the potential for errors in occupations that require 24-hour operations. The psychomotor vigilance task (PVT) is the gold-standard measure for evaluating the impact of sleepiness on performance, however, it is not practical to administer in many operational environments, because it only provides a snapshot of performance and requires an individual to focus on the task for several minutes, multiple times over a work shift. As a result, passive, continuous monitoring of sleepiness is desirable for operational environments. The goal of the present study was to determine if complex oculomotor behavioral metrics track PVT performance during sleep deprivation.

Methods: Twelve healthy adults (mean age 24.8 ± 5.4 years; 6F) maintained a fixed schedule with 8.5 hours in bed for two weeks, during which they abstained from caffeine, alcohol, and other medications, followed by a ~24 hours constant routine laboratory stay. Participants completed the PVT and a radial step-ramp ocular tracking task hourly throughout the study. Twelve oculometrics were derived from smooth pursuit and saccadic eye movements collected through video-oculography and were compared to the PVT and Karolinska Sleepiness Scale (KSS) using linear regression and receiver operating characteristic curves.

Results: Nine oculometrics spanning pursuit, saccade, and directional motion processing performance correlated with the PVT and KSS (p < 0.05), including: (a) pursuit latency; (b) open-loop pursuit acceleration; (c) proportion smooth; (d) steady-state pursuit

gain; (e) saccadic amplitude; (f) saccadic dispersion; (g) saccadic rate; (h) direction asymmetry; and (i) direction noise.

Conclusion: The oculometrics that we examined exhibited a distinct pattern that tracked PVT performance. Future studies should examine whether these metrics can be extracted through passive monitoring techniques.

Support: None

0311

THE IMPAIRMENTS IN EMOTIONAL PERCEPTION FOLLOWING TOTAL SLEEP DEPRIVATION AND THE RESTORATIVE EFFECTS OF NAPPING AND TIME OF DAY

Cunningham, T. J.¹ Bottary, R. M.² Kensinger, E. A.² Stickgold, R.¹ ¹Beth Israel Deaconess Medical Center, Boston, MA, ²Boston College, Boston, MA, ³Beth Israel Deaconess Medical Center, Boston, MA.

Introduction: The ability to perceive emotions is a socially-relevant skill critical for healthy interpersonal functioning, while deficits in this ability are associated with psychopathology. Total sleep deprivation (TSD) has been shown to have deleterious effects on emotion perception, yet the extent to which these impairments persist across the day with continued wakefulness, or if brief periods of recovery sleep can restore emotion perception abilities, remains unexplored. Methods: Participants viewed slideshows of faces ranging in emotional expression and were asked to categorize (Happy, Sad, Angry, Neutral) and rate the emotional intensity (1-9) of each face at baseline (2100; Session 1), at 0900 (Session 2) following a night of sleep or TSD, and at 1400 (Session 3) following either continued wakefulness (wake group) or a 90-minute nap opportunity (nap group). Results: Emotion categorization ability marginally improved from Session 1 to Session 2 following overnight sleep, however, no changes in emotion intensity ratings or vigilance were observed. TSD led to an increase in error rates during vigilance testing [t(46)=2.9, p=0.005] and impairment in emotion categorization ability [t(46)=5.5, p<0.001] from Session 1 to Session 2, although by Session 3 performance levels on both measures returned to baseline for all TSD participants. TSD also led to a decrease in emotional intensity ratings from Session 1 to Session 2, particularly for the highest tertile of emotional faces [6-9; t(46)=6.1, p<0.001]. These ratings remained suppressed at Session 3 in both the wake [t(25)=7.8, p<0.001] and nap [t(18)=3.1, p=0.006] groups.

Conclusion: These results indicate that time of day effects, with or without any additional benefit of a nap, can restore the impairments in vigilance and emotional categorization caused by TSD. The ability to discriminate levels of emotional intensity, however, is not restored by time of day or napping, suggesting that this ability is more sensitive to the impact of TSD. **Support:**

0312

PERFORMANCE ON A COGNITIVE INTERFERENCE TASK IN CHILDREN AFTER ONE-NIGHT OF SLEEP RESTRICTION

de Queiroz Campos, G.¹ Dickstein, D. P.¹ Carskadon, M. A.¹ Saletin, J. M.¹

¹Brown University, Providence, RI, ²Brown University, Providence, RI.

Introduction: Short sleep contributes to attention failure in conditions such as ADHD. Whether sleep loss affects attentional processes as a task varies in cognitive interference is unclear. We used a multi-source interference task (MSIT) in a sleep restriction paradigm in children with a range of ADHD symptoms to examine how short sleep disrupts attention in these youth.

Methods: Thirteen children (7F, 11.7 ± 1.28 years) with a range of ADHD symptom severity completed a repeated-measures experiment on two consecutive nights in the laboratory: baseline (BSLN; 9.5h time-in-bed) and sleep restriction (SR; 4h time-in-bed). Each morning they took part in an fMRI session including the MSIT, in which participants respond to a series of 3-digit numbers by indicating which digit is different on no-interference (e.g., 003; correct=3) or interference (e.g., 311, correct=3) trials. Performance measures were inverse reaction time (1/RT) and accuracy. A two-way within-subject ANOVA assessed performance across interference and sleep conditions respectively.

Results: 1/RT showed main-effects of sleep loss (BSLN vs. SR; $F(1,148)=4.01;p<0.05;\eta^2=0.026$) and trial type (no-interference vs. interference; F(1,148)=24.7;p<0.001; $\eta^2=0.143$). Responses were slower for interference (BSLN RT: 799.3ms, SR RT: 895.8ms) than no-interference (BSLN RT: 653.2ms, SR RT: 697.4ms) trials. No interaction between interference and sleep loss was found ($F(1,148)=0.11;p>0.05;\eta^2=0.001$). Likewise, accuracy was lower (F(1,148) = 31.1, p<.001; $\eta^2=0.174$) in interference trials (73.5%) than in no-interference trials (92.2%), however with no effect of sleep loss, nor an interaction of interference and sleep loss (all p's > .05).

Conclusion: These data provide evidence that partial sleep loss disrupts attention processes in children, yet these differences do not appear to depend on cognitive interference in our sample. Future analyses will examine whether ADHD symptoms distinguish individual differences, as well as analyze fMRI data to probe neural processes underlying attention control.

Support: K01MH09854 (to JMS); Brown University UTRA (to GDQC).

0313

IS HEALTHY SLEEP POSSIBLE FOR PROFESSIONAL FIREFIGHTERS? A COMPARISON OF "ON-DUTY" AND "OFF-DUTY" SLEEP QUANTITY AND QUALITY

Dautovich, N. D. Dzierzewski, J. Sabet, S. Soto, P. Donovan, E. Kleva, C.

Virginia Commonwealth University, Richmond, VA.

Introduction: Healthy sleep is vital for firefighter safety, health, wellness, and for public well-being. However, professional firefighters experience disturbed sleep at disproportionately high rates. The current study investigated whether firefighters can obtain healthy sleep by identifying (1) differences in sleep while "on-duty" and "off-duty" and (2) risk factors for poor sleep.

Methods: Professional firefighters in Richmond, Virginia's Department of Fire and Emergency Services (N=268), reported their sleep using the Pittsburgh Sleep Quality Index (PSQI) both on- and off-duty. Good and poor sleepers were identified using the PSQI global score cutoff of 5. Demographic and STOP-BANG questionnaires were also completed.

Results: Using a repeated measures MANOVA, on-duty sleep was significantly worse compared to off-duty sleep across PSQI component and global scores $F(7, 253)=45.24 p < .001, \eta^2=.56$. On-duty, 76.1% of firefighters were classified as poor sleepers compared to 42.9% off-duty. 34.7% were reclassified as good sleepers or stayed good sleepers (22.4%) when off-duty. A sizeable minority experienced consistently poor sleep while on- and off-duty (41.4%), and a small number reported worse sleep when off-duty (1.5%). More night calls and poorer self-rated mental health predicted worse

on-duty sleep (p<.001) and poorer self-rated mental health predicted worse off-duty sleep (p<.001).

Conclusion: Healthy sleep is possible for professional firefighters. Almost a quarter of the sample was classified as "good sleepers" on-duty and over half were classified as "good sleepers" off-duty. Nonetheless, sleep on-duty was significantly worse overall, with over a third of the sample experiencing consistently poor sleep. When working a 24-hour variable shift schedule, it appears that poor sleep may "carryover" from on-duty to off-duty. Poorer self-rated mental health and more night calls were identified as risk factors. Further research is needed to probe risk and protective factors within this population. **Support:** N/A

0314

RESILIENCE TO INHIBITORY DEFICITS DURING SLEEP DEPRIVATION IS PREDICTED BY GRAY MATTER VOLUME IN THE VENTROLATERAL AND VENTROMEDIAL PREFRONTAL CORTEX

Killgore, W. D.¹ Dailey, N. S.¹ Raikes, A. C.¹ Vanuk, J. R.¹ Taylor, E.¹ Grandner, M. A.¹ Alkozei, A.¹

¹University of Arizona, Tucson, AZ, ²University of Arizona, Tucson, AZ.

Introduction: Stable, trait-like inter-individual resilience to sleep loss has been demonstrated for psychomotor vigilance, mood, subjective sleepiness, and some reasoning tasks, some of which have been associated with specific genetic or neurobiological markers. Resilience to executive control deficits induced by sleep deprivation (SD) has not been explored in terms of neurobiological markers. We, therefore, collected magnetic resonance imaging (MRI) scans of healthy individuals when well rested and correlated gray matter volume with resistance to inhibitory declines during 29 hours of SD.

Methods: Forty-five healthy individuals (22 female) ranging in age from 20 to 43 underwent structural MRI. Within 2-4 days after scanning, participants returned to the lab to undergo one night of SD, during which they completed a standard go/no-go task involving inhibitory processing every 4 hours. Scores were calculated as throughput (correct responses per working minute). The difference between performance in the evening (22:45) versus the performance the next morning (06:45) was calculated as an index of "inhibitory resilience." Gray matter volume was regressed against the inhibitory resilience measure. Based on prior research, regions were constrained to the ventrolateral prefrontal cortex and ventromedial prefrontal cortex.

Results: Greater resilience against declines in inhibitory capacity during SD was predicted by 1) larger gray matter volume within the ventrolateral prefrontal cortex and 2) reduced volume within the ventromedial prefrontal cortex (p<.05, FWE cluster corrected). These two clusters contributed significant unique explanatory variance to the model (R^2 =.45, p<.0001).

Conclusion: The ability to sustain performance during an inhibitory go/no-go task during SD was predicted by greater gray matter volume within the ventrolateral prefrontal cortex, a region that has been previously associated with inhibitory capacity, and reduced volume within an area of the ventromedial prefrontal cortex, which is often related to the default mode network. Findings suggest that specific brain networks may confer taskspecific resistance to SD.

Support: Defense Advanced Research Projects Agency, DARPA Young Faculty Award: DARPA-12-12-11-YFA-FP-029

XI. Sleep Deprivation, Loss and Disruption

0315

GRAY MATTER VOLUME OF THE ROSTRAL MEDIAL PREFRONTAL CORTEX IS ASSOCIATED WITH RESILIENCE TO MOOD DECLINE DURING OVERNIGHT SLEEP DEPRIVATION

Anlap, I.¹ Taylor, E.¹ Grandner, M. A.¹ Killgore, W. D.¹ ¹University of Arizona, Tucson, AZ, ²University of Arizona, Tucson, AZ.

Introduction: Vulnerability to sleep deprivation (SD) has been attributed to inter-individual trait-like differences in the ability to sustain vigilance and subjective alertness, which may have distinct neurobiological substrates. We have previously shown that reduced suppression of the Default Mode Network (DMN) during a cognitive task was predictive of global vulnerability to SD. However, little is known about vulnerability to mood decrements during SD and the underlying neurobiological mechanisms. Using voxelbased morphometry (VBM), we assessed structural differences in gray matter volume (GMV) of a region of the anterior DMN, the medial prefrontal cortex and its association with self-reported mood during 29 hours of SD.

Methods: 45 healthy participants (23 male; Ages 20-43) underwent 3T structural magnetic resonance imaging (MRI). Within 4 days, participants underwent an overnight SD session (29 hours awake total) which included hourly mood assessments with several visual analog mood scales (VAMS) assessing positive and negative affect. Hourly VAMS data were converted into a comparative metric of percent worsening of mood scores from 19:00 until noon the next day. These scores were averaged to determine a "mood resilience" score, with higher scores indicating greater mood sustainment. Using SPM12, the mean mood resilience scores were correlated with whole-brain gray matter volume, restricted to the medial prefrontal cortex, p<.05, FWE corrected, with a cluster threshold of 137 voxels. **Results:** Overnight mood resilience was significantly correlated with greater grey matter volume in right rostral medial prefrontal cortex (p<.05, corrected; k=137).

Conclusion: Individuals with greater gray matter volume within a circumscribed region of the right medial prefrontal cortex demonstrated greater resilience to mood degradation over 29 hours of continuous wakefulness. This same region of the brain has been shown to be critical for the passive maintenance of emotions. We speculate that greater GMV could protect against mood decline by better sustaining emotional state during SD.

Support: Defense Advanced Research Projects Agency Young Faculty Award: DARPA-12-12-11-YFA11-FP-029

0316

EMOTION REGULATION DURING SLEEP DEPRIVATION AND REPEATED PHYSIOLOGICAL STRESS: IMPLICATIONS FOR MOTOR SKILL LEARNING AND PRODUCTION

LaFollette, K. Satterfield, B. C. Esbit, S. Lazar, M. Grandner, M. A. Killgore, W. D. University of Arizona, Tucson, AZ.

Introduction: The ability to perform learned motor procedures under stress is a critical skill for many high-risk occupations. Explicit motor skills require top-down cognitive control, which both sleep loss and stress have been found to produce significant degradations, whereas implicit skills rely less on cognitive control and are more resilient to physiological stress. We investigated whether differences in emotion regulation attenuated the effects of sleep deprivation (SD) and acute stress on discrete motor learning. **Methods:** 45 adults (21 F; 22 ± 3.4 years) participated in 28-hours of in-lab SD. Participants completed repeated batteries that included the Maastricht Acute Stress Test (MAST) Karolinska Sleepiness Scale (KSS), Psychomotor Vigilance Test (PVT), and Discrete Sequence Production Task (DSP). Stress response was quantified by salivary cortisol. We quantified DSP motor performance by total accurate sequences, and average movement time on accurate trials. Ability emotional intelligence (EI) was measured with the MSCEIT, while trait EI was measured with the Bar-On EQI. The CD-RISC was included as a measure of resilience.

Results: Using linear mixed effects models of motor performance indices, we found subjective, trait-based emotional intelligence (EQI) to be associated with worse motor performance over time, and objective, ability-based emotional intelligence (MSCEIT) to be associated with greater movement speed. We further found that greater psychological resilience (CD-RISC) but not emotional intelligence was predictive of stronger and less variable chunking structures during SD.

Conclusion: Emotional intelligence can influence motor learning under stressful SD, whereas psychological resilience can safeguard learning. Future work should further investigate how trait and ability metrics of EI have opposing effects on responses to stress under SD. Work in this direction could serve to identify difference factors that bolster motor skill production in operational environments where stress and SD are unavoidable.

Support: US Army Medical Research and Development Command: W81XWH-17-C-0088

0317

IMPACT OF RECURRENT PARTIAL SLEEP LOSS COMBINED WITH ACUTE EXERCISE ON CIRCULATING AND ADIPOSE TISSUE LEVELS OF LEPTIN, ADIPONECTIN AND GHRELIN IN HEALTHY YOUNG INDIVIDUALS

Schéle, E.¹ Dickson, S.¹ Benedict, C.² Cedernaes, J.² ¹University of Gothenburg, Gothenburg, SWEDEN, ²Uppsala University, Uppsala, SWEDEN, ³Uppsala University, Uppsala, SWEDEN.

Introduction: Chronic sleep loss and aerobic exercise have opposing effects on body weight maintenance. The effects of sleep loss on circulating levels of the orexigenic hormone ghrelin and the anorexigenic hormone leptin have been extensively studied. In contrast, how these changes interact with acute exercise, and whether these changes are reflected at the tissue level, remains poorly understood.

Methods: In a randomized, 2 session, crossover design, 16 normalweight young men were studied following three nights of partial sleep deprivation (4.25 hr sleep opportunity each night) and three nights of normal sleep (8.5-h sleep opportunity), monitored in a sleep laboratory. Each condition was followed by 30 min of intense morning ergometer cycling. Plasma levels of leptin and ghrelin, as well adipose tissue mRNA levels of leptin and adiponectin, were measured before and after each exercise intervention.

Results: In response to acute exercise, circulating levels of both leptin (ANOVA time effect: P<0.001) and ghrelin (time: P<0.001) decreased immediately (+15 min), and remained significantly lower +30, +60 and +240 min post exercise for leptin (all P<0.05), and up until +30 min post exercise for ghrelin. These effects were seen regardless of sleep condition (ANOVA sleep condition: P>0.10). In adipose tissue, mRNA expression of leptin and adiponectin was not different between the sleep conditions (ANOVA sleep condition: P>0.10). In contrast, mRNA levels of leptin decreased (P=0.017), whereas adiponectin mRNA increased (P=0.010) 3.5 hours post vs. pre exercise. The decrease in leptin in response to exercise appeared to mainly occur following sleep loss (P=0.066) and not after normal sleep (P=0.38).

Conclusion: Our results suggest that both circulating and adipose tissue levels of appetite-regulating hormones are altered in response to acute aerobic exercise, in a manner that does not depend on prior sleep history. Whether these findings apply to older, female, or metabolically ill individuals - and whether they may differ in response to circadian misalignment, or evening exercise - remains to be established.

Support: The Swedish Society for Medical Research, the Swedish Society of Medicine, the Swedish Research Council and the Göran Gustafsson; Swedish Brain; Åke Wiberg and NovoNordisk Foundations.

0318

PROSPECTIVE ASSOCIATION OF ACTIGRAPHY-ASSESSED SLEEP WITH PHYSICAL GROWTH IN THE FIRST 6 MONTHS OF LIFE

Li, X.^{1,2} Rueschman, M.¹ Kaplan, E. R.² Yu, X.^{2,3} Davison, K.^{1,3} Redline, S.¹ Taveras, E. M.⁴

¹Division of Sleep and Circadian Disorders, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, ²Department of Social and Behavioral Sciences, Harvard T.H. Chan School of Public Health, Boston, MA, ³School of Social Work, Boston College, Boston, MA, ⁴Division of General Academic Pediatrics, Massachusetts General Hospital for Children, Boston, MA.

Introduction: Suboptimal sleep is associated with weight gain and related chronic diseases in adults, adolescents, and older children. However, little is known regarding the associations between sleep and physical growth in infants. We investigated prospectively the associations between objectively-measured sleep patterns at 1 month and physical growth in the first 6 months of life.

Methods: We studied 344 full term infants in the ongoing longitudinal Rise & SHINE (Sleep Health in Infancy & Early Childhood) birth cohort study. At 1 month, infants underwent 7-day ankle actigraphy, estimating average sleep duration (24-hour, nighttime, and daytime) and sleep fragmentation (number of nighttime awakenings). Weight and length were measured at birth and 6 months and used to calculate weight-for-length z (WFL-z) scores. We used linear and logistic regression analyses to examine the associations between sleep patterns at 1 month with WLF-z at 6 months and rapid weight gain from birth to 6 months, defined as an increase in WFL-z greater than or equal to 0.67, controlling for covariates.

Results: Each 1-hour increase in 24-hour sleep duration was associated with a 0.07-unit (95% CI [0.01, 0.12]) increase in WFL-z at 6 months. Daytime, but not nighttime, sleep duration was positively associated with WFL-z. Greater number of nighttime awakenings was associated with higher WFL-z (beta = 0.28; 95% CI [0.08, 0.49]). 24-hour and nighttime sleep duration were positively associated with a 18.5% (95% CI [1.04, 1.35]) and a 23.4% (95% CI [1.02, 1.49]) higher odds of rapid weight gain from birth to 6 months, respectively.

Conclusion: Longer 24-hour sleep duration was associated with higher 6-month WFL-z and more rapid increases in WFL-z from birth to 6 months. Greater nighttime sleep fragmentation was associated with higher 6-month WFL-z. Sleep at 1 month might provide modifiable targets to help avoid lifetime complications of excess weight.

Support: R01DK107972.

0319

SLEEP ARCHITECTURE AND NEUROCOGNITIVE AND BEHAVIORAL FUNCTIONING IN YOUTH FROM THE GENERAL POPULATION

Ricci, A.¹ Fang, J.¹ He, F.¹ Cain, P.¹ Calhoun, S. L.¹ Liao, D.¹ Vgontzas, A. N.¹ Bixler, E. O.¹ Fernandez-Mendoza, J.¹ ¹Penn State College of Medicine, Hershey, PA, ²Penn State College of Medicine, Hershey, PA.

Introduction: The transition from childhood to adolescence is critical for the onset of psychopathology and reflects significant

changes in the sleeping brain. Sleep deprivation studies have shown that rapid eye movement (REM) and non-rapid eye movement (NREM) sleep are differentially involved in specific cognitive functions. The aim of this study was to examine the association of sleep architecture with neurobehavioral outcomes in a population-based sample.

Methods: We studied 700 children (5-12y, 47.1% female, 23.7% minority) and 421 adolescents (12-23y, 46.1% female, 21.8% minority) from the Penn State Child Cohort. All subjects underwent a 9-hour polysomnography and a 4-hour neurobehavioral evaluation. Neurocognitive outcomes included the Stroop test, digit span backwards (DSB), and coding to measure high- and low-order cognitive functions. Behavioral outcomes included the Child/Adult Behavior Checklist to measure internalizing symptoms and externalizing behaviors. Correlation analysis examined the cross-sectional association between sleep architecture and neurocognitive and behavioral outcomes.

Results: In childhood, %REM sleep was negatively associated with DSB scores (r=-0.088, p=0.027), particularly in males (r=-0.167, p=0.002). Furthermore, %NREM sleep was positively associated with DSB scores in males (r=0.126, p=0.021). In adolescent females, %NREM and %REM sleep were positively (r=0.146, p=0.044) and negatively (r=-0.158, p=0.029) associated with DSB scores, respectively. In adolescence, %NREM sleep was negatively associated with internalizing symptoms (r=-0.109, p=0.026).

Conclusion: Male children and female adolescents who spent a higher proportion of the night in NREM sleep had better working memory performance. Adolescent females who spent a lower proportion of the night in NREM sleep had greater internalizing symptoms. This study suggests a role for sleep architecture in neurobehavioral deficits in youth. Future studies are necessary to determine the contributions of low- and high-frequency sleep EEG dynamics to these clinical outcomes.

Support: National Institutes of Health (R01MH118308, R01HL97165, R01HL63772, UL1TR000127)

0320

PSYCHOLOGICAL CORRELATES OF MORNINGNESS/ EVENINGNESS IN LATINX PRE-ADOLESCENTS

Nguyen-Rodriguez, S. T.¹ Buxton, O. M.^{2,3,4}

¹California State University, Long Beach, Long Beach, CA, ²Pennsylvania State University, University Park, PA, ³Brigham and Women's Hospital, Boston, MA, ⁴Harvard Medical School, Boston, MA.

Introduction: Chronotype refers to a preference for morning hours (morningness) vs. evening hours (eveningness) when individuals tend to feel their best (e.g., higher energy levels). People may be classified at either end of this spectrum or along a continuum between these preferences. Among adolescents, eveningness is positively related to depression and anxiety, whereas morningness is negatively related to depression. However, less is known about the relationship of chronotype and psychological health in preteens and Latinx youth. The present study explored associations of morningness/eveningness with anxiety symptoms, depressive symptoms, and perceived stress among Latinx pre-adolescents in Southern California.

Methods: A purposive sample of 100 Latinx children, ages 10-12 years old, completed self-report surveys in their homes or

a preferred location chosen by the parent. Measures included the Morningness/Eveningness Scale for Children (higher scores indicate morning preference), Revised Child Anxiety and Depression Scale and the Perceived Stress Scale (higher scores indicate higher anxiety, depression and stress, respectively). Associations were tested with Pearson correlations.

Results: The sample was 47% male with a mean \pm SD age of 10.9 \pm 0.8 years. Average score for morningness/eveningness was M=30.2 \pm 4.4 (range: 18-41), for anxiety symptoms was M=0.7 \pm 0.7 (range: 0-2.8), for depression symptoms was M=0.5 \pm 0.4 (range: 0-1.9) and for perceived stress was M=15.2 \pm 5.8 (range: 2-30). Greater morningness/eveningness scores, indicating more morningness, were associated with lower scores for anxiety symptoms (r=-.41, p<.001), depressive symptoms (r=-.36, p<.001) and perceived stress (r=-.33, p=.001).

Conclusion: As has been found for adolescents, higher morningness in Latinx pre-teens was related to less frequent anxiety and depression symptoms, as well as lower perceived stress. Youth experience a circadian phase delay during adolescence, shifting their preference toward eveningness, which may exacerbate stressors and negative mental health. Therefore, interventions to promote psychological well-being in pre-adolescents may help prevent worse psychological outcomes in Latinx children as they transition to adolescence.

Support: This work was supported by the National Institute of General Medical Sciences of the National Institutes of Health under Award Numbers UL1GM118979, TL4GM118980, and RL5GM118978.

0321

INCREASED SLOW-WAVE ACTIVITY PREDICTS SLOWER PROCESSING SPEED IN TODDLERS

*Waddle, A. E.*¹ *Kurth, S.*² *Harsh, J.*¹ *Lassonde, J. M.*¹ *Lee, D.*¹ *LeBourgeois, M. K.*¹

¹University of Colorado Boulder, Boulder, CO, ²University Hospital Zurich, Zurich, SWITZERLAND.

Introduction: Slow-wave activity (SWA) shows an inverted U-shaped time course during development. Specifically, maximal SWA undergoes a posteroanterior shift from 2 to 20 years of age, which may reflect cortical maturation. Previously, we showed that greater slow sigma power during sleep predicted faster reaction time in preschool-aged children. To date, little is known about the relationship between SWA and processing speed (PS), a basic fundament underlying complex cognitive skills in early development.

Methods: This project examined the relationship between SWA and PS in 2.5-3.0-year-old children (n=26, 50% males) via homebased assessments. After a 5-day stabilization sleep schedule, a baseline sleep EEG recording was performed on participants at 4 electrode placements: Fz, Oz, C3, and C4. SWA EEG spectral power was quantified in the 0.75-4.5 Hz rangeduring the first 60 minutes of NREM sleep. PS was obtained as part of a standard cognitive assessment via a computer-based task one hour after waking from a midday nap. **Results:** On average, reaction time (PS) was 2111 ± 08 ms and SWA was $856.4 \pm 300.7 \,\mu\text{V}^2/\text{Hz}$. Increased SWA in the occipital region was predictive of a longer reaction time and therefore slower PS (r = 0.44, p = 0.03). This relationship showed differences between sexes, suggesting that females (r = 0.26, p = 0.07) may show a stronger association between SWA in the occipital brain region and PS than males (r = 0.09, p = 0.33).

Conclusion: Interestingly, these findings contradict our hypothesis based on previous data with older children indicating that greater SWA was associated with more advanced behavioral and cognitive skills. This discrepancy may reflect the stark individual differences present within this rapidly maturing age group.

Support: Research support from NIH R01-MH086566 to MKL.

0322

NAPPING, INHIBITORY CONTROL, AND SELF-REGULATION IN 2-YEAR-OLD CHILDREN

Shalowitz, E. L.¹ Miller, A. M.² Harsh, J. R.¹ LeBourgeois, M. K.¹ ¹University of Colorado, Boulder, CO, ²The University of Michigan, Ann Arbor, MI, ³University of Colorado, Boulder, CO.

Introduction: Poor sleep in early childhood is linked to reduced school readiness. This study examined the role of acute sleep loss in behavioral self-regulation using a delay of gratification task. We hypothesized that after acute nap deprivation, toddlers would have worse inhibitory control and resort to more maladaptive self-regulation strategies than after a nap.

Methods: 25 healthy children (11 males, 34.1 ± 2.3 months-old) followed a strict sleep schedule for ≥ 5 days before a baseline (nap) and an acute nap deprivation condition (no-nap). After being introduced to an age-appropriate toy, children were instructed not to touch the toy and left alone for 3-minutes. To assess inhibitory control, videos of the waiting period were behaviorally coded for latency to touch and 11 self-regulation strategies. We combined strategies into adaptive and maladaptive composites; higher scores on each composite indicated greater use.

Results: During the nap condition, 19 children touched the toy (latency to touch=70.0 \pm 60.7 sec); during the no-nap condition, 18 children touched the toy (latency to touch=65.4 \pm 71.6 sec). The adaptive composite score was 1.58 \pm 0.25 for the nap condition and 1.17 \pm 0.27 for the no-nap condition. The maladaptive score was 0.92 \pm 0.17 for the nap condition and 0.83 \pm 0.19 for the no-nap condition. We found no differences between conditions in the number of children who touched the toy (X^2 =0, p=0.50), mean latency to touch (t=0.27, p=0.39), or the composite scores of adaptive (z=0.35, p=0.12) and maladaptive strategies (z=0.09, p=0.69).

Conclusion: Findings indicate that acute nap deprivation may not have an immediate impact on inhibitory control and self-regulation in toddlers. 30-36 months old children may not have sufficient cognitive resources to exert inhibitory control and self-regulate whether or not they have obtained adequate daytime sleep. Future research should examine developmental changes in the effects of acute sleep restriction on behavioral self-regulation.

Support: Research support from NIH R01-MH086566 to MKL.

0323

ASSOCIATIONS BETWEEN SLEEP AND ACTIVITY PATTERNS IN THE MOTHER-CHILD DYAD

Driscoll, B. J.¹ Quattrucci, J.² Sharkey, K. M.¹ ¹Brown University, Providence, RI, ²Rhode Island Hospital, Providence, RI.

Introduction: Animal studies show links between light-dark patterns in gestating dams and offsprings' sleep and circadian rhythms. Activity patterns between mothers and infants show synchrony as early as 12 weeks postpartum. Our goal was to investigate maternal sleep/activity patterns at two points in the perinatal period and assess associations with activity in their young children.

Methods: Participants were 20 mother-child dyads recruited from previous studies. Mothers (age \pm SD = 31.7 \pm 5.5 years, range 21-40 years) wore wrist actigraphs during their 33rd week of pregnancy and 2nd week postpartum. Children (age \pm SD = 2.13 \pm 1.36 years, range 8 months-4.6 years) were assessed for 5 days and nights. Circadian patterns were analyzed using Maximum Entropy Spectral Analysis (MESA) to estimate best-fitting circadian period, tau. We used cosinor analysis to calculate rhythm amplitude, Midline Statistic of Rhythm (MESOR, representing mean activity), and acrophase (time of peak amplitude). We used circadian quotient (CQ; amplitude \pm MESOR) to assess rhythm strength while normalizing for intersubject variation in activity levels. Autocorrelation, or degree to which data is consistent for a particular period, was calculated to analyze regularity of activity patterns.

Results: Mothers' activity pattern autocorrelation was significantly correlated at the two time points (r=.530, p=.016), such that women with inconsistent activity patterns in pregnancy also demonstrated more irregularity at postpartum week 2. Child CQ correlated with age, with older children showing greater rhythm strength (r=.530, p=0.016). We observed a moderate correlation between mothers' CQs during pregnancy and children's CQs (r=.413,p=.07). In mother-child dyads, longer tau in mothers during pregnancy predicted lower autocorrelation of the child's rhythm to a 24-hr period (r=-.520, p=.019). Finally, later maternal acrophase at postpartum week 2 was associated with longer tau in children (r=.504, p=.024). **Conclusion:** These data show that associations between mother-child sleep/activity patterns may begin during pregnancy and support the notion that mothers' perinatal sleep patterns could affect the health of both mothers and their children.

Support: Supported by R34MH104377, K23MH086689, the Seleni Institute, the Depression and Bipolar Disorder Alternative Treatment Foundation, and a Karen T. Romer Undergraduate Teaching and Research Award from Brown University.

0324

EFFECT OF SLEEP RESTRICTION ON SLEEP ELECTROENCEPHALOGRAM WAVEFORMS IN ADOLESCENTS

Campbell, I. G.¹ Cruz Basilio, A.¹ Zhang, Z. Y.¹ Darchia, N.² Feinberg, I.¹

¹University of California, Davis Department of Psychiatry and Behavioral Sciences, Davis, CA, ²Ilia State University, Tbilisi, GEORGIA.

Introduction: Over the past 18 years, our laboratory has been carrying out longitudinal studies of sleep and sleep need across adolescence. Our current study uses a dose-response design to examine daytime performance and sleep EEG after varied sleep

durations. Here we present results for 1-30 Hz EEG power in NREM and REM sleep.

Methods: Home EEG recording in children 10-16 years old (N=77, mean age = 13.2). Adhering to their habitual rise time participants kept an assigned TIB schedule of 7, 8.5, or 10 hours for four consecutive nights. Participants completed all three conditions each year of the 3 year study. EEG recordings from the fourth night of each condition were scored and analyzed with FFT.

Results: Reducing TIB from 10 to 7 hours effectively decreased total sleep time (TST) from an average of 531 min to an average of 407 min. Decreasing TIB (from 10 to 7 h) produced a small increase (4.6%, p=0.0004) in delta (1-4 Hz) power and a larger decrease (9.0%, p=0.0032) in alpha (8-11 Hz) power in the first 5 h of NREM sleep. In REM periods 2 and 3, the same TIB reduction also increased (12.1%, p<0.0001) delta power and decreased (14.2%, p<0.0001) alpha power. Decreasing TIB reduced (11%, p<0.0001) sigma (11-15 Hz) power in the first 5h of NREM sleep and reduced (28%, p<0.0001) all night NREM sigma energy.

Conclusion: Reducing TST changes EEG power in several frequency bands. The increase in NREM delta power, expected from homeostatic models, may be too small to be biologically significant. The larger loss of sigma power may be of greater consequence. Sigma frequency activity is an indicator of sleep spindles which have been affected in aging, learning, memory and psychopathology. The sigma response to sleep restriction could be used to study these relations.

Support: PHS grant R01 HL116490 supported this work.

0325

BETTER AEROBIC FITNESS IS ASSOCIATED WITH DISTINCT SLEEP CHARACTERISTICS IN CHILDREN AND ADOLESCENTS - A PILOT STUDY

Neikrug, A. B. Radom-Aizik, S. Chen, I. Y. Stehli, A. Lui, K. K. Chappel-Farley, M. G. Lim, A. N. Mander, B. A. Benca, R. M. University of California Irvine, Irvine, CA.

Introduction: Aerobic fitness facilitates brain synaptic plasticity, which influences global and local sleep expression. While it is known that sleep patterns/behavior and non-rapid eye movement (NREM) sleep slow wave activity (SWA) tracks brain maturation, little is known about how aerobic fitness and sleep interact during growth and development in children and adolescents. The aim of this pilot study was to characterize relationships among aerobic fitness, measures of global/local sleep expression, and habitual sleep patterns in children and adolescents. We hypothesized that greater aerobic fitness would be associated with better sleep quality, indicated by increased SWA.

Methods: Twenty healthy youth (11-17 years-old, 11 female) were evaluated during summer vacation (no school schedule constraints). Aerobic fitness (VO_{2peak}) was measured using ramp-type progressive cycle ergometry, habitual sleep (i.e., sleep-time consistency and circadian activity patterns) was assessed with 7-day actigraphy, and *ad lib* sleep was evaluated during overnight polysomnography (PSG) with high-density electroencephalography (hdEEG; 128 channels). Spectral analysis was implemented to quantify SWA (0.5-4.5Hz). Data were analyzed using linear regression analyses and exploratory independent samples t-tests.

Results: Negative correlations were observed between VO_{2peak} and sleep measures including sleep-time consistency (partial r=-0.53, p=0.045) and timing/acrophase of the circadian activity rhythm (partial r=-0.64, p=0.01) while controlling for sex and age. Additionally, after accounting for Tanner stage and sex, data

demonstrated significant effects in SWA at frontal derivations (p=0.024) between low and high fitness levels at topographically specific and meaningful EEG derivations, e.g. over frontal cortex.

Conclusion: These results suggest that children and adolescents with greater fitness have less variability in sleep-times (improved sleep consistency), tend to have a more advanced circadian activity phase (i.e., go to sleep earlier), and express greater frontal SWA, supporting the hypothesis that fitness is associated with improved local and global sleep quality. Future research with larger samples is necessary to further evaluate these relationships, and to determine if interventions that improve fitness also improve sleep and related brain plasticity.

Support: NCATS grant #UL1TR001414 & PERC Systems Biology Fund

0326

DAILY SLEEP AND AFFECT IN ADOLESCENTS: DIFFERENTIAL ASSOCIATIONS DURING CONSTRAINED VERSUS UNCONSTRAINED SLEEP

Shen, L. Wiley, J. F. Bei, B.

Turner Institute for Brain and Mental Health, School of Psychological Sciences, Faculty of Medicine, Nursing and Health Sciences, Monash University, Melbourne, AUSTRALIA.

Introduction: Few studies have examined bi-directional associations between daily sleep and affect in adolescents, and even fewer assessed both high and low arousal affect under naturalistically-occurring constrained (school) and unconstrained (vacation) sleep opportunities.

Methods: 205 adolescents (54.1% females, age $M\pm SD=16.9\pm 0.87$ years) completed daily measures of sleep and affect over 28 continuous days (2-week school and 2-week vacation). Total sleep time (TST) and sleep efficiency (SE) were measured using actigraphy and sleep diary. High- and low-arousal positive and negative affect (PA, NA) were self-reported each afternoon. Cross-lagged, multilevel models were conducted: affect predicted same-night sleep controlling for previous-night sleep; sleep predicted next-day affect controlling for previous-day affect. Day of week, study day, and sociodemographics were controlled.

Results: During both school and vacation, adolescents with overall higher low-arousal PA also had greater self-reported SE. Other aspects of sleep-affect associations differ between school and vacation (all p<.05). During school, significant associations were between, not within individuals. Bi-directional associations were found between longer actigraphy-TST and greater high-arousal PA, and between higher self-reported SE and lower low-arousal NA. High-arousal PA and NA were associated with self-reported TST (positive and negative respectively). During vacation, between-individuals, higher NA was bi-directionally associated with lower self-reported SE, regardless of arousal. Longer self-reported TST was associated with lower low-arousal NA. On the within-person level, regardless of sleep measurement, nights with longer-than-average TST were associated with lower NA the next day (high- and low- arousal). Nights with higher-than-average SE predicted lower next-day low-arousal NA.

Conclusion: Sleep-affect associations differed based on sleep opportunity and arousal, suggesting potentially different mechanisms of action. When sleep is typically constrained, overall levels of sleep-affect associations were stronger than daily fluctuations. When sleep is typically unconstrained, significant associations were found both between- and within- persons. In particular, daily fluctuations in sleep were predictive of next-day NA, rather than the other direction.

Support: Monash International Postgraduate Research Scholarship and Monash Graduate Scholarship

0327

NREM SLEEP EEG IN TYPICALLY DEVELOPING AND DRUG-NAÏVE ADHD ADOLESCENTS: DATA FROM A LONGITUDINAL STUDY

Basishvili, T.¹ Eliozishvili, M.¹ Oniani, T.¹ Tchintcharauli, T.² Sakhelashvili, I.¹ Oniani, N.¹ Campbell, I. G.³ Feinberg, I.³ Darchia, N.¹

¹Ilia State University, T. Oniani Laboratory of Sleep-Wakefulness Cycle Study, Tbilisi, GEORGIA, ²Ilia State University, Child Development Institute, Tbilisi, GEORGIA, ³University of California, Davis, Department of Psychiatry and Behavioral Sciences, Davis, CA.

Introduction: Structural MRI studies suggest delayed brain maturation in children with attention deficit hyperactivity disorder (ADHD). The steep adolescent decline in sleep slow wave EEG activity provides an opportunity to investigate brain electrophysiological evidence for this maturational delay. Most ADHD sleep EEG studies have been cross-sectional. Here we present data from an ongoing longitudinal study of the maturational trajectories of sleep EEG in drug-naïve ADHD and typically developing adolescents.

Methods: Nine children diagnosed with ADHD (combined subtype, DSM-V criteria, mean age 12.39 ± 0.61 years), and nine typically developing controls (12.07 ± 0.35 years) were recruited. Subjects underwent an adaptation night and all night polysomnography twice yearly at the Laboratory. Sleep EEG was analyzed using fast Fourier transform. NREM delta and theta EEG activity were compared across first two recordings.

Results: Group effects (ADHD vs. control) on all night delta and theta energy, and delta power were not significant (p>0.2 for all). All night theta power was lower (p=0.035) for the ADHD group, and all night NREM sleep duration trended (p=0.060) toward being lower for the ADHD group. Controlling for sleep duration differences by examining only the first 5 h of NREM sleep showed no group effect on delta power (p=0.77) and a trend toward lower theta power (p=0.057) for the ADHD group.

Conclusion: At age 12 to 13 years, NREM sleep delta EEG did not differ between ADHD and control subjects. Theta power, which declines at a younger age than delta, was lower in control subjects. The two recordings thus far differ only by 6 months. The entire study will provide 5 semiannual recordings and allow us to determine if the higher theta power in the ADHD group will hold and if delta power will be greater as well, and thus provide electrophysiological support for the delayed brain maturation suggested by MRI findings.

Support: Shota Rustaveli National Science Foundation Grant FR17_94; Subjects Recruitment Support - Mental Health Service in Tbilisi "Kamara".

0328

THE ASSOCIATIONS BETWEEN SELF-REPORTED SYMPTOMS OF SLEEP DISORDERS, OBJECTIVE SLEEP PATTERNS AND AFFECT IN ADOLESCENCE

Dimakos, J.^{1,2} Gauthier-Gagne, G.¹ Somerville, G.³ Boursier, J.⁴ Gruber, R.^{1,2}

¹Douglas Mental Health University Institute, Montreal, QC, CANADA, ²McGill University, Montreal, QC, CANADA, ³Riverside School Board, Saint Hubert, QC, CANADA, ⁴Heritage Regional High School, Saint Hubert, QC, CANADA.

SLEEP, Volume 43, Abstract Supplement, 2020

Introduction: Developmental changes in adolescence make adolescents prone to experiencing negative mood and increased emotional lability. Experimental studies employing sleep restriction paradigms have shown that decreased sleep increased negative affect, but a gap exists regarding the association between sleep disorders and negative affect in adolescence. The objective of this study was to examine this association. It was hypothesized that higher levels of reported symptoms of sleep disorders would be associated with lower positive affect and higher negative affect in adolescents.

Methods: Participants: 101 adolescents (65 females) aged between 13 and 18 years old (M=14.69, SD=1.16). Measures: Sleep Disorders Inventory for Students was used to measure symptoms of sleep disorders and sleep patterns were measured objectively using actigraphy. Positive and Negative Affect Schedule (PANAS) was used to measure positive and negative affect.

Results: Correlational analyses were conducted to assess the relationship between symptoms of sleep disorders, sleep patterns, and positive and negative affect. Higher levels of reported symptoms of sleep disorders were associated with later bedtimes (r= .26, p< .01), shorter sleep durations (r= -.20, p< .05), increased sleep onset latency (r= .21, p< .05), decreased sleep efficiency (r= -.23, p< .05), and less immobile minutes (r= -.23, p< .05) measured by actigraphy. Higher levels of reported symptoms of sleep disorders were associated with lower levels of positive affect (r= -.20, p< .05) and higher levels of negative affect (r= .39, p< .001)

Conclusion: Reported symptoms of sleep disorders were associated with overall poorer sleep patterns in adolescents as well as decreased positive affect and increased negative affect. Sleep specialists assisting adolescents with sleep disorders should inquire about mood regulation.

Support: Social Sciences and Humanities Research Council

0329

POOR PRENATAL SLEEP AND POSTPARTUM DEPRESSION ARE ASSOCIATED WITH INFANT SLEEP PROBLEMS

Chen, I. Y.¹ Glynn, L. M.² Benca, R. M.¹

¹Department of Psychiatry and Human Behavior, University of California, Irvine, Irvine, CA, ²Department of Psychology, Chapman University, Orange, CA.

Introduction: Childhood sleep problems are associated with increased risk of psychiatric conditions later in life. Sleep disturbances are prevalent during pregnancy and associated with postpartum depression and persistent sleep disturbance. Although maternal sleep and mood likely contribute to infant sleep problems, relationships between these factors are understudied. The present study examined associations of prenatal maternal sleep and postpartum depression with infant sleep patterns.

Methods: The sample included 235 women (29.2±5.8 years old), who were enrolled in a longitudinal study beginning in the first trimester of pregnancy. Maternal sleep and mood were assessed with the Pittsburgh Sleep Quality Index, the Center for Epidemiologic Studies Depression Scale, and the Edinburgh Postnatal Depression Scale during 3 prenatal and 2 postpartum visits. Infant sleep patterns were assessed with the Brief Infant Sleep Questionnaire at 2-, 6-, and 12-months. Mixed model repeated measure analyses were conducted to examine changes in maternal and infant sleep across time. Partial correlation adjusted for age, depression, and postpartum maternal sleep was performed to estimate the association between prenatal maternal sleep and infant sleep.

ANCOVAs controlling for age were conducted to assess the effect of postpartum depression on infant sleep.

Results: Maternal sleep quality deteriorated during the third trimester and 2-months postpartum, and improved at 6-months postpartum (ps<.001). Infant sleep became more consolidated with age, with decreased nocturnal awakenings (frequency and duration) and increased nighttime sleep duration (ps<.001). Poorer prenatal maternal sleep was associated with shorter infant sleep duration at 6 months (r=-0.33, p<.001). Mothers with persistent postpartum depression reported their child as having longer day-time sleep compared to their counterparts (F=3.55, p<.05).

Conclusion: Prenatal sleep problems and persistent postpartum depression are associated with poorer infant sleep. Our findings suggest that screening and preventive interventions for sleep problems during pregnancy may have beneficial impact on infant sleep. **Support:** Research supported by National Institutes of Health MH-96889.

0330

SLEEP HYGIENE IN PRESCHOOLERS: ASSESSING BIOPSYCHOSOCIAL FACTORS ASSOCIATED WITH PRESCHOOLERS' READINESS TO LEARN

Messnick, R.¹ Evert, L.¹ Dixon, B.¹ Everse, C.¹ Manthei, M.¹ Rakus, A.¹ Trent-Brown, S.¹ Gall, A. J.¹

¹Hope College, Holland, MI, ²Hope College, Holland, MI.

Introduction: Sleep is crucial during early development to promote health, education, growth, and quality of life. Insufficient sleep is a public health problem, and this is clearly true for young children. The National Institutes of Health reported that preschoolers need 11-12 hours of sleep daily. We hypothesized that more physically active children would experience better sleep quality which would be associated with higher scores on cognitive and socioemotional measures. We also hypothesized that increased parental awareness of their child's sleep patterns would predict more successful sleep health indicators in their children, leading to positive impact on preschoolers' readiness to learn.

Methods: 82 preschoolers (ages 3-5) completed memory tests, a sleep hygiene scale, and a socioemotional assessment. Following initial cognitive and socioemotional testing, Fitbit devices collected activity and sleep measures for 12 weeks. Additionally, parents recorded bedtimes, wake times, total sleep time, naps, and activity levels in daily sleep journals. We assessed the association between physical activity and sleep quality, bedtime, wake time, memory, sleep hygiene, and socioemotional measures.

Results: Physical activity was positively associated with better sleep habits, including earlier bedtimes, earlier wake times, more consistent sleep-wake patterns, socioemotional scores, and working memory capacity. Higher child and parent sleep hygiene scales were associated with more consistent bedtimes. On average, the 82 pre-schoolers tested fell short of the recommended 11-12 hours of sleep per night.

Conclusion: Greater child and parent awareness of practicing good sleep hygiene increased the likelihood for the child to practice better sleep habits. Since physical activity was positively associated with sleep health measures, it is critical that parents and teachers prioritize sleep and activity in young children. Results of this study provide greater knowledge regarding associations between sleep, physical activity, and biopsychosocial outcomes that may be useful in implementing better education for parents and children geared toward improving sleep.

Support: This research was generously supported by the Caplan Foundation for Early Childhood and the Hope College Psychology Department.

0331

NEUROLOGICAL IMPACT ON THE REWARD PROCESSING OF FOOD IMAGES IMPOSED BY MILD SLEEP RESTRICTION IN ADOLESCENTS

Alsameen, M.¹ DiFrancesco, M. W.² St-Onge, M.³ Beebe, D. W.² ¹University of Cincinnati, Department of Physics, Cincinnati, OH, ²Cincinnati Children's Hospital Medical Center, Cincinnati, OH, ³Columbia University Irving Medical Center, New York, NY.

Introduction: Previous studies indicate that lack of sleep might increase risk for unhealthy eating and obesity. This is particularly important during adolescence, when most youth sleep less than the recommended 8-10 hours/night and dietary habits are developed that extend into adulthood. This study investigated the relationship between multi-night sleep restriction and the appeal of food, including neuroimaging to examine the impact on brain responses to food-related stimuli.

Methods: Healthy 14-17 year-old adolescents (n=39) completed an experimental sleep manipulation across consecutive five-night periods to compare nightly sleep of approximately 9 hours (healthy sleep duration) vs. about 6.3 hours (mild sleep restriction). At the end of each week, participants underwent functional MRI while performing a visual food appeal task. The task included 42 photos in each of four food categories (sweets, snacks, fast-food, meat/ fruit/vegetable) and one non-food category. Photos were presented every 3 seconds in blocks of 7 within each category; 6 interleaved blocks per category. Teens rated the appeal of each block of photos. General linear modeling explained regional brain response according to categorical presentation time-courses.

Results: The pattern of brain responses to the different food types was similar across the two sleep conditions. However, the sleep manipulation led to significant regional effects when contrasting the totality of food vs non-food images. Specifically, when compared to non-food, food images overall resulted in greater activation in the ventral tegmental area (VTA) and substantia nigra (SN) during sleep restriction. Both regions have been previously shown to be involved in processing reward-related information.

Conclusion: Our findings suggest neuronal responses in reward circuitry for teens viewing food images are influenced by sleep duration. Sleep restriction may affect reward processing of food in teens by increasing brain activation in VTA/SN network components that underlie dopamine-mediated motivational drive. **Support:** Supported by NIH R01HL120879

0332

EFFECT OF PRIOR SLEEP DURATION ON DISTINCT MEASURES OF DAYTIME COGNITIVE PERFORMANCE IN LATE ADOLESCENCE

*Campbell, I. G.*¹ *Zhang, Z. Y.*¹ *Cruz Basilio, A.*¹ *Lawrence-Sidebottom, D.*² *Van Dongen, H.*² *Feinberg, I.*¹

¹University of California, Davis Department of Psychiatry and Behavioral Sciences, Davis, CA, ²Sleep and Performance Research Center, Washington State University, Spokane, WA.

Introduction: A recent longitudinal study of sleep need changes across adolescence reported how prior sleep duration affects daytime sleepiness and vigilant attention in children ages 10-16 years. In a follow-up study, we extend the age range in a new group of participants and add additional performance tests. Here we report year 1 data on the effect of systematically varied time in bed (TIB) on daytime vigilance, working memory, and decision making.

Methods: Data are for 52 participants aged 15.0-20.4 years (mean \pm SD: 17.7 \pm 1.8 years). Annually, participants keep each of three different TIB schedules: 7h, 8.5h or 10h TIB for 4 consecutive nights. The 4th night is followed by a laboratory day of performance testing. The day includes four 10-minute psychomotor vigilance tests (PVT); a serial position Sternberg working memory task; and an AX continuous performance test with switch (AX-CPTs) measuring cognitive flexibility in decision making.

Results: PVT performance evaluated by the log of the signal to noise ratio (LSNR) improved monotonically with increasing TIB (p<0.0001). TIB also affected serial position Sternberg task accuracy (p=0.008) but not the probe position effect (p=0.66), indicating that TIB did not affect working memory. TIB also affected AX-CPTs accuracy (p<0.0001), but TIB did not significantly affect decision making and cognitive flexibility measures extracted from this task (all p>0.09).

Conclusion: The initial data from this longitudinal study on older adolescents confirm what we observed for younger adolescents. Increasing TIB improves daytime vigilance but does not affect working memory. These initial results also do not indicate a TIB related improvement in decision making. Data from the entire three year longitudinal study will allow us to further investigate relations of performance to prior sleep duration and whether these relations change with age. Results from dose-response studies such as these can help guide sleep duration recommendations.

Support: PHS grant R01 HL116490 supported this work.

0333

ELECTRONIC MEDIA USE IS ASSOCIATED WITH POOR SLEEP IN 3-6 YEAR-OLD CHILDREN

Wong, S. Hartstein, L. E. LeBourgeois, M. K. University of Colorado Boulder, Boulder, CO.

Introduction: Recent surveys estimate that electronic media use among young children is increasing and that behavioral sleep problems are prevalent. In this study, we employed assessments of sleep and media use and tested the hypothesis that poor sleeping children would be more likely to engage with media than good sleeping children.

Methods: Participants were 44 children from two different cohorts: (1) Healthy, good sleepers (n=26, 13 males, 4.3 ± 0.4 years) who reportedly obtained ≥ 10.5 hours per night and had no behavioral sleep problems and (2) Poor sleepers (n=18, 9 males, 5.5 ± 0.7 years) who reportedly obtained chronic insufficient sleep ≤ 9 hours per night and/or had behavioral sleep problems for ≥ 6 months. Sleep duration and sleep onset latency (SOL) were quantified through 7 nights of actigraphy and verified with sleep diaries. Media use, defined as any electronic device involving screen time that engages children, was assessed across 2 weekdays and 2 weekend days through a parental media diary. Independent t-tests compared the duration of media use and actigraphy variables between groups.

Results: Poor sleeping children on average had longer SOL (28.6 ± 17.9 vs. 17.3 ± 8.66 minutes, t=-2.5, p<0.05) and shorter sleep duration (589.6 ± 37.5 vs. 627.4 ± 27.4 minutes, t=3.7, p<0.01) compared to good sleeping children. Additionally, average daily media use (125.1 ± 88.5 vs. 66.5 ± 48.3 minutes, t=-2.6, p<0.05), evening media use (22.0 ± 21.3 vs. 4.2 ± 10.4 minutes, t=-3.3, p<0.01), and weekend media use (154.4 ± 105.9 vs. 79.8 ± 55.6 minutes, t=-2.7, p<0.05) duration was higher in poor than good sleepers.

Conclusion: Our findings indicate that media use duration and timing likely play an important role in early childhood sleep health. Young children who use more evening media are more likely to take longer to fall asleep and have shorter sleep duration overall. Time displacement (time spent using media instead of sleeping), psychological stimulation, and the effects of screen light on circadian timing are potential mechanisms underlying these associations. **Support:** NIH R01-MH086566 and R21-MH110765 to MKL

0334

HIPPOCAMPAL DEVELOPMENT, SLOW WAVE ACTIVITY, AND NAP-DEPENDENT MEMORY CONSOLIDATION IN EARLY CHILDHOOD

Lokhandwala, S.¹ Allard, T.² Riggins, T.² Spencer, R. M.¹ ¹University of Massachusetts Amherst, Amherst, MA, ²University of Maryland, College Park, MD.

Introduction: Naps support memory consolidation in early childhood. In adults, nap-dependent declarative consolidation is associated with SWA. SWA increases from early childhood into adulthood, and the shift of SWA from occipital to frontal distribution (F/O-ratio) is a marker of brain maturation. Thus, we explored how electrophysiological and structural characteristics of brain development relate to nap-dependent declarative learning in early childhood.

Methods: Twelve preschool-age children (8 female, M=48 months, SD=0.44) have completed three sessions (~1wk apart) within a larger study. In the first two sessions, children completed a visuo-spatial task before and after a 2-hr nap or wake interval. During the third visit, children underwent MRI assessment. Using PSG, SWA was measured in the delta band over frontal and occipital regions for nREM2 and nREM3 sleep.

Results: While F/O-ratio of SWA does not currently predict the F/O-ratio of cortical thickness (r(12)=.383, p=.219), right parahippocampal thickness positively correlates with F/O-ratio of SWA in nREM2 (r(12)=.591, p=.043). Nonetheless, children's performance change following the nap was not associated with either parahippocampal thickness or F/O-ratio of SWA in any sleep stage (all *ps*>.538). However, performance in children who showed a postnap benefit (n=5) positively correlated with right parahippocampal thickness (r(5)=.915, p=.029). This was not the case for children who did not show a post-nap benefit (r(7)=-.199, p=.668).

Conclusion: Although the F/O-ratio of SWA did not predict a similar ratio of cortical thickness, the association between right parahippocampal thickness and F/O-ratio of SWA is evidence that development of SWA parallels cortical development. While there is no overall association between post-nap performance and brain development characteristics, the relation between performance and right parahippocampal thickness in children showing a nap benefit suggests that memory during this age may depend on structural (rather than electrophysiological) brain development changes. **Support:** NIH R21 HD094758 & NSF BCS 1749280

0335

FRONTAL EXPRESSION OF NREM SLEEP OSCILLATIONS ARE ASSOCIATED WITH EXECUTIVE FUNCTION IN CHILDREN AND ADOLESCENTS

Lui, K. K.¹ Mander, B. A.¹ Radom-Aizik, S.² Chappel-Farley, M. G.¹ Dave, A.¹ Chen, I. Y.¹ Benca, R. M.¹ Neikrug, A. B.¹ ¹Department of Psychiatry and Human Behavior, University of California, Irvine, Irvine, CA, ²Pediatric Exercise and Genomics Research Center, Department of Pediatrics, University of California, Irvine, Irvine, CA. **Introduction:** The prefrontal cortex, an area known for executive functioning (including inhibition and self-monitoring) develops during childhood and adolescents, with a pattern of posterior to anterior brain development. Slow-wave activity (SWA) in NREM sleep, tracks brain development with high SWA power migrating from occipital to frontal region as brain maturation occurs. This pilot study aimed to examine whether slow wave topography is correlated with executive function in youth.

Methods: Seventeen healthy children and adolescents (ages 11-17; 10 females) underwent overnight polysomnography (PSG) with high-density electroencephalography (hdEEG). Behavior Rating Inventory of Executive Function (BRIEF) was administered to assess executive function. SWA (SWA1: 0.5-1 Hz; SWA2: 1-4.5 Hz) and spindle (slow sigma: 11-13 Hz; fast sigma: 13-16 Hz) activity was analyzed with spectral analysis using Welch's method. BRIEF subscales of inhibition and monitor were correlated with SWA and sigma power across all derivations, with Holm-Bonferroni correction (126 channels). Significant derivations were then controlled for sex and self-reported Tanner stage using multiple regression

Results: BRIEF-Inhibition scale (i.e., ability to repress impulsivity) and SWA1 in anterior frontal derivations were negatively correlated (R^2 =0.58, p=0.047 corrected). BRIEF-Monitor scale (i.e., self-perception of one's own behavior and interpersonal awareness) was negatively correlated with fast sigma in anterior frontal derivations (R^2 =0.65, p=0.013 corrected). These associations were significant after controlling for sex and Tanner stage.

Conclusion: These results support the hypothesis that NREM sleep oscillations are associated with executive function and reflect changes in neuroplasticity related to "back-to-front" brain maturation. Future longitudinal studies should combine multi-modal neuroimaging of brain structure and local sleep with comprehensive assessments of executive function to evaluate the possible link between local sleep and development of higher-order cognition in frontal brain regions in youth.

Support: NCATS grant #UL1TR001414 & PERC Systems Biology Fund

0336

THE RELATIONSHIP BETWEEN CHRONOTYPE, SLEEP AND SPORT MOTIVATION IN ADOLESCENTS

Bourgon, V. Forest, G.

Université du Québec en Outaouais, Gatineau, QC, CANADA.

Introduction: Studies have shown that sleep affects physical performance and certain aspects of motivation in general. Even though it is well known that adolescents are generally sleep deprived, very few studies have investigated the impact of sleep on the inactivity of teens. The aim of this study was to determine the association between sleep and sport motivation in teenagers.

Methods: 176 young, physically active participants (10-18y; 86 male) completed an online survey comprised of questions extracted from the Adolescents Sleep Habits Survey, the Pediatric Daytime Sleepiness Scale, the Morningness Eveningness Questionnaire, and the Sports Motivation Scale-28. The self-determination theory of motivation was used to determine the different types of motivation, from the most autonomous motivation (intrinsic motivation) to the least autonomous (extrinsic motivation. Pearson correlations were computed between the sleep variables (sleep habits, daytime sleepiness, chronotype score) and the motivation variables (different types of motivation scores).

Results: Results show that amotivation is significantly associated with daytime sleepiness (r=.16, p=.03). The external regulated form of extrinsic motivation score is significantly associated with daytime sleepiness (r=.157, p=.037), chronotype score (r=-.164, p=.03), and bedtimes on weekends (r=.156, p=.042). The intrinsic motivation score is significantly associated with wake times on weekdays (r=-.189, p=..012).

Conclusion: These results suggest that eveningness, higher daytime sleepiness, and later bedtimes on weekends are associated with amotivation and external regulation of sport motivation. Research has shown that teens who present those two characteristics are more likely to drop out of sports teams or leagues. This could have important implications when addressing inactivity and sport motivation problems in adolescents. **Support:** N/A

0337

ASSOCIATION BETWEEN FREE-LIVING SLEEP AND MEMORY AND ATTENTION IN HEALTHY ADOLESCENTS

Stefansdottir, R.¹ Gundersen, H. S.² Haraldsson, H.¹ Rognvaldsdottir, V.¹ Lundervold, A. S.² Gestsdottir, S.¹ Gudmundsdottir, S. L.¹ Chen, K. Y.³ Brychta, R. J.³ Johannsson, E.¹ ¹University of Iceland, Reykjavik, ICELAND, ²Western Norway University of Applied Sciences, Bergen, NORWAY, ³Diabetes, Endocrinology, and Obesity Branch, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, DC.

Introduction: Sleep is important for people of all ages, especially children during development. However, adolescents often sleep less than the recommended eight hours per night. Clinical trials have found that even partial sleep deprivation- shorter than the recommended duration- can reduce cognitive function in adolescents. The association between objectively measured free-living sleep and cognition function in adolescents has not been studied.

Methods: Free-living sleep duration and sleep efficiency were measured over one week with wrist actigraphy in 199 healthy normal adolescents (140 girls, mean±SD, 17.7±0.3 years). The day after the sleep measurement concluded, sustained attention was assessed with a validated Posner cue-target task, and working memory was measured with an n-back task. Associations between sleep measures and response times during attention and memory tasks were explored with multiple linear regression adjusted for task accuracy. Results: Over the entire week, participants' average sleep duration was 6.2±0.7 h/night and average sleep efficiency was 88±4.4% and averages for sleep the night prior to the cognitive testing were similar. Response times on memory (1-back: 420.6±73.9, 2-back: 522.6±101.9, and 3-back: 551.8±137.2 msec) and attention tasks (valid cue: 309±31.2, invalid cue: 365.8±36, and no cue: 393.6±38.9 msec) were similar to previous reports and not associated with average weekly sleep measures. Sleep duration of the night before cognitive testing was negatively associated with response times for the most challenging memory task (3-back; p=0.02). However, sleep measures of the night before did not correlate with any of the attention task scores.

Conclusion: Our data suggests that performance on difficult memory tasks may be negatively impacted by shorter free-living sleep durations the night prior to testing, even in healthy adolescents who average less than the recommended amount of sleep. Future studies should explore whether recovery sleep or other improvements in sleep habit might mitigate such effects on memory.

Support: The Eimskip University of Iceland Fund, Icelandic Centre for Research, National Institute of Diabetes and Digestive and Kidney Diseases.

0338

INTERACTIONS BETWEEN SLEEP, STRESS REACTIVITY AND COGNITION IN EARLY CHILDHOOD

Jablin, T.¹ LeBourgeois, M. K.¹ Harsh, J.¹ Brown, S.² ¹University of Colorado, Boulder, CO, ²Colorado State University, Fort Collins, CO.

Introduction: During early childhood, sleep impacts the development of the cognitive, behavioral and stress systems. Specifically, acute sleep restriction reduces the subsequent cortisol awakening response, predicts self-regulation strategies and moderates correlations between self-regulation strategies and response inhibition. However, little is known about the interaction between sleep, stress reactivity and cognition in early childhood. This preliminary cross over study aimed to determine how acute sleep restriction moderates the relationship between stress reactivity and cognition in 4-year-olds.

Methods: Healthy children (N=17; 57.4 months +/- 2.1; 10 female) participated in a sleep restriction protocol that included counterbalanced cognitive and behavioral assessments during baseline and sleep restriction conditions. An age appropriate inhibitory control task was administered and salivary cortisol samples (N=6) were collected during the task. Mean processing speed was measured, and stress reactivity was computed as area under the curve with respect to ground (AUCg).

Results: Two tailed correlation analyses were performed to examine the relationship between AUCg and mean processing speed. Under baseline conditions, AUCg and mean processing speed were positively associated (r=0.45; p=0.05). When children were sleep restricted, there was no association between AUCg and mean processing speed (r=0.05; p=0.83). Although not statistically significant, AUCg was predicted by an interaciton between sleep condition and mean processing speed B=-1.92; p=0.06).

Conclusion: These results suggest that healthy sleep may promote the coupling of stress and cognitive systems, which is likely adaptive when facing life's challenges in early childhood. Examining the developmental trajectory of these interactions and incorporating individual difference factors will build upon this model that may eventually be applied in intervention approaches to sleep, stress and behavioral problems in preschool-aged children.

Support: NIH R01-MH086566 to Dr. Monique LeBourgeois

0339

WHERE DOES THE TIME GO? REPORTED ACTIVITIES AROUND THE DLMO IN OLDER ADOLESCENTS

Crowley, S. J. Janevski, K. Eastman, C. I. Rush University Medical Center, Chicago, IL.

Introduction: Older adolescents show heightened alertness in the evening close to the time of their Dim Light Melatonin Onset (DLMO), a time when the circadian system is most responsive to delaying light. We examined reported activities of adolescents around the time of their DLMO.

Methods: Forty-six adolescents (14.2-17.9 years; 24 females) who reported \leq 7 h sleep on school nights and late bedtimes (school-night \geq 23:00; non-school night \geq midnight) slept at home on their usual school-year sleep schedule for 2 weeks. Participants reported their main activity via text message every hour from 16:00 until

self-selected bedtime. After these 2 weeks, their DLMO was measured. We examined reported activities in the hour around the DLMO and the 2 hourly responses that followed on weeknights (Sunday-Thursday) to determine the most common activities (n=1380 responses). Logistic regression tested whether frequency of activities predicted whether a participant's DLMO fell within the earliest (n=15; 19:31 ± 00:44), middle (n=16; 20:49 ± 00:20), or latest (n=15; 22:29±1:15) tertile.

Results: Overall, reported activities that consumed the most time were cell phone use (19.5%), homework (18.3%), and watching TV (15.1%). Adolescents who reported more homework, were more likely to have a DLMO in the middle tertile compared to the earliest and latest tertiles. Cell phone use was least likely in adolescents in the earliest DLMO tertile. TV watching did not predict DLMO group.

Conclusion: Adolescents who had the earliest DLMOs spent less time on their phones when light has the greatest delaying effect. These data may indicate that light from cell phone screens may delay circadian phase in this age group. Alternatively, cell phone use may be more likely if adolescents cannot fall asleep due to a later circadian cue for sleep onset.

Support: R01 HL112756 (Crowley)

0340

REGIONAL CHANGES IN SLEEP ELECTROENCEPHALOGRAPHY POWER IN YOUTH WITH SLEEP-DISORDERED BREATHING: A HIGH-DENSITY EEG STUDY

*Myers, A.*¹ *Matthews, C.*² *Kille, T.*³ *Riedner, B.*¹ *Flaherty, B.*¹ *Jones, S.*¹

¹Department of Psychiatry, University of Wisconsin Madison, Madison, WI, ²Department of Pediatrics, University of Wisconsin Madison, Madison, WI, ³Department of Surgery, University of Wisconsin Madison, Madison, WI.

Introduction: Daytime neurobehavioral impairments are commonly associated with sleep disordered breathing (SDB) in children. However, a large number of studies have shown only minimal differences in sleep between children with SDB relative to control children, suggesting that sleep dysfunction is not responsible for daytime impairment. Importantly, however, previous studies have measured sleep EEG using only frontal scalp electrodes, failing to capture the regional features of sleep that are prominent during development. Here we measure sleep using hdEEG in SDB and healthy children to determine if regional sleep impairment is related to daytime neurobehavioral performance.

Methods: Overnight high-density electroencephalography (hdEEG, 256 channels) was recorded in 17 children with sleep disordered breathing (SDB) (age: M = 8.46, SD = 1.82, AHI: M = 11.3, SD = 8.6, 53% female) and 17 age and sex matched controls (age: M = 8.47, SD = 1.66, AHI: M = 1.5, SD = .64). Attentional capacity was assessed using the Test of Variables of Attention (TOVA) before and after sleep. Group differences in sleep macrostructure variables were assessed using unpaired t-tests. All-night spectral analysis was performed for NREM sleep and averaged across groups. Topographic differences between groups were assessed using statistical non-parametric mapping. Pearson correlations were used to determine associations between sleep and TOVA variables.

Results: Sleep macrostructure did not differ between groups. Allnight spectral density analysis revealed a global increase in highfrequency activity in N2N3 and N3, in the alpha band (8-12 Hz, p<0.05). Global alpha power was higher in SDB youth, although this effect reached significance during N3 in a large cluster of posterior channels (N=55, p=.02).

Conclusion: Elevated alpha during NREM is frequently considered a correlate of nonrestorative sleep. In this sample of youth with SDB, posterior alpha is robustly increased during the deepest stage of NREM sleep. In this small sample, however, alpha power did not predict performance on an attentional task sensitive to the effects of impaired sleep.

Support: R21 HD092986-02 to SJ

0341

PRELIMINARY EFFECTS OF A MUSIC INTERVENTION ON ACTIGRAPHY-MEASURED SLEEP AMONG OLDER ADULTS WITH DEMENTIA

Mu, C. Lee, S. Risal, P. G. Vigoureux, T. F. Bugos, J. Meng, H. University of South Florida, School of Aging Studies, Tampa, FL.

Introduction: Music may benefit sleep and daytime alertness by decreasing stress, increasing attention, and potentially, slowing the progression of dementia. This study examined preliminary effects of a group-based music intervention on sleep health among older adults with dementia.

Methods: Participants were older adults with dementia living in an assisted living facility (n=9; $M_{age}=80.11$; $M_{range}=63-89$ years). Cohort 1 (n=4) received the intervention in the morning and cohort 2 (n=5) received the intervention in the afternoon. Participants completed a 4-week intervention protocol (12 sessions) along with a oneweek actigraphy sleep assessment before and after the intervention. Informed by sleep literature, we constructed a composite sleep health score encompassing Regularity, Satisfaction or quality, Alertness, Timing, Efficiency, and Duration (higher scores indicating more daily sleep problems). Using descriptive statistics and multilevel modeling, we evaluated preliminary effects of the intervention on overall sleep health and each of the sleep dimensions.

Results: Six out of nine participants exhibited a decrease in overall sleep problems at post-intervention. All 4 participants in cohort 1 showed improvement in overall sleep health at post-intervention. Specifically, in cohort 1, participants exhibited a decline in nightly sleep problems, decreased daytime nap duration, and number of naps. In contrast, while two out of five participants in cohort 2 exhibited improvement in overall sleep health, the remainder of the participants exhibited no improvement in daily sleep problems, especially in nap domains. Across cohorts, those younger in age, with vascular dementia, lower weight, and not taking sleep or hypertension related medications tended to respond better to the intervention.

Conclusion: Our preliminary results demonstrate the feasibility and potential benefit of a group-based music intervention in improving overall sleep health among older patients with dementia. Implications for conducting community-based non-pharmacological interventions to improve sleep and daytime functioning among older adults with dementia will be discussed.

Support: This work was supported, in part, by the Florida Department of Health Ed and Ethel Moore Alzheimer's Disease Research Award (PI: Meng, Grant #9AZ28).

0342

SELF-SILENCING WITHIN INTIMATE RELATIONSHIPS, SLEEP, AND SUBCLINICAL ATHEROSCLEROSIS IN MIDLIFE WOMEN

Jakubowski, K. P.¹ Chang, Y.² Barinas-Mitchell, E.³ Matthews, K. A.^{1,4,3} Maki, P. M.^{5,6,7} Thurston, R. C.^{1,4,3}

¹Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, ²Department of Neurosurgery, University of Pittsburgh, Pittsburgh, PA, ³Department of Epidemiology, University of Pittsburgh, Pittsburgh, PA, ⁴Department of Psychology, University of Pittsburgh, Pittsburgh, PA, ⁵Department of Psychiatry, University of Illinois at Chicago, Chicago, IL, ⁶Department of Psychology, University of Illinois at Chicago, Chicago, IL, ⁷Department of OB/GYN, University of Illinois at Chicago, IL.

Introduction: Social relationships are important for health. In some relationships, women learn to self-silence, or to inhibit self-expression to avoid conflict or loss. Self-silencing is associated with reported psychiatric and physical symptoms, but no studies have examined whether self-silencing is related to worse sleep or cardiovascular (CV) health. We tested relationships of self-silencing to sleep and carotid plaque in midlife women; secondary analyses examined whether sleep mediated or moderated relationships between self-silencing and plaque.

Methods: In an ongoing community-based study of nonsmoking women, 304 women aged 40-60 were assessed at baseline; 157 of these women have been assessed 5 years later. At baseline, women reported on self-expression in their current/ last intimate relationship via the Silencing the Self Scale. At both visits, women provided self-reports (demographics, medical history, CESD depression, PSQI sleep quality), physical measures, actigraphy (total sleep time [TST], wake after sleep onset [WASO], and efficiency), and carotid artery ultrasound to quantify plaque. Relationships of self-silencing and subscales to sleep (subjective and actigraphic sleep at baseline and averaged across visits) and carotid plaque $(0, 1, \ge 2)$ were tested in linear regression and multinomial regression models, respectively, adjusted for demographic and health indices, including depressive symptoms and snoring.

Results: At baseline, women (72% White) were on average 54 years old; 44% reported poor sleep quality, 46% had plaque (24% score \geq 2), and average TST, WASO, and efficiency were 6.2 hrs, 46 min, and 84%, respectively. At baseline, self-silencing (particularly the tendency to judge oneself by external standards) was related to worse sleep quality (p=.001), but better actigraphic WASO (p=.02) and efficiency (p=.02). Self-silencing was related to worse average sleep quality across visits (p=.001). Self-silencing related to higher odds of baseline plaque \geq 2 [OR(95% CI)=1.14 (1.02,1.28), p=.02], yet sleep did not explain or moderate this relationship.

Conclusion: Self-silencing was associated with worse subjective, but better actigraphic sleep at baseline, and with poorer sleep quality over 5 years. Self-silencing related to carotid atherosclerosis, yet sleep did not appear to impact this relationship. Emotional expression is relevant to midlife women's sleep and CV health.

Support: R01HL105647, K24123565 (RCT); RF1AG053504 (RCT & PM); T32MH018269 (KPJ)

0343

ASSOCIATIONS OF ENDOGENOUS HORMONES AND PHTHALATE EXPOSURE WITH SUBJECTIVE AND OBJECTIVE SLEEP MEASURES IN MIDLIFE WOMEN

Hatcher, K. M.^{1,2} Smith, R. L.³ Li, Z.⁴ Flaws, J. A.² Davies, C. R.⁵ Mahoney, M. M.^{1,2}

¹Neuroscience Program, University of Illinois at Urbana-Champaign, Urbana, IL, ²Department of Comparative Biosciences, University of Illinois at Urbana-Champaign, Urbana, IL, ³Department of Pathobiology, University of Illinois at Urbana-Champaign, Urbana, IL, ⁴Roy J. Carver Biotechnology Center, University of Illinois at Urbana-Champaign, Urbana, IL, ⁵Carle Regional Sleep Disorders Center, Carle Foundation Hospital, Urbana, IL.

Introduction: Impaired sleep during the menopausal transition reduces quality of life and increases risk of multiple diseases. The changing hormonal milieu during midlife is associated with impaired sleep. Endocrine disrupting chemicals, such as phthalates, may also contribute to the increased prevalence of sleep disturbances in midlife women. Phthalates are known to impact the endogenous hormones associated with sleep. However, the link between phthalate exposure and sleep quality remains unexplored.

Methods: We recruited 26 midlife women (median age 50 years) through the Carle Regional Sleep Disorders Center in Urbana, Illinois. Subjective sleep was assessed through the Pittsburgh Sleep Quality Index, Epworth Sleepiness Scale, and self-reported frequency of sleep disturbances, insomnia, and restless sleep. Objective sleep was measured using actigraphy and manual sleep logs (7-day average). Serum levels of follicle-stimulating hormone, estradiol, progesterone, testosterone, free estradiol, and free testosterone were quantified using ELISAs from a single sample from each participant. Phthalate metabolites were quantified from urine using high performance liquid chromatography-mass spectrometry (HPLC-MS). Covariates, including depression, hot flashes, quality of life, demographics, and lifestyle factors, were measured via surveys.

Results: Preliminary unadjusted logistic regression was used to determine the association between hormone values and subjective sleep quality binomial variables, including daytime sleepiness, sleep efficiency, sleep onset latency, sleep duration, number of sleep disturbances, and frequency of sleep disturbances. Each of these subjective sleep measures is significantly associated with one or more hormones.

Conclusion: Our results are consistent with literature identifying associations between hormones and subjective sleep in midlife women. Additional analyses will determine associations between hormones and objective sleep, and phthalates with both subjective and objective sleep measures. Excitingly, our study will be among the first to investigate the association between endocrine disruption and sleep quality in this population.

Support: Carle Illinois Seed Grant Program

0344

AGE-RELATED LONGITUDINAL TRAJECTORIES IN NREM AND REM SPECTRAL POWER

Gao, C. Scullin, M. K. Baylor University, Waco, TX.

Introduction: Knowledge of how aging impacts sleep physiology is based almost exclusively on cross-sectional studies. Longitudinal studies, by contrast, can inform how macro- and micro-features of NREM and REM sleep change dynamically across time in individual trajectories. For the current work, we conducted quantitative EEG analyses from a longitudinal polysomnography study to inform age-related trajectories in sleep macro- and micro-architecture.

Methods: We conducted a secondary data analysis on 2208 participants in the Sleep Heart Health Study (mean age =62.47, SD=10.46, 55.30% females). Participants underwent one night of in-home polysomnography recording during two study visits (M=5.21 years apart, SD=0.53). Spectral power density was calculated for each 0.5 Hz frequency bin for NREM and REM sleep separately.

Results: In cross-sectional analyses, older chronological age was significantly associated with worse sleep macro-architecture. Plots of the individual trajectories over 5 years, however, revealed considerable inter-individual variability in whether sleep physiology was preserved or declined. Interestingly, there were strong associations between the longitudinal changes in power density in NREM and REM sleep (slow oscillations: r=.53 [.50-.56]; delta: r=.58 [.55-.60]; alpha: r=.69 [.67-.71]; sigma: r=.74 [.73-.76]; beta: r=.82 [.80-.83]; ps<.001). The strongest NREM-REM association was for theta band power (r=.85 [.83-.86]), particularly in the 5.5-6.0 Hz bin (r=.94, [.94, .95]).

Conclusion: There is substantial inter-individual variability in how aging impacts sleep physiology. Nevertheless, within individuals, power density declines similarly across NREM and REM stages, with nearly perfect convergence for theta activity, indicating a common age-related neurobiological mechanism.

Support: The National Sleep Research Resource is supported by NIH HL114473.

0345

POOR SLEEP HEALTH IN INFORMAL CAREGIVERS MEDIATED BY DEPRESSED AFFECT

Marino, V. R. Lee, S. Haley, W. E. University of South Florida, Tampa, FL.

Introduction: Informal caregiving has been linked to higher perceived stress. Caregiving stress may be associated with poor sleep. Studies report low sleep quality among caregivers, however, little is known about whether informal caregivers have poorer sleep health than non-caregivers across multiple sleep dimensions. Less is known about whether potential poor sleep health in informal caregivers is explained by depressed mood. This study examined the mediating effect of depressed affect on the relationship between caregiving status and multidimensional sleep health.

Methods: 208 current informal caregivers were compared to 3,342 non-caregivers from the Midlife in the United States study $(M_{age}=55.77\pm12.29)$. Using seven domains of sleep (i.e., regularity, satisfaction in sleep, alertness, sleep duration, sleep latency, insomnia, and chronic sleep problems), a composite score of sleep health was constructed using clinically-relevant and previously-used cutoffs (*Range=0-7*; higher scores indicating better sleep health). Depressed affect was measured by a continuous scale based on 7 items. The Hayes PROCESS macro with bootstrapping method was used to test mediation with sociodemographic covariate adjustment.

Results: Caregivers had poorer sleep health than non-caregivers ($\beta = -0.17$; 95% CI = [-.331, -.016]). This association was mediated by depressed affect (indirect effect = -.026; 95% CI = [-.061, -.000]). Specifically, caregiving status was associated with greater depressed affect ($\beta = 0.234$; 95% CI = [.019, .450]) and greater depressed affect was associated with poorer sleep health ($\beta = -.112$; 95% CI = [-.136, -.089]). The direct effect of caregiving status on sleep health was attenuated when the mediator was considered ($\beta = -0.148$; 95% CI = [-.303, -.008]) indicating full mediation.

Conclusion: Compared to non-caregivers, informal caregivers have poorer sleep health, and this is mostly due to their greater depressed affect. Stress adaptation techniques such as mindfulness and positive reappraisal may promote better sleep health in informal caregivers through improved affect.

Support: N/A

0346

METABOLIC AGING AND SLEEP LOSS: METABOLITE SIGNATURES LINK SLEEP DEPRIVATION AND AGING ACROSS TISSUES

Sengupta, A.¹ Tudor, J. C.² Cusmano, D.¹ Baur, J. A.¹ Abel, T.³ Weljie, A.¹

¹University of Pennsylvania, Philadelphia, PA, ²Saint Joseph's University, Philadelphia, PA, ³University of Iowa, Iowa City, IA.

Introduction: Insufficient sleep is a hallmark of modern society, and sleep deprivation (SD) is a risk factor for neurodegenerative and cardiometabolic disorders. The interactions of aging with systemic and local metabolic alterations induced by sleep deprivation are essentially unexplored. In this study, we demonstrate a shared metabolic imprint of SD and aging in plasma, liver, and hippocampus.

Methods: Young (2 - 4 months) and aged (22 - 24 months) mice were sleep deprived (N = 10/group) for 5 hours followed by collection of blood plasma, liver and hippocampus. The samples were extracted and subjected to UPLC-MS/MS based targeted metabolomics analysis.

Results: Young animals displayed greater sensitivity to SD induced metabolic changes with >40% more metabolites perturbed in each sample type measured compared to aged animals. Enrichment analysis based on known disease-associated metabolites suggests that plasma change in young animals are of pathological relevance, but not in aged animals. A common hepatic signature of sleep-loss across the two age groups consisted of ketosis and urea cycle perturbation. Approximately 20-30% of measured metabolites exhibit similar changes when the sleep deprivation induced signature is compared with the aging metabolic imprint in a tissue-dependent manner. Central energetics, urea cycle and aromatic amino acid metabolism highlight the common pathways altered by sleep and aging in the periphery. In the hippocampus, choline and acetylcholine pools were depleted, potentially providing insight into the changes in metabolism that accompany analogous defects in memory consolidation.

Conclusion: These results support the notion that SD makes the 'young seem old'. The results further connect neurobehavioral observations tying together aging and sleep loss, by implicating molecular mechanisms at the level of metabolism.

Support: This work was supported by NIH grant R21AG052905 (AMW, AS), P50AG017628 (TA; A.I. Pack, PI) and R01AG062398 (TA, JT). TA was supported by the Brush Family Chair in Biology at Penn and is currently supported by the Roy J. Carver Chair of Neuroscience at Iowa.

0347

AGING AND RETIREMENT ARE INDEPENDENTLY ASSOCIATED WITH REDUCED SOCIAL JETLAG

Sprecher, K. E. Hagen, E. W. Ravelo, L. A. Barnet, J. H. Peppard, P. E.

University of Wisconsin- Madison, Madison, WI.

Introduction: Social jetlag (SJL; difference in sleep timing on freedays versus workdays) is a measure of chronic circadian misalignment due to a mismatch between preferred biological sleep timing and societal scheduling demands. Cross-sectionally, greater SJL is associated with poorer health and younger age. We assessed longitudinal changes in SJL across aging and retirement.

Methods: In 1137 participants of the Wisconsin Sleep Cohort (WSC), sleep timing was collected via sleep diaries every 4 years (2802 data points, 47% female, mean age 51 years at baseline (+/-8 SD, range 31 - 74)). In 829 participants of the Retirement and Sleep Trajectories study (REST, an ancillary study of the WSC), sleep timing and retirement status were collected in 4 annual mailed surveys (1700 data points, 55% female, mean age 59 years at baseline (range 46-81)). Midsleep was defined as the time midway between bedtime and waketime. SJL was defined as the absolute difference between midsleep on workdays/weekdays and freedays/weekends. Mixed models were used to test associations of change in sleep timing with change in age, retirement status and self-reported health.

Results: In the WSC, aging 10 years was associated with 16 minutes reduction in SJL (p<.001), driven by a shift of midsleep to 7 minutes later on weekdays and 10 minutes earlier on weekends. The effect of age on SJL remained significant when tested only in employed people. In the REST study, the transition from full time employment to full time retirement was associated with ~ 1 hour shorter SJL, maintained 1, 2 and 3 years post-transition (p<.001, controlling for age). Greater SJL was associated with worse self-rated health (p=.02).

Conclusion: Aging and the transition from employment to retirement are independently associated with reduced social jetlag. Greater social jetlag is associated with poorer self-reported health. **Support:** This study was supported by grants from the National Institutes of Health (NIH): R01HL62252, R01AG036838, R01AG058680, and 1UL1RR02501.

0348

SLEEP EEG-BASED BRAIN AGE INDEX IS REDUCED UNDER CONTINUOUS POSITIVE AIRWAY PRESSURE TREATMENT

*Sun, H.*¹ *Dunham, K.*² *Cunningham, L.*² *Ni, Y.*² *Westover, M.*¹ *Thomas, R.*²

¹Massachusetts General Hospital, Boston, MA, ²Beth Israel Deaconess Medical Center, Boston, MA.

Introduction: Continuous positive airway pressure (CPAP) is a treatment for apnea. With long-term CPAP, changes in electroencephalogram (EEG) include increased delta power (1 - 4Hz) and sigma power (11 - 15Hz, spindle). However, the short-term EEG response to CPAP in a split-night study is less quantified. We recently developed a "brain age" model using sleep EEG features. The brain age index (BAI) is defined as the difference between chronological age and brain age (BA - CA). Here we first quantify how BAI changes during CPAP in the same patient, and then investigate how much brain age features during the diagnostic part can predict the reduction in apnea-hypopnea index (AHI) during CPAP.

Methods: The dataset consisted of 160 subjects. The average age was 59 years with 53% male, 24% female and 23% unknown. We extracted 480 features including band powers, and then computed the BAIs for both diagnostic and CPAP parts. To predict the reduction in AHI during CPAP, we fit a Bayesian regression model using the brain age features, demographics, and sleep parameters

during the diagnostic part, and assessed the feature importance using dominance analysis.

Results: The BAI from the diagnostic part is significantly reduced compared to BAI during CPAP for the same subject (paired t-test, p < 0.01). The diagnostic part has an average BAI 2.24 years; and the CPAP part -4.75 years. The brain age features that are increased during CPAP include sigma powers in N2 and N3. The prediction of AHI reduction has Pearson's correlation 0.85. The features predictive of reduced AHI are the diagnostic AHI (explained variance 69%), followed by high/low waveforms during N2 (e.g. K-complex, measured by kurtosis) (8.6%), delta power during REM (4.5%) and N1 (2%). The feature predictive of increased AHI is frontal alpha power during quiet awake (2.6%).

Conclusion: The average BAI is reduced during CPAP. BAI provides a novel view of the acute response to CPAP in sleep EEG. Future study with more CPAP failure patients has the potential of predicting CPAP failure.

Support: MBW is supported by Glenn Foundation for Medical Research. RJT is supported by Category I AASM Foundation.

0349

CIRCADIAN PATTERNS OF BLOOD PRESSURE DIFFER BETWEEN RETIRED NIGHT SHIFT WORKERS AND RETIRED DAY WORKERS

Lehrer, M. Bowman, M. A. Buysse, D. J. Hall, M. H. University of Pittsburgh, Pittsburgh, PA.

Introduction: Approximately 15% of full-time employees in the U.S. work outside the traditional daytime schedule. These individuals exhibit circadian rhythm abnormalities and increased rates of cardiovascular disease compared to day shift workers. Altered circadian patterns of cardiovascular function may persist into retirement even after returning to a normal nocturnal sleep schedule, and may contribute to the elevated cardiovascular disease burden among retired night shift workers. The purpose of this study was to determine whether circadian rhythms of cardiovascular indicators differed between retired night shift workers and retired day workers.

Methods: Participants (N = 72, 53% females, 83% non-Hispanic White, mean age: 68.66 years) were 33 retired night shift workers and 39 retired day workers who completed a 60-hour sleep/circadian laboratory assessment. Blood pressure and heart rate were assessed hourly during a 24-hour constant routine protocol. Multilevel cosinor analysis estimated group differences in circadian rhythms of systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR). Covariates included age, sex, race, education, and body mass index. **Results:** The 24-hour pattern of DBP (b_{sine} *shiftwork = 1.47, [95% CI: 0.58, 2.35], *p* = 0.002) and MAP (b_{sine} *shiftwork = 1.38, [95% CI: 0.28, 2.50], *p* = 0.015) differed between retired night shift workers and retired day workers. Retired night shift workers displayed a later trough of DBP and MAP compared to retired day workers. No group differences were found for circadian rhythms of SBP or HR.

Conclusion: Retired night shift workers exhibited altered circadian rhythms of blood pressure, which may indicate a circadian "scarring" of night shift work that persists after a return to normal daytime schedule. Future research should investigate explanations for observed differences and the extent to which chronic alterations in cardiovascular circadian rhythms affect morbidity and mortality. **Support:** R01AG047139, T32HL082610, T32HL07560, UL1TR001857

0350

CHARACTERIZING CONTINUOUS CHANGES IN SPECTRAL DYNAMICS OF SLEEP EEG AS A FUNCTION OF AGE

Kim, H.^{1,2} Prerau, M.¹ Redline, S.¹

¹Brigham and Women's Hospital, Division of Sleep and Circadian Disorders, Boston, MA, ²Departments of Neurology and Medical Science, Ewha Womans University School of Medicine and Ewha Medical Research Institute, Seoul, KOREA, REPUBLIC OF.

Introduction: Sleep is a continuous and dynamic physiological process. Current research practice, however, limits our ability to observe electroencephalography (EEG) oscillation dynamics by breaking sleep into discrete stages. In this study, we propose a novel quantitative framework that represents population-level changes in sleep EEG spectral dynamics as a function of age, preserving the information-rich spectral dynamics of sleep data. Rather than relying on sleep stages, our approach uses slow-oscillation power (SO-power) as an objective, continuous-valued correlate of sleep depth.

Methods: We analyzed the EEG signal (Fz-Cz, 256 Hz sampling rate) from a subset of the Multi-Ethnic Study of Atherosclerosis (MESA) study participants (n = 2056, 53.6% female, age: mean 69.37 \pm 9.12, range 54 - 94) who underwent polysomnography. For each subject, we computed the sleep EEG multitaper spectrogram and extracted the total baseline-normalized SO-power (0.1 - 1.5 Hz). We next computed mean EEG spectral power as a function of SO-power, which we then tracked across all subjects as a function of age in sliding windows.

Results: The population analysis shows apparent, continuous changes in time-frequency domain features of the EEG as a function of a sleep depth along with age, that would be otherwise lost in traditional analyses. Moreover, by analyzing the directionality of the SO-power, we show that there is no apparent difference in neural activity during deepening sleep and lightening sleep; thus EEG sleep state is likely non-directional.

Conclusion: Our results show that state-based sleep dynamics of the EEG power spectrum can comprehensively be represented using SO-power as a surrogate of sleep depth. This representation identifies state-based activity independent of the temporal evolution of sleep architecture. As such, it is a powerful tool for analysis and phenotyping of EEG activity in large cohorts.

Support: The Biomedical Global Talent Nurturing Program through the Korea Health Industry Development Institute, funded by the Ministry of Health & Welfare, Republic of Korea (HI19C1065) to HK, National Institute of Neurological Disorders and Stroke (NINDS, R01 NS-096177) to MP.

0351

SLEEP CONTINUITY, SLEEP-RELATED DAYTIME DYSFUNCTION, AND PROBLEM ENDORSEMENT: DO THESE VARY CONCORDANTLY BY AGE?

Boyle, J. T.^{1,2} Rosenfield, B.¹ DiTomasso, R. A.¹ Vargas, I.^{3,4} Grandner, M.⁵ Perlis, M. L.²

¹Department of Clinical Psychology, Philadelphia College of Osteopathic Medicine School of Professional and Applied Psychology, Philadelphia, PA, ²Behavioral Sleep Medicine Program, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA, ³Department of Psychological Sciences, University of Arkansas, Fayetteville, AR, ⁴Sleep and Stress Research Laboratory, University of Arkansas, Fayetteville, AR, ⁵Sleep and Health Research Program, Department of Psychiatry, University of Arizona, Tucson, AZ.

Introduction: It is well documented that sleep continuity (i.e., SC [ability to initiate and/or maintain sleep]) worsens with age. It is unclear whether problem endorsement and/or daytime dysfunction show similar age-related trends. Accordingly, an analysis was undertaken to assess whether initial, middle, and/or late insomnia all exhibit age related change and whether problem endorsement and/or daytime dysfunction show comparable age-related changes. Methods: The study utilized a cross-sectional group design in an archival/community dataset (www.sleeplessinphilly.com). This dataset (N=932) was comprised of adults between 18 and 89 years of age with self-reported sleep complaints. Participants were categorized as: Young Adults (18-29 years); Adults (30-44 years); Middle Age Adults (45-64 years); and Older Adults (65-89 years). Age groups were matched to the Older Adults group (n=233)by sex, race, and BMI. ANOVAs with Bonferroni corrections (alpha = .001), and contingency analyses were performed to assess for age group differences.

Results: It was found that, as expected, SC worsens with age but that this was limited to middle and late insomnia. Further, problem endorsement increased with age (except for SL) but sleep-related daytime dysfunction did not (except for concentration issues).

Conclusion: These results have several implications. Methodologically speaking, when evaluating the effects and/or correlates of SC, it may be wise to concomitantly assay "is this a problem for you" and "does this affect your daytime function", as SC can occur without perceived daytime consequences, especially in older adults. Conceptually speaking, the observed discordance requires further exploration. In the past, it has been argued that sleep need is reduced in older adults. While this is a reasonable hypothesis (no need, no functional consequence), it remains to be demonstrated that older adults require less sleep. **Support:**

0352

A LARGE-SCALE EEG STUDY AT HOME TO OBJECTIVISE EFFECTS OF AGEING ON SLOW WAVE SLEEP AND PROCESS S.

El Kanbi, K.¹ Thorey, V.¹ Artemis, L.¹ Chouraki, A.¹ Trichet, T.¹ Pinaud, C.¹ Debellemaniere, E.¹ Arnal, P. J.¹ ¹Dreem, Paris, FRANCE, ²Dreem, Paris, FRANCE.

Introduction: Several studies have shown slow wave sleep (SWS) is altered with ageing. However, most of these studies have been conducted in-lab and usually over a single night. In this study, we assessed the evolution of process S with ageing by analysing the dynamics of endogenous and auditory-evoked slow waves in a large population.

Methods: 300 participants (200 M, 20 - 70 y.o.) were selected from volunteers users wearing a sleep headband for at least 3 nights, meeting the criteria of high signal quality and having no subjective sleep complaints nor being shift-workers. The Dreem headband is a connected device able to monitor EEG signals as well as pulse and movement and performs sleep staging in real-time automatically. Slow waves were detected as large negative deflections on the filtered EEG signals during NREM sleep. The auditory evoked slow waves were done using a previously validated closed-loop procedure.

Results: In our study, age was strongly correlated with N3 sleep duration (r=-0.34, p<0.0001), slow wave amplitude (r=-0.25, p<0.0001), and slow wave density (r=-0.40, p<0.0001). The slope of the slow wave activity, representing the process S here, was significantly decreased (r=-0.32, p<0.0001). This effect was mainly

due to changes in the density of slow waves in the first 2 hours of sleep (r=-0.41, p<0.0001). Finally, our results show a decrease in the probability of auditory evoked slow waves (r=-0.43, p<0.0001). **Conclusion:** These results confirmed the in-lab studies showing a heterogeneous alteration of homoeostatic process S with age, as well as a general decrease of slow wave occurrences, that is observed in parallel of a decrease of the probability of evoking slow waves, suggesting a global change in the system responsible for slow wave generation.

Support: This study was supported by Dreem sas and ANR, FLAG ERA 2015, HPB SLOW-Dyn

0353

OBJECTIVELY MEASURED SLEEP AND COMPONENTS OF METABOLIC SYNDROME IN WELL-FUNCTIONING OLDER ADULTS

Hossain, S.¹ Alfini, A. J.² Wanigatunga, A. A.³ Rojo-Wissar, D. M.² Schrack, J. A.⁴ Simonsick, E. M.¹ Zipunnikov, V.⁵ Wanigatunga, S. K.² Spira, A. P.²

¹National Institute on Aging, Baltimore, MD, ²Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ³Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ⁴Center on Aging and Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ⁵Department of Biostatistics, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ⁶Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD.

Introduction: Prior studies tying sleep to metabolic syndrome in older adults have mostly used self-report sleep measures. We investigated the association between actigraphic sleep parameters and metabolic syndrome components in well-functioning older adults. **Methods:** We studied 434 participants in the Baltimore Longitudinal Study of Aging (aged 71.1 \pm 12.8 years, 41.4% women) with 6.6 \pm 1.0 nights of wrist actigraphy and data on metabolic syndrome components: blood triglyceride (TG) level >150 mg/ dL; high density lipoprotein (HDL) <50 mg/dL; and waist circumference (WC) >88.9 cm for women and >101.6 cm for men. Sleep parameters were the primary predictors and metabolic syndrome components the outcomes. Logistic regression was performed, and results are expressed as odds ratio (OR) with p-values.

Results: After adjusting for age, sex, race and education, higher sleep efficiency (SEFF; per 10%) was associated with a lower odds of high WC (SEFF OR=0.60, p=0.01) and, compared to participants in the intermediate total sleep time tertile (5.5 to 6.8 h), those in the shortest tertile (<5.5 h) had a slightly lower odds of high WC (TST OR=0.98, p=0.02). In adjusted models, greater wake after sleep onset (WASO; per 30 min), greater average wake bout length (WBL; per min), and lower SEFF (per 10%) were associated with a greater odds of poor HDL level (<50 mg/dL) (WASO OR=1.37, p=0.05; WBL OR=1.49, p=0.007; SEFF OR=0.72, p=0.04). After further adjustment for BMI and depressive symptoms, only the association between longer WBL and poor HDL level remained significant (OR=1.48, p=0.01). There were no associations between sleep parameters and TG level.

Conclusion: Among well-functioning older adults, greater WASO but lower TST and SE are associated with poorer metabolic syndrome components. Longitudinal research is needed to evaluate the temporal associations of objectively measured poor sleep and metabolic syndrome components and evaluate the roles of BMI and depressive symptoms in these associations.

Support: The first author is supported by a Postdoctoral Fellowship by the Intramural Research Program (IRP) at the National Institute on Aging (NIA). This study was supported in part by National Institute on Aging (NIA) grant R01AG050507, the NIA Intramural Research Program (IRP), and Research and Development Contract HHSN-260-2004-00012C.

0354

AGE-RELATED SPECTRAL CHANGES IN NREM AND REM SLEEP IN MICE ARE GLOBAL AND NOT LOCAL

Dube, J.¹ Lina, J.¹ Soltani, S.² Chauvette, S.² Bukhtiyarova, O.² Carrier, J.¹ Timofeev, I.²

¹Center for advanced research in sleep medicine, CIUSSS du Nord de l'Ile de Montreal, Montreal, QC, CANADA, ²CERVO Brain Research Center, Laval University, Quebec, QC, CANADA.

Introduction: Brain topography modulates age-related changes in the human sleep electroencephalogram, which are linked with differences in integrity of specific cortical areas and may reflect local changes in sleep homeostasis. In mice, there is conflicting evidence regarding the topography of age-related changes for NREM and REM sleep. To disambiguate this issue, we investigated in mice the topography of age-related spectral differences for REM and NREM sleep.

Methods: LFP electrodes were implanted in 5 cortical areas and in the hippocampus of 17 C57/BL6 mice (8 young and 9 old, mean age = 7.5 and 16 months). Mice LFPs were recorded for a week and states of vigilance were semi-automatically detected in light and dark periods (12h-12h). Spectral analysis was run on 4s windows. Values were averaged for each electrode and in each period of the light/dark cycle in REM/NREM sleep for slow delta (0.25-2Hz), delta (2-4Hz), theta (4-8Hz), sigma (10-16Hz) and ripples (150-200Hz). Mixed models were computed separately for REM and NREM in dark and light period, with age as group factor and electrode and frequency as repeated factors.

Results: Two-way interactions were found between age and frequency and between electrode and frequency, for NREM and REM in dark and light periods. Each frequency band, except ripples, showed a topographical signature in NREM and REM (e.g. higher power in anterior compared to posterior areas for delta band in NREM sleep). These relative patterns did not change in older mice, but global changes occurred on all electrodes: in older mice, delta power was globally higher in NREM and REM sleep whereas sigma power was lower in REM sleep.

Conclusion: Age-related changes in spectral power of sleeping mice do not vary according to brain topography as in humans. Sleep deprivation studies are needed to investigate whether age is associated with global changes in sleep homeostasis in mice.

Support: This work has been supported by the Quebec Fonds de Recherche Nature et Technologies (FQRNT).

0355

NEUROBIOLOGICAL CORRELATES OF SLEEPINESS IN MIDDLE AGED AND OLDER ADULTS: A FDG-PET STUDY

Carvalho, D. Z. St. Louis, E. K. Schwarz, C. G. Bradley, B. F. Lowe, V. J. Przybelski, S. A. Reddy, A. Mielke, M. M. Knopman, D. S. Petersen, R. C. Jack, C. R. Vemuri, P. Mayo Clinic, Rochester, MN.

Introduction: Sleepiness has been associated with functional and cognitive decline, and may present with excessive daytime sleepiness (EDS) and/or increased sleep duration. We investigated

whether sleepiness and changes in sleep patterns are associated with FDG-PET levels in wake-promoting regions.

Methods: From the Mayo Clinic Study of Aging cohort, we identified 373 cognitively-unimpaired middle-aged and older adults (mean +/- s.d. 66.1 +/- 13.2 yo) who underwent FDG-PET. EDS was defined as ESS score >=10. Changes in sleep patterns (sleeping more, less, or no change) were assessed using question #16 of the Beck Depression Inventory-2. We used probabilistic maps to create regions of interest (ROIs): the locus coeruleus (LC), posterior lateral hypothalamus (PLH), and the basal forebrain divided in 1) medial septum/diagonal band of Broca (MS/DB) and 2) nucleus basalis of Meynert (nbM). FDG-PET levels were referenced to the pons (SUVR). In this cross-sectional analysis, we fit linear models to assess the association between EDS and changes in sleeping patterns with FDG SUVR in in each ROI, while controlling for age, sex, education, BMI, witnessed apneas, and cardiovascular risk factors.

Results: 10.5% had EDS, 15% reported sleeping more and 21% reported sleeping less than usual. 30.7% of participants with EDS reported sleeping more, 25.6% less, and 43.5% the same. EDS was associated with an elevation in FDG-PET SUVR in the MS/DB region (.035 [95% CI .008; .063], p=.012), while sleeping more was associated with a decrease in FDG-PET SUVR in the same region (-.027 [95%CI -.052; -.002], p=.036). Sleeping less was associated with an increase in FDG-PET SUVR in the PLH (.021 [95% CI .005; .03], p=.019). No associations were found in other ROIs.

Conclusion: Our results suggest that sleepiness and changes in sleep patterns in cognitively-unimpaired middle-aged and older adults were associated with measurable metabolic changes in areas of the brain involved in sleep and wakefulness. Further research should clarify whether these findings could represent different phenotypes of sleepiness with potential diagnostic and prognostic implications. **Support:** NIA/NIH

0356

ASSOCIATIONS BETWEEN ETHNICITY, SOCIOECONOMIC DEPRIVATION AND PRESCHOOLERS' SLEEP HEALTH IN AOTEAROA/NEW ZEALAND

Muller, D.¹ Paine, S.² Wu, L. J.¹ Signal, T. L.¹ ¹Massey University, Wellington, NEW ZEALAND, ²University of Auckland, Auckland, NEW ZEALAND.

Introduction: In Aotearoa/New Zealand (NZ) ethnic and socioeconomic inequities exist in adult sleep health but less is known about relationships between ethnicity, socioeconomic position and sleep in early childhood.

Methods: Maternally-completed questionnaire data from a pregnancy-birth cohort were analysed cross-sectionally. Logbinomial regression models investigated independent associations between ethnicity, socioeconomic position and sleep of 340 Māori (Indigenous) and 570 non-Māori 3-4 year olds. Independent variables included child ethnicity, gender, area-level deprivation (NZDep quintiles; 5=most deprived) and individual-level deprivation (NZiDep scores 1-5; 5=most deprived). Dependent variables included typical weekday and weekend sleep duration (>10hrs/10-13hrs), difference in week/weekend sleep duration (>1hr/ \leq 1hr) and midsleep time (\geq 1hr/<1hr), problems falling asleep and problematic sleep patterns (no vs. moderate/large problem).

Results: Māori preschoolers were more likely to have short sleep (weekdays: PR=2.23, 95% CI 1.31-3.82; weekends: PR=2.04, 95% CI 1.24-3.36), week/weekend sleep duration difference >1hr (PR=2.47, 95% CI 1.59-3.84), week/weekend midsleep difference ≥1hr (PR=2.38, 95% CI 1.30-4.36) and a moderate/ large problem falling asleep (PR=1.43, 95% CI 1.00-2.06) than non-Māori preschoolers. Preschoolers living in most deprived areas were more likely to have short sleep on weekdays (NZDep quintile 4: PR=3.91, 95% CI 1.43-10.72; NZDep quintile 5: PR=4.14, 95% CI 1.54-11.12) and week/weekend sleep duration difference >1hr (NZDep quintile 4: PR=2.34, 95% CI 1.23-4.43) than preschoolers in least deprived areas. Children with higher individual-level deprivation scores were more likely to have short sleep on weekends (NZiDep 5: PR=2.38, 95% CI 1.21-4.67) and a moderate/large problem falling asleep (NZiDep 3: PR=1.72, 95% CI 1.10-2.67) compared to children with lowest scores.

Conclusion: Ethnic and socioeconomic sleep health inequities exist as early as 3 years of age in NZ. Socio-political drivers of social and economic disadvantage experienced by Māori children and children from families who hold low socioeconomic position must be addressed to achieve equitable sleep health early in the lifecourse.

Support: Funding support was provided by Massey University New Zealand (Massey University Strategic Innovation Fund; Massey University Research Fund; and Massey University Doctoral Scholarship) and the Health Research Council of New Zealand (HRC 09/233, 08/547).

0357

CPAP ADHERENCE IS LOWER IN MINORITY NEIGHBORHOODS

Carmona, E. T.¹ Nouraie, S. M.¹ Bakker, J. P.² Stitt, C. J.² Aloia, M. S.² Patel, S. R.¹

¹Center for Sleep and Cardiovascular Outcomes Research, University of Pittsburgh, Pittsburgh, PA, ²Philips Respironics, Murrysville, PA. **Introduction:** The effectiveness of continuous positive airway pressure (CPAP) in treating obstructive sleep apnea (OSA) is limited by adherence. Small, single-center studies have reported CPAP adherence is lower in racial minorities suggesting disparities in OSA care. We used nationally representative data to assess racial differences in CPAP adherence at a neighborhood level.

Methods: Telemonitoring data were obtained from a therapy database maintained by a CPAP manufacturer. Usage over the first 90 days in patients initiated on CPAP between 11/01/2015 and 10/31/2018 who had at least one usage session, age 18-90 years, and valid U.S. zip code were mapped to a zip code tabulation area (ZCTA). Age- and sex-adjusted CPAP usage was calculated for each ZCTA with greater than 10 CPAP users. Ecologic analyses were performed to model the association of the proportion of blacks and Hispanics in each ZCTA (obtained from the 2013-2017 American Community Survey) on CPAP usage controlling for proportion of adults with bachelor's degree and proportion of adults with household income below the poverty line.

Results: Our analysis included 13,118 ZCTAs averaging data over 737,274 patients. In adjusted analyses, each 10% increase in the proportion of blacks and Hispanics was associated with a 0.12 (95% CI 0.11-0.12) hour and 0.14 (95% CI 0.14-0.15) hour decrease in nightly CPAP use, respectively. Mean usage in ZCTAs with <1%, 1-2.5%, 2.5-10%, 10-25%, and 25-100% blacks were 4.96, 4.81, 4.67, 4.56, and 4.14 hours respectively (p<0.001). Mean usage in ZCTAs with <1%, 1-2.5%, 2.5-10%, 10-25%, and 25-100% Hispanics were 4.87, 4.86, 4.75, 4.50, and 4.10 hours respectively (p<0.001).

Conclusion: CPAP adherence is lower in neighborhoods with higher proportions of black and Hispanic residents independent of differences in education or poverty. These differences lead to lower likelihood of meeting insurance coverage requirements for CPAP therapy, potentially exacerbating sleep health disparities.

Support: Philips Respironics, NIH R25HL130600 and K24HL127307.

0358

SLEEP AMONG TRANSGENDER AND CISGENDER ADOLESCENTS

Levenson, J. C.¹ Thoma, B. C.¹ Hamilton, J. L.¹ Choukas-Bradley, S.² Salk, R. H.¹

¹University of Pittsburgh School of Medicine, Pittsburgh, PA, ²University of Pittsburgh, Pittsburgh, PA.

Introduction: Sleep problems are germane to the majority of adolescents, but stigmatized youth may experience poorer sleep than those who have not experienced stigma. However, no prior studies have examined sleep among transgender adolescents (TGAs). Investigating these sleep disparities is critical, since low sleep duration and poor sleep quality are predictive of depressive symptoms and suicidality among adolescents, two mental health outcomes experienced disproportionately by TGAs. Using a nationwide survey of adolescents, we examined sleep duration, sleep quality, and rates of insufficient sleep among a sample of adolescents, and we compared those parameters between TGAs and cisgender adolescents (CGAs).

Methods: Adolescents (n=1784) ages 14-18 completed an anonymous survey including measures of sleep, sexual and gender identity, depressive symptoms, and demographic variables. Participants were grouped as TGA or CGA. Unadjusted associations between gender identity and sleep outcomes were examined, followed by multivariate regression models examining associations adjusted for demographics and depressive symptoms.

Results: TGAs reported sleeping fewer hours, higher odds of being a 'poor sleeper', and lower odds of getting the right amount of sleep and getting 'enough sleep' than CGAs. After adjusting for key demographic variables, TGAs were still more likely to report that they were poor sleepers and less likely to report getting enough sleep compared to CGAs. When adding depressive symptoms as a covariate, the finding that TGAs reported they got enough sleep

less often than CGAs remained marginally significant. **Conclusion:** Transgender adolescents reported receiving poorer sleep than cisgender adolescents. Future studies should focus on longitudinally examining the emergence of sleep problems among TGAs and CGAs, which may also serve to identify specific biopsychosocial pathways that contribute to heightened risk for sleep problems among TGAs. Sleep disparities may be a promising target for prevention and intervention programs to improve health outcomes among stigmatized youth.

Support: This study was funded by the University of Pittsburgh Central Research Development Fund through an award to Drs. Salk, Choukas-Bradley, and Thoma. Dr. Levenson was supported by grant K23HD087433. Dr. Thoma was supported by grants T32MH018951 and K01MH117142, Dr. Salk was supported by grant T32MH018269, and Dr. Hamilton was supported by grant T32HL082610.

0359

THE MODERATING ROLE OF RACE IN THE ASSOCIATION OF ADOLESCENT SLEEP DURATION AND MARIJUANA USE

Levenson, J. C.¹ Atuahene, B.¹ Bear, T.² Hacker, K.² Ricci, E.² Goldstein, T. R.¹ Miller, E.¹

¹University of Pittsburgh School of Medicine, Pittsburgh, PA, ²University of Pittsburgh Graduate School of Public Health, Pittsburgh, PA.

Introduction: Insufficient sleep and marijuana use during adolescence vary by race and are associated with poor outcomes in various domains. Sleep difficulties predict drug-related problems, but not all adolescents with insufficient sleep go on to use substances. We examined whether race/ethnicity moderates the association of sleep duration and marijuana use among Black/ African-American and White adolescents using a countywide probability-based survey of adolescents.

Methods: Using cross-sectional data (n=1447), logistic regression examined whether race moderated the association of adolescent sleep duration and recent marijuana use alone and after controlling for covariates in both weighted and unweighted models.

Results: Hours of sleep was significantly negatively associated with recent marijuana use. Black/African-American youth in our sample had up to a 60% increased odds of marijuana use in the past 30 days compared to White youth, and they reported significantly shorter sleep duration than their White peers. In weighted models, Black/African-American youth had an increased probability of marijuana use with fewer hours of sleep as compared to White youth, even after accounting for covariates.

Conclusion: In our countywide survey of adolescents, we found racial differences in the association of sleep duration and marijuana use. Future work should replicate our analyses with a longitudinal sample of adolescents to better evaluate the direction of these effects. Future efforts should also focus on identifying contextual factors that may explain racial differences in the sleep duration substance use relationship, as well as developing strategies to reduce disparities in this relationship. **Support:** The Heinz Foundation, the Hillman Foundation, the Grable Foundation, and the FISA Foundation. Dr. Levenson's effort was supported by NICHD.

0360

NEIGHBORHOOD DISADVANTAGE IS ASSOCIATED WITH LOWER QUALITY SLEEP AND MORE VARIABILITY IN WEEKNIGHT SLEEP DURATION AMONG URBAN ADOLESCENTS

Nahmod, N. G.^{1,2} Master, L.¹ McClintock, H. F.² Hale, L.³ Buxton, O. M.^{1,4,5}

¹The Pennsylvania State University, University Park, PA, ²Arcadia University, Glenside, PA, ³Stony Brook University, Stony Brook, NY, ⁴Harvard Medical School, Boston, MA, ⁵Brigham and Women's Hospital, Boston, MA.

Introduction: Differential social and contextual environments may contribute to adolescent sleep disparities. Yet, most prior studies are limited to self-reported sleep data, and the actigraphic studies of sleep are not conducted at a national level, thus limiting the variation in neighborhood contexts. This study examined the association between neighborhood disadvantage and actigraphic assessment of adolescent sleep.

Methods: Participants (682 adolescents, mean age 15.4 years) were racially/ethnically diverse (44% Black, 26% Hispanic, 17% White, 14% other race/ethnicity), sampled from 20 large US cities in the Fragile Families and Child Wellbeing Study. Neighborhood disadvantage was calculated from American Community Survey 2015 census data using the Standardized Neighborhood Deprivation Index (SNDI), consolidating five variables (proportion of female-headed households, public assistance recipients, households in poverty, adults without high school degrees, and unemployed) into an index. SNDI quartiles 1-3 fell below national averages of SNDI variables ("most disadvantaged") and were compared to quartile 4 ("least disadvantaged"). Sleep indicators (duration, quality, and timing) were measured over ≥ 5 nights using wrist-worn accelerometers. Separate multilevel models estimated differences in sleep indicators, adjusting for weekday/weekend and summer/school year. General linear models used within-person standard deviations of sleep indicators (controlling for number of days) to test for associations between neighborhood disadvantage and consistency of weeknight sleep patterns. Models adjusted for individual-level sociodemographic covariates (age, sex, race/ ethnicity, household income, caregiver education, and family structure).

Results: In fully adjusted models, adolescents living in more disadvantaged neighborhoods spent more time awake after falling asleep (4.0 minutes/night, p<0.05), spent greater percentage of nighttime sleep intervals awake (1%, p<.01), and had less consistent sleep duration (11.7% higher standard deviation, p<.05). Sleep duration and timing did not differ across neighborhood groups.

Conclusion: Living in more disadvantaged neighborhoods is associated with lower quality adolescent sleep; more research is needed to identify causal mechanisms.

Support: Research was supported by the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) of the National Institutes of Health (NIH), award numbers R01HD073352 (PI: Hale), R01HD36916, R01HD39135, and R01 HD40421, and private foundations. The content is the responsibility of the authors and does not represent official NIH views.

0361

BIRTHPLACE MODERATES RACIAL/ETHNIC DISPARITIES IN MULTIPLE SLEEP CHARACTERISTICS AMONG NON-HISPANIC WHITES AND HISPANIC/ LATINO HERITAGE GROUPS IN THE UNITED STATES

Gaston, S. A.¹ Martinez-Miller, E. E.^{2,3} Nguyen-Rodriguez, S.⁴ Aiello, A.⁵ McGrath, J.² Jackson, W.² Nápoles, A.⁶ Pérez-Stable, E. J.⁶ Jackson, C. L.^{1,6}

¹National Institute of Environmental Health Sciences, Research Triangle Park, NC, ²Social & Scientific Systems, Durham, NC, ³University of Texas Southwestern Medical Center, Dallas, TX, ⁴California State University Long Beach, Long Beach, CA, ⁵University of North Carolina at Chapel Hill, Chapel Hill, NC, ⁶National Institute on Minority Health and Health Disparities, Bethesda, MD.

Introduction: Sleep duration disparities by Hispanic/Latino heritage exist; however, few studies have additionally investigated sleep quality disparities by heritage and birthplace, nor have studies compared foreign-born to US-born Non-Hispanic Whites (NHWs).

Methods: Using pooled 2004-2017 National Health Interview Survey data, we investigated whether sleep disparities varied by birthplace among adult NHWs and Hispanic/Latino heritage groups. Adjusting for sociodemographic and behavioral/clinical characteristics, survey-weighted Poisson regressions with robust variance estimated prevalence ratios (PRs) and 95% confidence intervals (CIs) of self-reported sleep characteristics. Sleep characteristics were compared among foreign-born NHWs and Hispanic/ Latino heritage groups vs. US-born NHWs. Sleep characteristics were also compared across Hispanic/Latino heritage groups vs. foreign-born NHWs.

Results: Among 254,699 participants (Mean_{age} \pm SE: 47 \pm 0.9 years; 49% female), 81% self-identified as NHW (n=207,154), 12% Mexican (n=30,100), 2% Puerto Rican n=5,077), 1% Cuban(n=2,518), 1% Dominican (n=1,658), and 3% Central/South American (n=8,162). Compared to US-born NHWs, foreign-born NHWs were more likely to report >9-hours sleep duration (PR=1.11[95% CI: 1.01-1.21]) and poor sleep quality (e.g., PR_{trouble staying asleep}=1.27[1.17-1.37]), and US-born Mexicans were no more likely to report shorter sleep duration while foreign-born Mexicans were less likely (PR_{<6-hours}=0.52[0.47-0.57], PR₆-

 $_{\text{c7-hours}}$ =0.72[0.68-0.76]). Although US-born and foreign-born Mexicans had lower prevalence of poor sleep quality compared to US-born NHWs, PRs were lowest for foreign-born Mexicans. Compared to foreign-born NHWs, US-born Mexicans were more likely to report shorter sleep duration, but foreign-born Mexicans were more likely (e.g., PR_{<6-hours}=1.37[1.24-1.60]) and Cubans were less likely (e.g., PR_{<6-hours}=0.81[0.68-0.96]) to report shorter sleep duration vs. US-born NHWs. Compared to US-born NHWs, Dominicans reported better sleep duration and quality. Sleep duration and quality did not differ among Dominicans vs. foreign-born NHWs.

Conclusion: Sleep disparities varied by birthplace and Hispanic/ Latino heritage. Birthplace of both NHWs and racial/ethnic minority groups should be considered in disparities research.

Support: This work was funded by the Intramural Program at the National Institutes of Health (NIH), National Institute of Environmental Health Sciences (NIEHS, Z1AES103325-01) and the Division of Intramural Research, National Institute on Minority Health and Health Disparities.

0362

WOMEN ARE UNDERREPRESENTED IN MAJOR US SLEEP SOCIETIES RECOGNITION AWARDS Naime, S.¹ Karroum, E. G.²

¹Children's National Hospital, Washington, DC, ²George Washington University School of Medicine and Health Science, Washington, DC.

Introduction: Recognition awards are reflective of personal achievement and contribute to professional growth and academic promotion. Underrepresentation of women in recognition awards of various medical societies has been described. The Wavne A. Hening Sleep Medicine Investigator Award of the American Academy of Neurology was only given to two women since 2011. This finding prompted us to further investigate the gender distribution of major recognition awards in national US sleep societies. Methods: Publicly available lists of recognition awards recipients were retrieved and analyzed from the American Academy of Sleep Medicine (AASM) and the Sleep Research Society (SRS) websites. Recognition awards that reflect major contribution to the sleep field were included. The primary outcome measures were the overall proportion of women award recipients across selected recognition awards and the trend over time (1981-2019) analyzed by decade using the Cochran-Armitage test.

Results: We identified seven major sleep recognition awards (four by the AASM and three by the SRS) with a wide variation in the proportion of awards presented to women (4.8% to 31.3%). There were overall 184 individual awards presented by the two US sleep societies, including 154 (83.7%) awarded for men and 30 (16.3%) awarded for women. The analysis of the awards over time by decade revealed a significant increasing trend (P < 0.0001) in the proportion of awards recognizing women relative to men with a progression from 0.0% in the 1980s, to 3.0% in the 1990s, to 14.3% in the 2000s, and to 27.0% in the 2010s.

Conclusion: Overall in the last four decades, women have been underrepresented among major recognition awards from the AASM and the SRS, but it is encouraging to see a reduction in the gender gap, particularly over the last ten years. The reasons behind gender inequality in sleep recognition awards are unclear and need further investigation.

Support: This study was not funded.

0363

RACIAL/ETHNIC DIFFERENCES IN ACTIGRAPHY, QUESTIONNAIRE, AND POLYSOMNOGRAPHY-MEASURED INDICATORS OF SLEEP HEALTH AND SLEEP QUALITY: THE MULTI-ETHNIC STUDY OF ATHEROSCLEROSIS

Chung, J.¹ Goodman, M. O.¹ Huang, T.¹ Wallace, M.² Bertisch, S.¹ Johnson, D.³ Redline, S.¹

¹Brigham and Women's Hospital, Boston, MA, ²University of Pittsburgh, Pittsburgh, PA, ³Emory University, Atlanta, GA.

Introduction: Paradigm shifts in sleep research suggest the importance of considering multi-dimensional sleep health, compared to single metrics, to promote physical and mental well-being and to understand racial/ethnic disparities in sleep.

Methods: We used data from the Multi-Ethnic Study of Atherosclerosis (MESA; n = 1,740) to create a Sleep Health Score (SHS), including questionnaire (quality, sleepiness); 7-day actigraphy (total sleep time, sleep continuity [sleep maintenance efficiency], timing consistency [midpoint variability],

fragmentation, wake after sleep onset, sleep onset latency); and in-home polysomnography (%N3 sleep, %REM sleep, AHI). Sleep parameters were dichotomized based on prior literature or by healthiest quartile(s), with positive values denoting healthier sleep (e.g. Epworth scores < 10). All 11 dichotomized parameters were summed to calculate the SHS (mean=4.9, sd=1.58). We used modified Poisson and linear regression for individual sleep outcomes and the SHS, respectively, adjusting for age and sex.

Results: The sample was older (mean age=68.28, sd=9.08) and 54% female. SHSs were associated with Black race (β =-0.60 [-0.78, -0.42]) and Hispanic ethnicity (β =-0.40 [-0.59, -0.21]), but not Chinese ethnicity (β =-0.16 [-0.41, 0.08]). Compared to Whites (n=644), Blacks (n=485) showed lower adjusted probability of obtaining favorable levels of: sleep continuity, fragmentation, timing consistency, alertness/sleepiness, and sleep depth (%N3 sleep). Chinese respondents (n=202) had lower probability of obtaining favorable levels of: sleep continuity and timing consistency, but higher probability of quality. Hispanics (n=409) had lower probability of obtaining healthy levels of: sleep continuity, timing consistency, and fragmentation. Neither healthy total sleep time (middle quartiles) nor AHI (<30) differed by race/ethnicity.

Conclusion: Among MESA-Sleep participants, summary SHSs were lowest in Blacks, followed by Hispanics. Multiple dimensions of sleep - particularly related to continuity and timing consistency - were less favorable across race/ethnic minority groups. A summary SHS may help monitor sleep health across populations, while measurement of specific sleep components may help identify modifiable targets.

Support: Joon Chung is supported by a T-32 NIH training grant.

0364

SEX AND RACE INFLUENCE OBJECTIVE AND SELF-REPORT SLEEP AND CIRCADIAN MEASURES IN EMERGING ADULTS AT RISK FOR BIPOLAR SPECTRUM DISORDER

Titone, M. K.¹ McArthur, B.² Ng, T. H.¹ Burke, T. A.³ McLaughlin, L. E.¹ MacMullen, L. E.⁴ Alloy, L. B.¹ Goel, N.⁵ ¹Department of Psychology, Temple University, Philadelphia, PA, ²Department of Psychology, University of Calgary, Calgary, AB, CANADA, ³Department of Psychiatry and Human Behavior, Alpert Medical School Brown University, Providence, RI, ⁴Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, ⁵Biological Rhythms Research Laboratory, Department of Psychiatry and Behavioral Sciences, Rush University Medical Center, Chicago, IL.

Introduction: There is a critical need to understand key factors that impact sleep and circadian rhythm function for emerging adults at risk for bipolar spectrum disorder (BSD). Sex and race are common demographic factors contributing to differences in health outcomes; however, the influence of these variables on sleep and circadian rhythm patterns for emerging adults at risk for BSD has not been characterized.

Methods: Multiple objective and self-report facets of sleep and circadian function, including dim light melatonin onset (DLMO), and measures derived from actigraphy and sleep diaries, were assessed in a 20-day ecological momentary assessment (EMA) study of 150 emerging adults (mean \pm SD, 21.8 \pm 2.1y; 58.7% female; 57.9% White, 23.4% Black, 10.3% Asian or Pacific Islander, 8.0% Other ethnicity) at-risk for BSD. Bivariate Pearson correlations (two-tailed, p < .05) were conducted between the sleep and

circadian measures. ANCOVAs, controlling for BSD status, were conducted to evaluate differences on sleep and circadian characteristics by sex and race.

Results: Males exhibited better actigraphic sleep efficiency and later DLMO phase than females, whereas females exhibited more actigraphic discrete sleep periods. White participants exhibited more actigraphy-measured total sleep time, better sleep efficiency, and fewer sleep periods, and self-reported more total sleep time and better sleep efficiency than Black participants.

Conclusion: We show for the first time that sex and race are significant predictors of objective and self-reported sleep and circadian rhythm measures in a large sample of emerging adults at risk for BSD participating in an EMA study. Our findings extend the existing literature to a novel clinical population and to a naturalistic setting and inform ongoing research on sex and racial health disparities in sleep and circadian rhythms.

Support: This work was supported by NIH R01 MH77908 and R01 MH102310; a Banting Postdoctoral Fellowship from the Social Sciences and Humanities Research Council; and a National Science Foundation Graduate Student Research Fellowship.

0365

SLEEP QUALITY ASSOCIATED WITH PERCEIVED STRESS AND AUTONOMIC NERVOUS SYSTEM MEASURES: IMPLICATIONS FOR SLEEP AND HEALTH DISPARITIES

Ng, L.¹ Tkacs, N. C.² Richmond, T. S.² Hanlon, A. L.³ Grandner, M. A.⁴

¹Singapore General Hospital, Singapore, SINGAPORE, ²University of Pennsylvania, Philadelphia, PA, ³Center for Biostatistics and Health Data Science, Roanoke, VA, ⁴University of Arizona College of Medicine - Tucson, Tucson, AZ.

Introduction: It is not known whether the restorative nature of sleep mediates the effects of perceived stress on the autonomic nervous system (ANS). This study explored the relationships between stress and autonomic biomarkers, and aimed to assess potential mediation by sleep quality.

Methods: A secondary data analysis was performed using data from the Midlife in the United States (MIDUS, MIDUS II & Milwaukee). Multiple regression models examined the association between perceived stress using the Perceived Stress Scale (PSS) and two autonomic biomarkers-heart rate variability(HRV) (n=888) and urine catecholamines (n=1,058). The roles of sleep quality (using the Pittsburg Sleep Quality Index (PSQI)) and race/ethnicity were explored in post-hoc analyses.

Results: No statistically significant relationships were found between PSS and autonomic stress measures. However, perceived stress (b=0.09;p<0.01; 95% CI=0.06,0.13) and the covariate Likert stress scale at baseline (b=0.13;p=<0.04;95% CI=0.005,0.26) were significantly related to PSQI. Post hoc analyses explored racial differences. Baseline stress, PSS, depressive symptoms and PSQI scores were significantly higher in Blacks/African-Americans than Non-Hispanic Whites. Yet, Blacks/African-Americans had lower sympathetic responses (epinephrine means 1.56 vs 2.00;t=-4.82;p<0.01, norepinephrine means, 24.15 vs 27.30;t=3.14;p<0.01) and higher parasympathetic responses (lnHF, natural log of High Frequency HRV means 5.48 vs 4.75; t=6.17;p<0.01), compared to Non-Hispanic Whites.

Conclusion: Blacks/African-Americans and Non-Hispanic Whites had significant differences in their sleep quality and ANS biomarkers. Sleep quality may play a role in the effect of discrimination

on mental and physical health. Different stress sources may lead to variable expression in biomarkers of autonomic tone. Future prospective studies incorporating longitudinal biomarkers and alternative statistical models will help elucidate the relationships among stress, sleep, and the pathways linking these factors to poor health, and effect targeted treatments.

Support: The MIDUS I study was supported by the John D. and Catherine T. MacArthur Foundation Research Network. MIDUS II was supported by the NIA(P01-AG020166), M01-RR023942(Georgetown), M01-RR00865(UCLA) and 1UL1RR025011(UW) grants. Many thanks to Dr. James McNally, Dr. Barry Radler, Dr. Gayle Love and Suzanne Hodge for access to the Milwaukee dataset.

0366

SLEEP HEALTH DIMENSIONS, DISTURBANCES, AND DISRUPTORS AMONG WHITE, BLACK, HISPANIC/ LATINA, AND ASIAN WOMEN

Jackson, C. L.¹ Gaston, S. A.¹ McGrath, J.² Sandler, D. P.¹ ¹National Institute of Environmental Health Sciences, Research Triangle Park, NC, ²Social and Scientific Systems, Durham, NC.

Introduction: Despite the importance of sleep for health promotion and disease prevention, data are limited regarding the distribution of multiple sleep health dimensions, disturbances, and disruptors among women, especially racial/ethnic minorities who disproportionately experience poor sleep.

Methods: To determine the prevalence of sleep health, disturbances, and disruptors (e.g., short sleep duration, sleep debt, insomnia symptoms, light exposure at night) overall and among Black, Hispanic/Latina, and Asian compared to White women, we used cross-sectional data collected by the Sister Study at enrollment (2003-2009) and two follow-ups (2012-2014, 2014-2016). Adjusting for sociodemographics, health behaviors, and health conditions including depression, we used Poisson regression with robust variance to estimate prevalence ratios (PRs) for unfavorable sleep among racial/ethnic minority compared to White women.

Results: Of the 49,874 eligible women (mean age \pm standard deviation: 55.7 \pm 9.0 years, 84.8% \geq high school education, 74.7% married) 85.3% were White, 9.0% Black, 5.1% Hispanic/Latina, and 0.7% Asian. Overall, 70% reported the recommended amount of sleep, 15.7% inconsistent weekly sleep patterns, 26% sleep debt, and 14% insomnia symptoms plus short sleep. Racial/ethnic minorities were much more likely than whites to report very short (≤5 hours) sleep (PR_{Black})=5.98[95% Confidence Interval: 4.67-7.66]; $PR_{Latina} = 2.83[1.98-4.04]$; $PR_{Asian} = 5.41[2.41-12.13]$ and to report needing <7 hours to feel their best (PR_{Black} =2.95[2.75-3.17]; PR_{Latina}=1.85[1.65-2.07]; PR_{Asian}=2.66[2.10-3.37]). Black and Hispanic/Latina women had a higher prevalence than whites of insomnia, short sleep plus insomnia, inconsistent sleep, sleep debt, and frequent napping; however, all racial/ethnic minorities were less likely to report daytime sleepiness ($PR_{Black} = 0.82[0.78-0.85]$; PR_{Latina}=0.94[0.89-0.98]; PR_{Asian}=0.79[0.69-0.92]) and restless leg syndrome. Witnessed sleep apnea was higher among Black women, and REM sleep disorder did not differ across racial/ethnic groups. Sleeping with room lights or a television on was more prevalent among racial/ethnic minorities (PR_{Black}=1.78[1.71-1.86]; $PR_{Latina} = 1.27[1.17-1.37]; PR_{Asian} = 1.62[1.32-1.99]).$

Conclusion: Poor sleep health, disturbances, and disruptors were prevalent among women and varied across racial/ethnic groups in ways that may contribute to health disparities.

Support: This work was funded by the Intramural Program at the National Institutes of Health, National Institute of Environmental Health Sciences (Z1A ES103325-01 to (CLJ) and Z01 ES044005 to (DPS)).

0367

SOCIAL DETERMINANTS OF BLACK-WHITE DISPARITIES IN THE WORK-SLEEP RELATIONSHIP BY OCCUPATIONAL CLASS: A SEQUENTIAL MIXED METHODS APPROACH

Jackson, C. L.¹ Wright, I.² Winful, O. T.³ Feinstein, L.² Adams, I.⁴ ¹National Institute of Environmental Health Sciences, Research Triangle Park, NC, ²Social and Scientific Systems, Durham, NC, ³Vanderbilt University, Nashville, TN, ⁴Harvard T.H. Chan School of Public Health, Boston, MA.

Introduction: Although Black adults disproportionately work in lower-wage, lower-skilled jobs and experience short sleep (<7 hours), which has been shown to vary by employment industry and occupation, there is scant literature regarding the influence of the work-sleep relationship on racial/ethnic sleep disparities. Our prior quantitative research based on nationally-representative data revealed a novel finding that the prevalence of short sleep was generally highest at professional occupational classes among Black adults but was the least prevalent among their White counterparts.

Methods: To identify reasons for short sleep generally increasing with increasing professional occupations among blacks but decreasing among whites, we conducted a qualitative study using a sequential mixed methods design among Black and White workers across a range of industries and occupations. Occupations were classified as "professional" (e.g., doctors; lawyers) or "non-professional" (e.g., retail; food service). Racematched trained facilitators conducted 36 focus groups that were homogenous in terms of race-sex/gender-occupational class and 63 one-on-one interviews (N=334 overall participants) using semi-structured interview guides. NVivo software was used to identify themes/patterns.

Results: Participants were a mean age of 41 ± 11 years, 42% were men, 58% had an annual income of ≥\$50,000, and 57% were professionals. Black professionals overwhelmingly described less informational and emotional support as well as needing to "work twice as hard to get half as far" (i.e., John Henryism) compared to coworkers as potential explanations for work-sleep disparities. Both Black and White professionals identified longstanding social structures, interpersonal discrimination, income disparities, and familial or self-imposed pressures to succeed. White professional women frequently reported experiences with gender discrimination, which - they reported - may intersect with and amplify the effects of racial discrimination among Black women. Regardless of occupational class, Black men additionally described unique stressors (e.g., political climate; finances; police). White men frequently avoided discussing disparities, and the existence of disparities was often denied/questioned by non-professionals across race and sex/gender.

Conclusion: Our findings inform future research and interventions designed to illuminate and/or address sleep disparities emanating from the workplace.

Support: This work was funded by the Intramural Program at the National Institute of Environmental Health Sciences (Z1AES103325-01).

SLEEP HEALTH AMONG TRANSGENDER WOMEN OF COLOR IN NEW YORK CITY: PRELIMINARY ANALYSES OF THE TURNNT STUDY

Duncan, D.¹ Schneider, J.² Radix, A.³ Harry-Hernandez, S.⁴ Callander, D.⁵

¹Columbia University Mailman School of Public Health, New York, NY, ²University of Chicago, University of Chicago School of Medicine, IL, ³Callen-Lorde Community Health Center, Callen-Lorde Community Health Center, NY, ⁴Columbia University, Columbia University Mailman School of Public Healt, NY, ⁵Columbia University Mailman School of Public Health, Columbia University Mailman School of Public Health, Columbia University Mailman School of Public Health, NY.

Introduction: Little is known about sleep health among transgender and gender diverse populations. Even less is known about sleep among transgender women of color, a population that experiences considerable health disparity.

Methods: Interim baseline data were analyzed from the TURNNT (Trying to Understand Relationships, Networks and Neighborhoods among Transgender women of color) Study, an ongoing cohort of 350 HIV-negative transgender women of color in New York City. At baseline, items from the widely-used Pittsburgh Sleep Quality Index (PSQI) were used to measure typical sleep duration and subjective sleep quality. For example, typical sleep duration was measured with the PSQI item, "During the past month, how many hours of actual sleep did you get each night?". Participant responses were analyzed descriptively; the Mann-Whitney U test was used to assess bivariate associations.

Results: As of November 2019, there were n=31 participants enrolled in TURNNT. Nearly half of participants earned less than \$30,000 per year (48%) and in the 6 months before participation 61% had experienced food insecurity and 13% reported being unstably housed. Participants reported typically receiving 2-12 hours of sleep per night (median=6 hours). With short sleep defined as <7 hours per night, 55% reported this during the month prior to participation in the study, while 29% of participants rated their overall sleep quality as poor. Typical sleep duration was equivalent among participants with lower or higher incomes (5.5 vs 7 hours, p=0.3), but those who faced food insecurity reported less sleep (5 vs 7 hours, p<0.05) as did those who experienced housing instability (3.5 vs 7 hours, p<0.05).

Conclusion: Poor sleep health was common among our sample of transgender women of color, especially among those experiencing food insecurity and housing instability. Future research should examine multi-level correlates of poor sleep health such as network structures and neighborhood environments.

Support: The TURNNT (Trying to Understand Relationships, Networks and Neighborhoods among Transgender women of color) Study is funded through grants from the National Institute on Minority Health and Health Disparities (Grant Numbers: R01MD013554 and 3R01MD013554-02S1; Principal Investigator: Dustin T. Duncan, ScD).

0369

SLEEP DISPARITIES ARE ESTABLISHED BY THE FIRST MONTH OF COLLEGE

Bermudez, V. Fearon, D. Wheelis, M. Cohenour, M. Scullin, M. K. Baylor University, Waco, TX.

Introduction: Short and poor quality sleep are particularly common in college students, likely impacting their ability to persist and succeed in difficult courses. In the current study, we investigated demographic-based sleep differences (sleep disparities) and demographic-based academic differences (achievement gaps) in first-semester college students, with the goal of informing whether sleep disparities contribute to achievement gaps.

Methods: From 2017 to 2018, first-semester undergraduate students at Baylor University completed the New2BU Survey [N=6,048, 61.9% female, 18.7% first-generation, 23.8% underrepresented racial/ethnic minority (URM)]. Data collection occurred within three to five weeks of classes beginning. The survey included self-reported weekday total sleep time (TST), which we classified as short sleep (\leq 6.9 hours), normal sleep (7-9 hours), or long sleep (>9 hours). Semester GPA data were obtained from university records for students' first 4 semesters.

Results: There was evidence for both achievement gaps and sleep disparities. The risk for short sleep was increased in female students (p<.001; OR=1.20, 95%CI: 1.08-1.33), first-generation students (p=.02; OR=1.17, 95%CI: 1.03-1.33), and URM students (p<.001; OR=1.32, 95%CI: 1.16-1.50). The risk for long sleep increased substantially in first-generation students (p=.003, OR=1.92, 95%CI: 1.25-2.97) and URM students (p<.001; OR=2.41, 95%CI: 1.57-3.70), but not in female students (OR=0.88, 95%CI: 0.59-1.30). First-generation and URM students showed a 0.2-0.3 GPA reduction each semester relative to comparison groups (ps<.001), but short sleep and long sleep predicted GPA data up to four semesters later. Sleep-GPA correlations were modest in size (rs=.10-.14), but remained significant even after controlling for numerous demographic variables, high school GPA, and college entrance test scores.

Conclusion: Sleep disparities are noteworthy within the first month of college, and predictive of academic performance across four semesters. Addressing sleep health in all students—but particularly female, first-generation, and URM students—may increase academic success, bridge achievement gaps, and reduce health disparities.

Support: National Science Foundation (DRL 1920730)

0370

OBJECTIVE DIFFERENCES IN SLEEP TIMING BETWEEN AFRICAN AMERICANS AND NON-HISPANIC WHITES

Combs, D. Patel, S. Mashaqi, S. Estep, L. Provencio-Dean, N. Lopez, S. Parthasarathy, S. University of Arizona, Tucson, AZ.

Introduction: Prior studies have shown a morning circadian preference for African Americans compared to Non-Hispanic whites, but prior research has shown that self-reported sleep timing is delayed in African Americans compared to whites. No prior studies have evaluated this difference in sleep timing using objective measures of habitual sleep such as actigraphy.

Methods: We analyzed the sleep data from the Multi-Ethnicity Study of Atherosclerosis (MESA), a multi-site community-based cohort with both self-reported and actigraphic measures of habitual sleep variables. Self-reported sleep timing, chronotype as measured by the modified Horne-Ostberg Morningness-Eveningness Questionnaire (MEQ) and actigraphic sleep timing were examined using logistic regression and independent t-tests.

Results: 1,430 participants had self-reported sleep data, and 1,405 participants had complete actigraphy data as well. There was no self-reported difference in circadian preference between African

Americans and whites (mean MEQ score 17.1 ± 3.6 vs. 17.0 ± 4.0). African Americans were 76% more likely to report a bedtime after midnight on weekdays (odds ratio [OR] 1.8, 95% confidence interval [CI] 1.4, 2.3, p<0.001), and 50% more likely on weekends (OR 1.5, 95% CI 1.2, 1.9, p=0.001) as compared to whites. Actigraphic data showed similar results. African Americans were 80% more likely to have actigraphic sleep onset after midnight on weekdays (OR 1.8, 95% CI 1.4, 2.2) and 137% more likely (OR 2.4,95% CI 1.9, 3.0, p<0.001) on weekends as compared to whites. Actigraphic midsleep time was delayed 42 (95% CI 20, 64, p<0.001) minutes on weekdays and 24 (95% CI 6,42, p=0.01) minutes on weekends in African Americans compared to whites.

Conclusion: African Americans had delayed sleep timing compared to whites even though no significant racial differences in chronotype were present. Delayed sleep timing in African Americans may be related to factors other than self-reported circadian preference.

Support: Funding to DC from the American Heart Association, University of Arizona Health Sciences Center and NIH-NHLBI. MESA data obtained from NIH-NHLBI BioLINCC and the National Sleep Research Resource.

0371

EDUCATIONAL DISPARITIES IN U.S. ELEMENTARY SCHOOL CHILDREN ARE RELATED TO SLEEP DURATION AND BEDTIMES

Woods, A. D.^{1,2} Morgan, P. L.^{1,2} Jiao, J. L.³ Buxton, O. M.^{3,4,5} ¹Department of Educational Policy Studies, The Pennsylvania State University, University Park, PA, ²Center for Educational Disparities Research, The Pennsylvania State University, University Park, PA, ³Department of Biobehavioral Health, College of Health and Human Development, The Pennsylvania State University, University Park, PA, University Park, PA, ⁴Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, Boston, MA, ⁵Division of Sleep Medicine, Harvard Medical School, Boston, MA.

Introduction: Sleep is vital for healthy development in children. Suboptimal sleep health may play an under-appreciated role in educational achievement gaps among vulnerable student populations. Students of color, students from economically disadvantaged homes, and students with disabilities are especially likely to experience poor sleep. Sleep deficiency could interfere with school functioning, including academic achievement.

Methods: Longitudinal data from the nationally-representative Early Childhood Longitudinal Study (ECLS-K: 2011) included ~12,000 students. We tested the hypothesis that parent-reported child sleep duration (typical hours per night) was associated with academic achievement trajectories (3rd-5th grade). We further tested the extent to which this relation is linked to parentreported bedtimes in kindergarten. Preregistered analyses (osf. io) used structural equation path modeling, stratified by racial/ ethnic group (White, Black, Hispanic, Asian), disability status, and socioeconomic status (SES) tertiles. Students were assessed using psychometrically-validated standardized academic achievement tests.

Results: Children with later kindergarten bedtimes had shorter sleep duration across 3rd-5th grade. Children with shorter sleep duration also had poorer achievement in 3rd grade. The path by which sleep associates with achievement differed by vulnerable

subgroups. Among children from average- or high-SES families, earlier bedtimes were related to higher reading achievement growth across 3rd-5th grade, but not among children from low-SES families. For children with disabilities, longer sleep duration was significantly and positively associated with growth in reading achievement across 3rd-5th grade, but this relation among children with disabilities was not evident within racial or ethnic groups or SES strata.

Conclusion: Sleep duration, a modifiable behavioral factor, may be a promising target of intervention in families for promoting healthy childhood sleep health behaviors. Results provide evidence that age-appropriate bedtimes and adequate sleep duration could be promoted among vulnerable populations including students with disabilities. For instance, although most screening instruments do not currently evaluate sleep or sleep disorders, assessing and treating disability or behavioral difficulties could include such evaluations for clinical and parent consideration.

Support: Penn State Center for Educational Disparities Research

0372

DISPARITIES IN SLEEP TIMING IN THE US: DATA FROM THE NATIONAL HEALTH AND NUTRITION EXAMINATION SURVEY 2015-2016

Mota Villalobos, K.¹ Seixas, A. A.² Williams, N. J.² Jean-Louis, G.² Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹ ¹University of Arizona, Tucson, AZ, ²New York University School of Medicine, New York City, NY.

Introduction: Several studies have demonstrated population-level disparities in sleep duration and sleep quality. Population-level estimates of bedtime and waketime have been unavailable. Considering the important role of circadian rhythms in health, population-level disparities in timing have important public health implications.

Methods: Data from the 2015-2016 National Health and Nutrition Examination Survey (NHANES) from the CDC were used (N=4,491). Typical time in and out of bed were assessed and were converted to minutes. Race/ethnicity was self-reported and coded as non-Hispanic White, Black/African-American, Mexican-American, Other Hispanic/Latino, Asian, and Multiracial/Other. Covariates included age, sex, education level, income/poverty ratio, body mass index, and overall health. Additional models controlled for habitual sleep duration, frequency of sleep disturbance, depressed mood, and daytime tiredness/fatigue. Multiple linear regression analyses with time as an outcome were weighted using CDC-provided NHANES sample weights.

Results: In adjusted analyses, compared to non-Hispanic Whites, Blacks/African-Americans went to bed 29.4 mins later (p<0.0005), Asians went to bed 37.0 mins later (p<0.0005) and woke 27.7 mins later (p<0.0005), and Mexican-Americans woke 16.3 mins earlier (p=0.018). After further adjustment for sleep duration and sleep disturbances, Blacks/African-Americans went to bed 22.1 mins later (p<0.0005) and woke 22.2 mins later (p<0.0005), and Asians went to bed 36.1 mins later (p<0.0005) and woke 40.6 mins later (p<0.0005). These relationships remained generally unchanged when depressed mood and daytime tiredness/fatigue were adjusted in the model.

Conclusion: This is the first nationally-representative study to demonstrate population-level disparities in sleep timing. Specifically, Blacks/African-Americans and Asians present with delayed sleep, even after adjusting for other aspects of sleep.

Support: Dr. Grandner is supported by R01MD011600

BLACKS WITH OBSTRUCTIVE SLEEP APNEA REPORT GREATER NIGHTTIME INSOMNIA SYMPTOMS THAN WHITES, BUT DON'T ENDORSE DAYTIME IMPAIRMENT

*Williams, N. J.*¹ *Butler, M.*¹ *Roseus, J.*¹ *Barnes, A.*² *Blanc, J.*¹ *Bubu, O. M.*³ *Ebben, M.*⁴ *Grandner, M. A.*⁵ *Krieger, A. C.*⁴ *Jean-Louis, G.*³ *Perlis, M.*⁶

¹NYU Langone Health, Department of Population Health, Center for Healthful Behavior Change, New York, NY, ²New York Harbor Healthcare System, Brooklyn Campus, Brooklyn, NY, ³NYU Langone Health, Department of Population Health, Department of Psychiatry, New York, NY, ⁴Weill Cornell Medical Center, Center for Sleep Medicine, New York, NY, ⁵University of Arizona, College of Medicine, Tuscon, AZ, ⁶University of Pennsylvania, Perelman School of Medicine, Department of Psychiatry, Philadelphia, PA.

Introduction: Few studies have assessed insomnia severity in racial/ ethnic minority patients with OSA. In recognition of the burden of OSA in blacks compared to whites, the current study sought to examine insomnia symptoms in a sample of black and white patients newly diagnosed with OSA, prior to treatment, at 3 and 6 months.

Methods: 94 patients newly diagnosed with OSA provided demographics (age, sex, race/ethnicity), socioeconomic status, and completed the well-known and validated Insomnia Severity Index (ISI). To assess insomnia complaints, we ascertained total ISI score, nighttime sleep complaints, and daytime impairment. Linear regression and repeated measures analysis were conducted.

Results: Mean age was 57.43 years ± 13.55 ; 63.8% were men and 35% were black. Mean BMI was 32.35 ± 7.04 and 35% were diagnosed with hypertension. The mean ISI score for the total sample was 13.06 ± 7.06 . The total ISI was significantly higher in blacks than whites, respectively (M= 15.00 ± 7.17 ; M= 12.02 ± 6.83 , p<0.05) indicating moderate clinical insomnia in blacks, but not whites. In covariate-adjusted linear regression, nighttime complaints were statistically more pronounced in blacks (b=1.71, SE=0.82, p<0.05) and women (b=2.05, SE=0.72, p<0.01). There were no significant racial/ethnic differences with daytime impairment, but gender differences in daytime impairment remained (b=2.93, SE=1.04 p<0.01). Results from repeated measures effects of race over time revealed that blacks had higher nighttime complaints across all time-points (b=2.51, SE=1.10, p<0.05), but we did not observe a race-by-time interaction effect (b=-0.89, SE=0.50).

Conclusion: For the first time, we observed that overall ISI score and nighttime complaints are more pronounced in blacks than whites. Notably, only women endorsed complaints of daytime impairment. Findings from the study may contribute to understanding who will need treatment for relief of insomnia complaints. **Support:** K23HL125939

0374

DECLINE IN HABITUAL SLEEP DURATION OVER 10 YEARS AND WORSENING SLEEP DISPARITIES: DATA FROM NHIS (2006-2015)

Grandner, M. A.¹ Fernandez, F.¹ Khader, S.¹ Jean-Louis, G.² Seixas, A. A.² Williams, N. J.² Patterson, F.³ Killgore, W. D.¹ Wills, C. C.¹

¹University of Arizona, Tucson, AZ, ²New York University School of Medicine, New York City, NY, ³University of Delaware, Newark, DE. **Introduction:** Despite claims in the media, evidence that habitual sleep has declined in recent years is scant. Few data sources exist that systematically document sleep duration in a nationally representative sample, in the same way, over several years.

Methods: Data from 10 years of the National Health Interview Survey were used (N=305,555). During all years, habitual sleep duration, age, sex, race/ethnicity, and height/weight were recorded in the same way. Weighted regression analyses examined sleep duration as the outcome, year as linear predictor, and sociodemographics as covariates. Then, interaction terms examined whether the linear change associated with years was differentially experienced by different sociodemographic groups.

Results: The linear trend of sleep duration over the past 10 years is a loss of 0.78 minutes per year (95%CI -0.91,-0.64; p<0.0001). After adjustment for age, sex, race/ethnicity and BMI, this remained relatively unchanged at 0.86 minutes (95%CI -0.99,-0.73; p<0.0001). A year-by-race/ethnicity interaction was observed (p<0.05). In stratified analyses, Non-Hispanic Whites showed a loss of 0.68 minutes per year (95%CI -0.84,-0.52, p<0.0001). This was 1.33 minutes/year in Blacks/African-Americans (95%CI -1.74,-0.92; p<0.0001), 1.57 minutes/year in Mexican-Americans (95%CI -1.98,-1.16; p<0.0001), 0.99 minutes/year in other Hispanics/ Latinos (95%CI -1.51,-0.47; p<0.0001), 0.74 minutes/year in Asians (95%CI -1.24,-0.25; p=0.003), and 1.80 minutes/year in American Indians/Alaskan Natives (95%CI -3.57,-0.03, p=0.046).

Conclusion: On average, the US population has lost 47 seconds of nightly sleep per year over a 10-year period, equating to about 4.7 hours of sleep per year, but racial/ethnic groups were impacted differently. Compared to Non-Hispanic Whites, Blacks/African-Americans lost 96% more sleep, Mexicans lost 131% more sleep, other Hispanics/Latinos lost 46% more sleep, Asians lost 9% more sleep, and American Indians lost 165% more sleep. Thus, sleep disparities may be widening.

Support: Dr. Grandner is supported by R01MD011600

0375

CULTURALLY CONSISTENT DIET AMONG INDIVIDUALS OF MEXICAN DESCENT AT THE US-MEXICO BORDER IS ASSOCIATED WITH SLEEP DURATION AND QUALITY

Ghani, S. Delgadillo, M. E. Killgore, W. D. Wills, C. C.

Grandner, M. A.

University of Arizona, Tucson, AZ.

Introduction: Previous studies have shown that people who consume culturally consistent foods have improved cardiometabolic profiles. Few studies have examined whether this finding extends to sleep health.

Methods: Data were collected from N=100 adults (age 18-60, 53% female) of Mexican descent in the city of Nogales, AZ (66% not born in the US, 33% 1st-generation). Surveys were presented in English or Spanish. Acculturation was assessed with the Acculturation Scale for Mexican-Americans (ARSMA-II), which has separate scales for Mexican and Anglo acculturation (subscale range 0-4). A supplemental ARSMA item asks how often "My family cooks Mexican foods." Responses were coded as either frequent or infrequent. Insomnia was assessed with the Insomnia Severity Index (ISI), Sleepiness with the Epworth Sleepiness Scale (ESS), Sleep quality with the Pittsburgh Sleep Quality Index (PSQI), and Sleep duration and sleep medication use with PSQI items. Regression analyses examined these outcomes relative to whether individuals frequently consumed Mexican foods. Covariates included age, sex, and acculturation scores. Parental education level was also

included, as an indicator of childhood socioeconomic status and since food culture likely involves parents.

Results: Regular consumption of Mexican foods was associated with 1.41 more hours of sleep, on average (95%CI 0.19,2.62, p<0.05). It was also associated with a decreased likelihood of snoring (oOR=0.25; 95%CI 0.07,0.93; p<0.05). No differences were seen for PSQI, ISI, or ESS score.

Conclusion: Individuals of Mexican descent at the US-Mexico border who regularly consume culturally consistent food report overall more sleep and less snoring. Previous studies show that Mexican acculturation may be associated with improved sleep sufficiency; it is possible that this reflects an overall healthier lifestyle that also includes a culturally consistent diet. Further studies would be beneficial to help determine the role acculturation plays in sleep and diet and how it effects cardiometabolic risk.

Support: Dr. Grandner is supported by R01MD011600. This work was supported by a UAHS grant.

0376

DEMOGRAPHIC AND SOCIOECONOMIC IMPLICATIONS OF EXCESSIVE DAYTIME SLEEPINESS IN THE COMMUNITY

Begay, T.¹ Tubbs, A.¹ Jean-Louis, G.² Hale, L.³ Branas, C.⁴ Patterson, F.⁵ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹

¹University of Arizona, Tucson, AZ, ²New York University, New York, NY, ³Stony Brook University, Stony Brook, NY, ⁴Columbia University, New York, NY, ⁵University of Delaware, Newark, DE.

Introduction: Daytime sleepiness impairs daily functioning and may be directly related to insufficient nighttime sleep. Previous studies have assessed disparities in sleep duration and quality, but community-level disparities in daytime sleepiness using validated measures are lacking.

Methods: Data were from the Sleep and Healthy Activity, Diet, Environment, and Socialization (SHADES) study of N=1007 adults age 22-60. Daytime sleepiness was assessed with the Epworth Sleepiness Scale (ESS). Predictors included age, sex, race/ethnicity, education, and social class ("Upper middle class or above," "Middle class," "Lower middle class," "Poor," or "Very Poor"). One-way ANOVAs evaluated group differences. Stepwise linear modeling evaluated ESS score relative to sociodemographic predictors. Final models included all variables entered together to evaluate independent effects. Finally, habitual sleep duration was entered as an additional covariate.

Results: ESS score was higher among racial/ethnic minorities (p=0.0006), men (p<0.0001), those with less education (p=0.008) and lower social class (p=0.0007), and those who are retired or unable to work (p=0.03); marginal differences were seen according to age (p=0.06). Using a model-building approach, age, sex, race/ ethnicity, education, social class, and employment were evaluated. Only race/ethnicity (F=5.1, p=0.0004), education (F=4.8, p=0.003), and social class (F=2.14, p=0.046) incrementally added variance to model R². No 2-way interactions were found. In the final model, significant predictors included Black/African-American race/ethnicity (B=0.94, p=0.01), some college (B=0.99, p=0.005), and being very poor (B=2.16, p=0.005). When controlling for nocturnal sleep duration, the increased sleepiness associated with being Black/African was attenuated (p=0.06), but the other relationships were still significant.

Conclusion: There is a "sleepiness disparity" in the population associated with race/ethnicity and socioeconomics. Daytime sleepiness in the community is associated with being Black/

African-American, having some college, and being "very poor." The race/ethnicity difference in daytime sleepiness may be partially explained by differences in total sleep time.

Support: This work was supported by a grant from Jazz Pharmaceuticals. The SHADES study was funded by R21ES022931. Dr. Grandner is supported by R01MD011600.

0377

A STRENGTHS-BASED APPROACH TO EXAMINE OBSTRUCTIVE SLEEP APNEA IN BLACK AND WHITE PATIENTS

Williams, N. J.¹ Butler, M.¹ Roseus, J.¹ Blanc, J.¹ Barnes, A.² Bubu, O. M.³ Ebben, M.⁴ Krieger, A.⁴ Jean-Louis, G.³ ¹NYU Langone Health, Department of Population Health, Center for Healthful Behvior Change, New York, NY, ²Brooklyn Campus VA of the NY Harbor Health Care System, New York, NY, ³NYU Langone Health, Department of Population Health, Department of Psychiatry, New York, NY, ⁴Weil Cornell Medical Center, Center for Sleep Medicine, New York, NY.

Introduction: The majority of studies on race/ethnic disparities in OSA are derived from a deficit-based perspective (i.e. >BMI, non-adherence to PAP). It would prove useful to identify which aspects are protective to inform potential treatment approaches. We focused on two potential factors: resilience and social support, in patients newly diagnosed with OSA. Given the high prevalence of insomnia complaints in patients with OSA, insomnia was our outcome of interest.

Methods: 91 patients newly diagnosed with OSA provided demographic and socioeconomic status, sleep measures (Epworth, DBAS), resilience (Connor Davidson Resiliency Scale), social support (MOS Social Support Scale) and completed the Insomnia Severity Index. The cross-sectional associations between ISI, race/ ethnicity, resilience, social support and their interaction effects were examined using linear regression models with covariate adjustment for participant age, sex and BMI. We ascertained total ISI score and individual items.

Results: The sample was 34.1% black (n=31), mean age of 57.6 years, SD=13.6, 64.8% male (n=59), and mean BMI of 32.4, SD=7.04. Mean sleep duration (as reported by sleep diary) was 6.64, SD=1.35. Black, white differences were not observed for sleepiness (M=8.60; 10.43, p=0.11) or DBAS (M=4.61; M=5.04, p=0.30). Blacks, reported clinically significant insomnia (M=15.00, SD=7.17) compared to whites (M=12.02, SD=6.83, p=0.05). On the individual ISI items, blacks were significantly more likely to endorse difficulty falling asleep (M=1.58, SD=1.54; M=0.75, SD=0.93,p=0.002) and waking up too early (M=2.09, SD=1.26; M=1.45, SD=0.93,p=0.021) compared to whites. Resilience (M=30.04, SD=6.42) and social support scores (M=74.13, SD=21.36) did not differ by race/ethnicity. In adjusted linear analysis, resilience had significant effect on ISI score (b=-0.36, SE=0.12, p=0.003) but not social support (b=-0.06, SE=0.08, p=0.31).

Conclusion: In this study we did not observe race/ethnic differences for sleepiness and dysfunctional beliefs about sleep. With respect to the protective factors, race/ethnic differences were not observed. Resilience, not social support, was related to insomnia complaints. Future studies should examine a variety of factors that may serve black and other racial/ethnic groups with OSA, and help elucidate protective processes.

Support: K23HL125939

SOCIAL WELL-BEING AS A LONGITUDINAL MEDIATOR OF THE ASSOCIATION BETWEEN DISCRIMINATION AND SLEEP QUALITY

Dautovich, N. Ghose, S.

Virginia Commonwealth University, Richmond, VA.

Introduction: Discrimination is a risk factor for poor sleep outcomes. Physiological activation is one mechanism tying the experience of discrimination to disturbed sleep. Discrimination, however, can also impact psychosocial well-being, which is a necessary precursor for healthy sleep. Feelings of safety derived from social connections can be threatened when individuals face discrimination. The objective of the current study was to examine the role of social well-being as a factor underlying the longitudinal association between discrimination and sleep quality.

Methods: An archival analysis was conducted with 937 adults participating in the longitudinal Midlife in the United States (MIDUS) study. Data was collected at three time points across 10 years. Perceived daily discrimination and overall social well-being were assessed via self-report. Sleep quality was assessed via the Pittsburgh Sleep Quality Index, Global Sleep Quality score. **Results:** The overall model accounted for 15.6% of variance in global sleep quality. Controlling for multiple covariates, more frequent experiences of discrimination predicted worse global sleep quality 10 years later (β =.06, *p*=.03). Worse overall social well-being was a significant mediator of the discrimination-global sleep quality association (95% CI [.0001, .0118]), such that more frequent discrimination predicted lower overall social well-being, which, in turn, was associated with worse global sleep quality.

Conclusion: Given the persistence of sleep disparities among stigmatized and marginalized groups and the importance of sleep as a means of health disparity reduction, there is a need to identify mechanisms linking discrimination to poor sleep outcomes. Daily experiences of discrimination, such as being given less respect or treated as though less intelligent, have long-lasting associations with social well-being. Furthermore, social well-being is a predictor of future sleep quality. In addition to addressing discriminatory practices, targeting the effects of social well-being on sleep is a direction for future research.

Support: N/A

0379

GREEN SPACE EXPOSURE AND SLEEP DURATION AMONG SUPPLEMENTAL NUTRITION ASSISTANCE PROGRAM PARTICIPANTS

Grigsby-Toussaint, D. Shin, J.

Brown University School of Public Health, Providence, RI.

Introduction: Emerging empirical evidence suggests green space exposure is protective against insufficient sleep. Limited studies exist, however, exploring the relationship between greenspace exposure and sleep among low income populations in the United States.

Methods: Using a sample of Supplemental Nutrition Assistance Program (SNAP) participants (n=104) recruited from Champaign County, Illinois, we examined the relationship between self-reported sleep duration and exposure to green space. Sleep duration was determined using the question, "On average, how many hours did you sleep each night during the past 4 weeks?" Green space exposure was determined using satellite imagery from the National Aeronautic Space Administration (NASA) normalized difference vegetation index (NDVI). An NDVI score was assigned to each participant based on residential geo-referenced data. Multiple linear regression was performed in SPSS to explore the relationship between self-reported sleep duration and green space.

Results: Higher number of hours of sleep within a 24-hour period was positively associated with higher scores for greenspace exposure (β =0.091, P=0.02), controlling for age, gender, smoking status, education, alcohol consumption, and sleep quality. Sleep duration was negatively associated with age (β =-0.03, P=0.007), but positively associated with sleep quality (β =0.856, P=0.008).

Conclusion: In a sample of SNAP participants, exposure to green space was associated with more hours of sleep per night. Additional studies with larger, and more geographically diverse samples of low income adults are needed to determine whether this relationship is robust.

Support: USDA UNC/DUKE BECR Center

ASSOCIATION OF DAY-TO-DAY VARIABILITY IN REST-ACTIVITY CIRCADIAN RHYTHM WITH SLEEP QUALITY AMONG LAW ENFORCEMENT OFFICERS

Fekedulegn, D.¹ Service, S.¹ Ma, C.² Gu, J.¹ Violanti, J.³ Andrew, M.¹

¹Statistician, CDC/NIOSH, WV, ²Epidemiologist, CDC/NIOSH, WV, ³Professor, University at Buffalo, NY.

Introduction: Poor sleep quality may be attributed to several occupational factors and has been linked to adverse health outcomes, including cardiovascular disease. Recent epidemiologic studies suggest rest-activity circadian rhythm (RAR) as a possible determinant of poor sleep quality. The focus of these studies has been on the magnitude of the parameters of RAR with little attention to the impact of their day-to-day fluctuation. We examined association of daily variation in parameters of RAR with sleep quality.

Methods: Participants (n=280) were officers from the Buffalo Cardio-metabolic Occupational Police Stress Study (2004-2009). Sleep quality was determined using the Pittsburgh Sleep Quality Index (PSQI). Participants wore wrist actigraph for a minimum of seven days. A cosine curve was fit to measure goodness of fit and estimate the mean values of the three parameters of RAR: Mesor, Amplitude, and Acrophase. Dayto-day variability of the parameters were assessed by fitting the cosine function separately for each day and computing the sample standard deviation across the days. Poisson regression models were conducted adjusting for demographic, lifestyle, and occupational factors.

Results: The prevalence of poor sleep quality was 50.3%. Poor sleep quality was 56% higher in officers with the largest day-to-day variability in Mesor (PR=1.56, 95%CI:1.11 - 2.19) compared to those with the lowest daily variation. Similar estimates were found for Amplitude (PR=1.42, 1.03 - 1.95), Acrophase (PR=1.86, 1.29 - 2.67), and measure of goodness of fit (PR=1.54, 1.13 - 2.11). On the other hand, mean values of RAR parameters were not significantly associated with poor sleep quality.

Conclusion: Results suggest larger daily variation in parameters of RAR is associated with a decrease in sleep quality. Given that day-to-day variation in RAR may increase the odds of poor sleep quality, future studies ought to address risk factors for higher daily fluctuations in RAR which could aid in developing intervention measures.

Support: CDC/NIOSH grant 1R01OH009640-01A1

0381

ASSOCIATION OF SOCIAL JETLAG WITH THE 2018 WCRF/AICR CANCER PREVENTION SCORE AMONG PARENT-ADOLESCENT DYADS IN THE FLASHE STUDY

O'Connor, S. G. Shams-White, M. M. Czajkowski, S. Oh, A. Division of Cancer Control and Population Sciences, National Cancer Institute, Bethesda, MD.

Introduction: Social jetlag (SJL), or the mismatch between biological and social time, is a marker of circadian misalignment. SJL is linked to biological and behavioral alterations (e.g., adiposity, unhealthy food intake) which over time may increase cancer and chronic disease risk. While developmental changes exacerbate SJL among adolescents, parents may also influence adolescent behavioral patterns. This study examined associations of SJL with multiple cancer preventive behaviors among parent-adolescent dyads

using the 2018 World Cancer Research Fund/American Institute of Cancer Research (WCRF/AICR) Cancer Prevention Score in the National Cancer Institute's Family Life, Activity, Sun, Health, and Eating (FLASHE) Study.

Methods: Online cross-sectional surveys examining cancerpreventive behaviors were administered to 1,479 parent-adolescent (aged 12-17y) dyads in 2014. SJL was defined as the absolute difference between sleep midpoint on weekend and week days. The adapted 2018 WCRF/AICR Score ranged from 0-6 and was the sum of six sub-components representing adherence to specific cancer prevention recommendations concerning body weight, physical activity, and diet. Dyads with missing data on either variable were excluded. Actor-partner interdependence modeling was used to determine within-dyad correlations, actor (i.e., effect of a dyad member's SJL on own score), and partner (i.e., effect of a dyad member's SJL on partner's score) effects.

Results: The analytical sample included 936 dyads. Mean SJL was 2.0 hours (SD: 1.2) for adolescents and 1.2 hours (SD: 0.9) for parents. Mean WCRF/AICR Scores were 4.1 (SD: 0.9) for adolescents and 4.0 (SD: 0.9) for parents. Within dyads, SJL (r=0.29) and the WCRF/AICR Score (r=0.41) were positively correlated (all p <0.0001). Among adolescents, greater SJL was associated with a lower WCRF/AICR Score (β = -0.077, SE: 0.03, p=0.003), but this relationship was not observed among parents. No partner effects were detected.

Conclusion: Among adolescents, SJL was associated with poorer adherence to cancer prevention recommendations. SJL may represent a modifiable risk factor with the potential to improve multiple health behaviors and decrease disease risk.

Support: FLASHE was funded with federal funds from the National Cancer Institute, National Institutes of Health (NIH), under contract number HHSN261201200039I issued to Westat, Inc

0382

SEX DIFFERENCES IN SLEEP AND QUALITY OF LIFE IN HEALTHCARE SHIFT WORKERS

Lammers-van der Holst, H. M.^{1,2} Zhang, Y.³ Barger, L. K.^{1,2} Wise, J. C.¹ Murphy, A. S.¹ Desnoyers, B. M.¹ Qadri, S.¹ Ronda, J. M.^{1,2} Duffy, J. F.^{1,2}

¹Division of Sleep and Circadian Disorders, Brigham and Women's Hospital, Boston, MA, ²Division of Sleep Medicine, Harvard Medical School, Boston, MA, ³Solomont School of Nursing, Zuckerberg College of Health Sciences, University of Massachusetts Lowell, Lowell, MA.

Introduction: Shift work is associated with insufficient and disrupted sleep and impaired quality of life due to misalignment between the timing of internal biological clock and work/sleep schedule. There are reported sex differences in the circadian timing system, in sleep, and in reported sleep complaints, but how these impact female shift workers remains unclear. Furthermore, relatively little is known about sex differences in quality of life in shift workers. The objective of this study is to investigate sex differences in sleepiness, insomnia and quality of life in healthcare shift workers.

Methods: Forty women $(31 \pm 6.4 \text{ yo})$ and 70 men $(31.2 \pm 6.7 \text{ yo})$ who work at least 4 night shifts a month, completed the Shift Worker Sleep and Health Survey. This REDcap administered survey included the Epworth Sleepiness Scale (ESS), Insomnia Severity Index (ISI), and Functional Outcomes of Sleep Questionnaire (FOSQ-10). Independent samples T-tests were carried out using SAS.

Results: Women reported significantly higher scores on the ESS compared to men $(13 \pm 4 \text{ vs. } 11 \pm 3.4, \text{ respectively; } t(108)=-2.74,P=0.007)$, as on the ISI $(13 \pm 5.2 \text{ vs. } 9.8 \pm 5.4; t(108)=-3.05,P=0.003)$, indicating greater levels of daytime sleepiness and insomnia-like symptoms in female shift workers. In addition, women scored significantly lower on the FOSQ-10 than men $(13.9 \pm 2.9 \text{ vs. } 15.3 \pm 2.5; t(108)=-2.68,P=0.009)$, suggesting a lower functional status related to activities of daily living in female shift workers.

Conclusion: These preliminary analyses suggest that in this group of healthcare shift workers, women are less tolerant to irregular work hours compared to men, in terms of sleep, sleepiness and quality of life. Our future goal is to understand how sleep quality and duration, daytime sleepiness, and quality of life interact, and what role sex plays in those interactions.

Support: The study was supported by NIH grant R01 AG044416.

0383

OUTDOOR ARTIFICIAL LIGHT AT NIGHT, SLEEP DURATION, AND SLEEP QUALITY IN THE CALIFORNIA TEACHERS STUDY COHORT

Zhong, C.^{1,2} Franklin, M.² Wiemels, J.² Chung, N.¹ Benbow, J.¹ Wang, S. S.¹ Lacey, J. V.¹ Longcore, T.³

¹City of Hope, Duarte, CA, ²University of Southern California, Los Angeles, CA, ³University of California, Los Angeles, Los Angeles, CA.

Introduction: Artificial light at night (ALAN) is believed to disrupt sleep by suppressing melatonin and altering normal circadian patterns. We assessed the association between self-reported sleep measures and outdoor ALAN in a large cohort of women.

Methods: The California Teachers Study (CTS) is a prospective cohort of 133,479 current and former Californian female public school professionals recruited and given a baseline questionnaire in 1995-1996. A follow-up questionnaire in 2012-2014 assessed selfreported measures of sleep habits, quality, and chronotype. Using geocoded residential addresses, participants were assigned exposures to outdoor ALAN based on the New World Atlas of Artificial Night Sky Brightness to assess the association between ALAN and self-reported sleep initiation, duration, and quality.

Results: Of the 42,706 women who completed the follow-up questionnaire and reported the same sleep patterns over the previous year, 5,968 reported poor sleep quality. The median outdoor ALAN was 2.16 (IQR: 1.04-3.61) millicandela per meter squared (mcd/m²). After adjusting for self-reported chronotype, use of sleep medication, age, race, and socioeconomic status, residing in the highest ALAN quintile was associated with poor sleep (OR 1.16, 95% CI 1.07-1.26). There did not appear to be an association between outdoor ALAN and time taken to fall asleep (OR 1.02, 95% CI 0.96-1.08), but it was associated with sleeping less than 8 hours (OR 1.41, 95% CI 1.33-1.50).

Conclusion: CTS participants who self-reported shorter sleep and poorer sleep quality were more likely to reside in areas with greater levels of outdoor ALAN. We did not see an association with ALAN and time to fall asleep, suggesting ALAN may be contributing to later sleep time or earlier waking. As we continue to follow this cohort, the data collected over the past 20 years provide a rich resource for studying both factors related to sleep and its effect on health.

Support: The California Teachers Study and the research reported in this publication were supported by the National Cancer Institute of the National Institutes of Health under

award number U01-CA199277; P30-CA033572; P30-CA023100; UM1-CA164917; R01-CA077398; and R01-CA207020.

0384

DO NURSES WITH HIGH BLOOD PRESSURE HAVE MORE SLEEP DISTURBANCES THAN THEIR PEERS?

Santiago, B. P.¹ Messman, B. A.¹ Slavish, D. C.¹ Alkire, C.¹ Wardle-Pinkston, S.² Dietch, J. R.³ Kelly, K.¹ Ruggero, C. R.¹ Taylor, D. J.²

¹University of North Texas, Denton, TX, ²University of Arizona, Tuscon, AZ, ³Palo Alto Veterans Affairs Health Care System, Palo Alto, CA.

Introduction: Nurses work in stressful environments and often have rotating work schedules, which may put them at risk for disturbed sleep and health. Poor quality and short sleep duration are strong risk factors for high blood pressure (HBP). Yet few studies have examined these associations in nurses, who may be a particularly at-risk sample. To address this gap, we examined group differences in self-reported and actigraphy-assessed sleep among nurses with and without self-reported HBP.

Methods: Participants were 392 nurses (91.8% female; 77.8% white, mean age = 39.54) recruited for a parent study. Participants completed baseline questionnaires including the Pittsburgh Sleep Quality Index (PSQI), followed by 14 days of actigraphy and sleep diaries to prospectively assess 14-day mean total sleep time (TST) and sleep efficiency (SE). An independent samples t-tests was used to assess group differences in sleep variables by HBP status. Linear regression was used to further examine the association between HBP status on sleep variables when controlling for age, race, gender, ethnicity, and body mass index (BMI).

Results: Twenty-nine (7%) nurses endorsed having clinicallydiagnosed HBP. Nurses with HBP had higher global PSQI scores (indicating worse sleep quality; t=2.71, p=0.007), compared to nurses who did not report HBP, with a mean difference of 1.24. When adjusting for covariates, the association between HBP and the PSQI became marginally significant (p=0.054). There were no group differences in sleep diary or actigraphy TST or SE by HBP status, nor did HBP predict these sleep variables when controlling for covariates.

Conclusion: We found that nurses who reported having clinically diagnosed HBP had poorer global sleep quality. Although limited by self-reported history of HBP diagnosis, and low endorsement of HBP in our sample, our results corroborate other findings which suggest there is a strong association between high blood pressure and disturbed sleep. Future studies should examine these associations in larger samples, assess blood pressure directly, and experimentally examine the effects of HBP treatment on sleep quality. **Support:** NIH/NIAID R01AI128359-01

0385

RISK ASSESSMENT OF SLEEP DISORDER COMORBIDITY ACROSS ACTIVE DUTY ARMY INSTALLATIONS FROM MILITARY MEDICAL DATABASES

Brager, A. J. Hosamane, N. Capaldi, V. Simonelli, G. Walter Reed Army Institute of Research, Silver Spring, MD.

Introduction: The impact of sleep disorders on active duty Soldiers' medical readiness is clinically significant. Sleep disorders present high comorbidity with disease states directly impacting medical readiness, ranging from musculoskeletal injury (MSK-I), obesity,

and drug dependence. The current study performed a risk assessment of sleep disorder comorbidity with MSK-I, obesity, and drug dependence across active duty United States Army installations.

Methods: Health incidences (percent active duty per installation) were queried from the Office of the Surgeon General Health of the Force (HoF) report, specifically for Fiscal Year (FY) 2017 (n = 471,000; 85.5% male, > 70% between 18 -34). Nonparametric ranked tests identified active duty Army installations at low risk (green; < 25% percentile relative to mean rank), moderate risk (amber; 25% - 50%), and high risk (red; > 75% percentile). Linear regressions determined extent of comorbidity of sleep disorders with MSK-I, obesity, and drug dependence (tobacco use and substance abuse).

Results: Mean rank comparisons for sleep disorders vs. injury index (p=0.499), obesity (p=0.306), tobacco use (p=0.378), and substance abuse (p=0.591) did not differ for each installation. Further, there was a high degree of co-morbidity for mean percentage of diagnosed sleep disorder with injury index (p<0.001; $r^2 = 0.517$), obesity (p<0.001; $r^2 = 0.963$), tobacco use (p<0.001; $r^2 = 0.928$), and substance abuse (p<0.001; $r^2 = 0.968$).

Conclusion: In general, large infantry and artillery training units located in the Southeastern United States were "in the red" for not meeting medical readiness standards. A few exceptions include Virginia-Maryland triangle, a heavily populated area. These data demonstrate strong geographical influences on health risk comorbidity in active duty Soldiers comparable to civilian sectors. **Support:** Military Operational Medicine Research Program

0386

IDENTIFICATION OF SLEEP COMPLAINTS USING SOCIAL MEDIA: EFFECT OF THE DAYLIGHT SAVINGS TIME TO STANDARD TIME TRANSITION

Veatch, O. J. Mazzotti, D. R.

University of Pennsylvania, Philadelphia, PA.

Introduction: Transitions to and from daylight savings time (DST) are natural experiments of circadian disruption and are associated with negative health consequences. Yet, the majority of the United States and several other countries still adopt these changes. Large observational studies focused on understanding the impact of DST transitions on sleep are difficult to conduct. Social media platforms, like Twitter, are powerful sources of human behavior data. We used machine learning to identify tweets reporting sleep complaints (TRSC) during the week of the standard time (ST)-DST transition. Next, we evaluated the circadian patterns of TRSC and compared their prevalence before and after the transition.

Methods: Using data publicly available via the Twitter API, we collected 500 tweets with evidence of sleep complaints, and manually annotated each tweet to validate true sleep complaints. Next, we calculated term frequency-inverse document frequency of each word in each tweet and trained a random forest to classify TRSC using a 3-fold cross-validation design. The trained model was then used to annotate a collection of tweets captured between Oct. 30, 2019-Nov. 6, 2019, overlapping with the DST-ST transition, which occurred on Nov. 3, 2019.

Results: Random forest demonstrated good performance in classifying TRSC (AUC[95%CI]=0.85[0.82-0.89]). This model was applied to 3,738,383 tweets collected around the DST-ST transition, and identified 11,044 TRSC. Posting of these tweets had a circadian pattern, with peak during nighttime. We found a higher frequency of TRSC after the DST-ST transition (0.33% vs. 0.27%,

p<0.00001), corresponding to a ~20% increase in the odds of reporting sleep complaints (OR[95%CI]=1.21[1.16-1.25]).

Conclusion: Using machine learning and Twitter data, we identified tweets reporting sleep complaints, described their circadian patterns and demonstrated that the prevalence of these types of tweets is significantly increased after the transition from DST to ST. These results demonstrate the applicability of social media data mining for public health in sleep medicine.

Support: NIH (K01LM012870); AASM Foundation (194-SR-18)

0387

EFFECT OF AIR POLLUTION ON THE QUALITY OF SLEEP - KOREAN NATIONWIDE CROSS-SECTIONAL STUDY

Hong, S.

St. Vincent's Hospital, College of Medicine, The Catholic University of K, Suwon, KOREA, REPUBLIC OF.

Introduction: Poor sleep quality is associated with adverse health outcomes, such as cardiovascular disease, diabetes and obesity. Several studies have indicated the association between exposure to air pollution and sleep quality. However, the evidence is very limited in South Korea.

Methods: We conducted a cross-sectional study using data from a nationwide sample of 165,193 individuals aged 19 years or older from the 2018 Korea Community Health Survey. Perceived air pollution was measured in the baseline survey. Sleep quality was evaluated using the Pittsburgh Sleep Quality Index (PSQI). Poor sleep quality was defined by the global score of PSQI > 5. The associations between perceived air pollution and sleep quality were examined using the regression models. Additionally, the average concentration of PM2.5 was measured in each region using a satellite-based prediction to see the association between perceived air pollution and long term exposure of fine dust.

Results: After adjusted for age, education, and income, the adjusted odds ratios (95% confidence interval) of poor sleep quality associated with perceived air pollution was 1.45. The average of daily concentrations of PM2.5 over 3 years was highest in Incheon area, followed by Seoul and Busan city. Among Gyeonggi Province, the most populous region in South Korea, the PM2.5 concentrations was the highest in Ansan. In each region in Korea, Incheon had the highest poor sleep quality rate of 48.0%, while Ansan in Gyeonggi Province had the highest rate of 61.5%. The results are in line with the distribution of PM2.5 concentrations by region.

Conclusion: Perceived air pollution and long term exposure of fine dust were associated with poor sleep quality in South Korea. Improvement of air quality may help to improve sleep quality. More studies are in need in the future to explore the biological mechanism for the relationship, and to also examine such relationship among different populations and in difference environments. **Support:** For this study, we used raw data from the 2018 Korea Community Health Survey (KCHS), conducted by the Korea

Community Health Survey (KCHS), conducted by the Korea Centers for Disease Control and Prevention

0388

PREDICTORS OF INCIDENT REDUCED SLEEP EFFICIENCY IN COMMUNITY-DWELLING OLDER WOMEN

Vo, *T.*¹ Blackwell, *T.*² Kats, *A.*¹ Langsetmo, *L.*¹ Taylor, *B.*^{1,3} Schousboe, *J.*^{1,4} Redline, *S.*⁵ Stone, *K.*² Smagula, *S.*⁶ Chu, *H.*¹ Rodriguez, *R.*¹ Schommer, *J.*¹ Carlson, *A.*¹ Ensrud, *K.*^{1,3}

¹University of Minnesota, Minneapolis, MN, ²California Pacific Medical Center Research Institute, San Francisco, CA, ³Minneapolis VA Health Care System, Minneapolis, MN, ⁴HealthPartners Institute, Bloomington, MN, ⁵Brigham and Women's Hospital, Boston, MA, ⁶University of Pittsburgh, Pittsburgh, PA.

Introduction: There is a paucity of longitudinal studies with sleep efficiency (SE) as an outcome measure. Our objective was to examine potential risk factors for incident reduced SE among community-dwelling women in late life.

Methods: We studied 700 women (mean age 82.5 [SD=3.0] years) with a SE \geq 70% at the Year 16 (2002-04) visit of the Study of Osteoporotic Fractures with a follow-up measure of SE at the Year 20 (2006-08) visit. SE (percentage of time sleeping while in bed) at both visits was measured using a wrist actigraph with data collected for an average of four 24-hour periods. Women were classified as having incident reduced SE if they had SE <70% at Year 20. Logistic regression was used to estimate the associations between potential risk factors (demographics, lifestyle, use of medications, self-reported medical conditions, functional impairment, frailty, mental and physical health) at Year 16 and reduced SE at Year 20. The association of each candidate risk factor with reduced SE at Year 20 was examined in models adjusted for age, clinical site and continuous SE at Year 16. Candidate risk factors with Benjamin Hochberg false-discovery rate q-values <0.10 were included in a final multivariate model.

Results: Among the 700 eligible women, 62 (8.9%) developed incident reduced SE between the Year 16 and Year 20 visits. After adjusting for age, site and baseline SE, antidepressant use [OR=3.06; 95% CI: 1.50-6.25], benzodiazepine use [OR=2.97; 95% CI: 1.30-6.80] and the presence of hypertension [OR=2.83; 95% CI: 1.47-5.45] at Year 16 were independently associated with a higher odds of having reduced SE at follow-up.

Conclusion: These findings suggest that antidepressant use, benzodiazepine use and hypertension are risk factors or markers for the development of reduced sleep efficiency in older women. Future studies are warranted to examine the underlying mechanisms for these associations.

Support: The Study of Osteoporotic Fractures (SOF) is supported by National Institutes of Health funding. The National Institute on Aging (NIA) provides support under the following grant numbers: R01 AG005407, R01 AR35582, R01 AR35583, R01 AR35584, R01 AG005394, R01 AG027574, and R01 AG027576.

0389

PREVALENT INSOMNIA SYMPTOMS AND NEED FOR AN INTERVENTION AMONG ONCOLOGY NURSES

Lee, S.¹ Vigoureux, T. F.¹ Gonzalez, B. D.² Small, B. J.¹ ¹University of South Florida, Tampa, FL, ²Moffitt Cancer Center, Tampa, FL.

Introduction: Insomnia is prevalent in the working population. Nurses may be particularly vulnerable to insomnia due to demands with shift work, limited recovery between shifts, lack of control over their work, and stress associated with proximity to life-threatening health conditions. Insomnia in nurses is a significant public health burden, because it can lead to degraded quality of patient care. This study examined the prevalence of insomnia symptoms among oncology nurses and the need for an intervention to improve insomnia symptoms. **Methods:** Participants were 62 nurses working full-time at a cancer hospital (M_{age} =35.26±11.69). Participants were asked about their (1) main sleep-related complaint, (2) willingness to participate in a sleep-focused intervention, (3) preferred delivery forms of the intervention (i.e., group-based, online, and/or one-on-one), and (4) preference for content to include in the intervention (e.g., sleep hygiene education, mindfulness, cognitive-behavioral therapy). We used content analysis to analyze open-ended responses as well as descriptive statistics to summarize data.

Results: Most (74%) reported difficulty falling or staying asleep or not feeling rested upon awakening as their primary or secondary sleep concerns. Nearly all nurses (95%) expressed interest in participating in a future sleep-focused intervention. In terms of preferred delivery forms of the intervention, an online intervention was most preferred (56%), followed by group meetings at the workplace (50%), and one-on-one meetings at the clinic (29%). Mindfulness strategies were preferred by most nurses (73%), followed by cognitive-behavioral therapy (48%), and sleep hygiene education (34%).

Conclusion: Most oncology nurses report insomnia symptoms and the majority are interested in participating in an intervention to improve their insomnia symptoms either online or in group sessions at the workplace. The information obtained from this pilot study will serve as the basis for developing a future intervention to improve insomnia and overall sleep health in oncology nurses.

Support: This work was supported, in part, by the University of South Florida College of Behavioral & Community Sciences Internal Grant Program (PI: Lee, Grant No. 0134930).

0390

ASSOCIATIONS OF THE NEIGHBORHOOD BUILT ENVIRONMENT WITH ADOLESCENT SLEEP OUTCOMES

Mayne, S.¹ Morales, K.² Williamson, A. A.¹ Grant, S. F.¹ Fiks, A. G.¹ Dinges, D. F.² Zemel, B.¹ Mitchell, J. A.¹ ¹Children's Hospital of Philadelphia, Philadelphia, PA, ²University of Pennsylvania, Philadelphia, PA.

Introduction: Over 75% of U.S. high school students obtain insufficient amounts of sleep. Identification of modifiable environmental determinants of adolescent sleep is needed to inform interventions and public health strategies, yet little is known about the influence of the built environment on adolescent sleep. We examined associations of the built environment with objectively-measured adolescent sleep outcomes.

Methods: In this longitudinal, prospective study, we used actigraphy to assess sleep outcomes for 14 days each in 8th grade and 9th grade: duration (hours/night), onset (hours from 00:00), offset (hours from 00:00), and sleeping >8 hours. Home addresses were linked to built environment exposures based on half-mile Euclidian buffers (overall/human-made sound levels, percent tree canopy cover, street density, intersection density) and census block group (population density, housing density). Mixed-effects linear (sleep duration, onset, offset) and logistic (>8 hours) regression estimated associations of each built environment measure with sleep outcomes, adjusting for sex, race, parent education, household income, grade and weeknight status (school or non-school night).

Results: Among 108 adolescents - 53% female and 25% Black - providing 2,388 nights of sleep data across 8th and 9th grades, a 1-standard deviation increase in neighborhood sound (overall and human-made) associated with 11 minutes later sleep onset (β =0.19; 95% CI: 0.01, 0.38) and 20% lower odds of sleeping for >8 hours (OR=0.80, 95% CI: 0.62,

1.02). A 1-standard deviation increase in neighborhood tree canopy cover associated with 11 minutes earlier sleep onset (β = -0.19, 95% CI: -0.35, -0.03) and 7 minutes earlier sleep offset ($\beta = -0.12$, 95% CI: -0.23, -0.02). No associations were observed for "density based" exposures.

Conclusion: Higher tree canopy cover associated with more favorable sleep timing while higher neighborhood sound level associated with later timing of sleep onset. These modifiable neighborhood built environment factors should be considered when intervening to support healthier sleep among adolescents.

Support: NIH/NHLBI K01HL123612 (JM) and Sleep Research Society Foundation and K23HD094905 (AAW)

0391

GATEWAYS, DISPARITIES, AND FINALS WEEK, OH **MY! TRANSLATING SLEEP SCIENCE FROM THE** LABORATORY TO THE CLASSROOM

Scullin, M. K.¹ Gao, C.¹ Bermudez, V.¹ Diaz, J.¹ Zinke, P.¹ George, C.¹

¹Baylor University, Waco, TX, ²Baylor University, Waco, TX.

Introduction: Organic chemistry can be an insurmountable "gateway" course for otherwise-qualified students in pre-health pathways. Recent data indicate that organic chemistry increases drop-out risk for females and underrepresented minority students (URMs), raising the provocative possibility that sleep disparities are an underrecognized contributor to achievement gaps in gateway STEM courses.

Methods: In Study 1, 481 students enrolled in organic chemistry courses completed sleep questionnaires at the beginning, midpoint, and end of the semester. In Study 2, non-chemistry majors were randomly assigned to normal sleep (8 hours) or sleep restriction (5.5 hours) before taking an organic chemistry virtual lecture and test. In Study 3, 35 students wore actigraphy for five nights and could earn extra credit on a mid-semester test by averaging \geq 8 hours of sleep; actigraphy sleep durations were compared to 40 active-control students who only received sleep education.

Results: In Study 1 (classroom), URM and female students earned lower organic chemistry grades than comparison students, p<.001. Baseline weekday sleep duration predicted test grades across the semester, and students who improved their weekday sleep subsequently improved their organic chemistry grades. In Study 2 (laboratory), mild sleep loss impaired meta-cognitive judgments of organic chemistry learning, a potential causal mechanism for reduced persistence in chemistry courses. In Study 3 (classroom), when better sleep behaviors were incentivized by extra credit, students slept an hour longer/ night than control groups (7.8 vs 6.8 hours, p < .001). These benefits persisted 1 month later into finals week when sleep behaviors were not externally incentivized (7.3 vs 6.3 hours, p=.001). Improving sleep improved performance on difficult short answer questions after correcting for pre-final grades ($M_{adjusted}$ =78% vs 72%, p=.04).

Conclusion: Sleep disparities contribute to achievement gaps in gateway STEM courses, but incentives can reverse poor sleep habits. University administrators should develop and implement behavioral change programs to reduce sleep disparities. Support: National Science Foundation (DRL 1920730)

0392

THE EFFECT OF BENZODIAZEPINE USE ON NON-REM SLEEP INSTABILITY IN COMMUNITY-DWELLING **OLDER MEN**

Hartmann, S. Baumert, M. University of Adelaide, Adelaide, AUSTRALIA.

SLEEP, Volume 43, Abstract Supplement, 2020

Introduction: Previous studies on the implications of benzodiazepine (BZD), a widely prescribed pharmacotherapeutic treatment method for sleep insomnia, on sleep architecture demonstrated significantly reduced EEG activity in low-frequency bands. In this study, we explore the effect of BZD on NREM sleep instability also known as cyclic alternating pattern (CAP) in community-dwelling older men.

Methods: CAP was scored in overnight EEG recordings from 30 older men on long-acting BZD (LBZD), 35 older men on shortacting BZD (SBZD), and 50 age-matched men who did not use BZD (NBZD), participating in the Osteoporotic Fractures in Men Sleep Study (MrOS sleep). A high performance automated detection system determined the ratio between CAP time and NREM sleep time (CAP rate), the number of A1-phases per hour of NREM sleep (A1 index), and the number of A2+A3-phases per hour of NREM sleep (A2+A3 index). The relationship between CAP parameters and BZD use was determined using the Kruskal-Wallis test by ranks with Bonferroni correction for post-hoc analysis.

Results: CAP rate was significantly decreased in older men using long-acting BZD (NBZD: 59.6±18.0%, LBZD: 46.9±13.1%, SBZD: $53.0\pm20.1\%$) as compared to non-BZD user (p < 0.01). All BZD users demonstrated significantly lower frequencies of A1-phases (NBZD: 19.9±23.0 no./h, LBZD: 6.9±13.3 no./h, SBZD: 4.5 ± 9.9 no./h) as compared to non-BZD users (LBZD: p < 0.01, SBZD: p < 0.001). The A2+A3 index did not show any variations between the three groups.

Conclusion: Older men using long-acting BZD demonstrate a significantly reduced CAP rate during sleep, particularly less frequent A1-phases, compared to the control group. Moreover, short-acting BZD user show significantly less frequent A1-phases but no difference in CAP rate and A2+A3-phases than older men using no BZD. Hence, BZD usage has a major adverse effect on the occurrence of EEG slow waves.

Support: The National Heart, Lung, and Blood Institute (NHLBI) provides funding for the MrOS Sleep ancillary study "Outcomes of Sleep Disorders in Older Men" under the following grant numbers: R01 HL071194, R01 HL070848, R01 HL070847, R01 HL070842, R01 HL070841, R01 HL070837, R01 HL070838, and R01 HL070839.

0393

THE EFFECT OF TRAZADONE USE ON NON-REM SLEEP INSTABILITY IN COMMUNITY-DWELLING OLDER MEN

Hartmann, S. Baumert, M.

University of Adelaide, Adelaide, AUSTRALIA.

Introduction: With steadily growing numbers of patients with a depressive disorder, the effect of antidepressants on sleep architecture is of increasing concern. One major oral antidepressant medication is trazadone, which has also been prescribed in low doses for sleep insomnia treatment. Here, we investigate the effect of trazadone on NREM sleep instability also known as cyclic alternating pattern (CAP) in community-dwelling older men.

Methods: CAP was scored in overnight EEG recordings from 41 older men on trazadone (TRZ) and 50 age-matched men who did not use trazadone (NTRZ), participating in the Osteoporotic Fractures in Men Sleep Study. A high performance automated detection system determined the ratio between CAP time and NREM sleep time (CAP rate), the number of A1-phases per hour of NREM sleep (A1 index), and the number of A2+A3-phases per hour of NREM sleep (A2+A3 index). The effect of TRZ on CAP parameters was determined using the Mann-Whitney U test.

Results: CAP rate was significantly decreased in men using trazadone (NTRZ: $58.2\pm19.7\%$, TRZ: $47.9\pm15.9\%$) as compared to non-trazadone user (p < 0.01). Subtype indices did not show any significant difference between both groups but to some extent less frequent A2-A3 phases for TRZ user (A1-phases: NTRZ 13.0±18.7 no./h vs. TRZ 10.8±20.4 no./h, p = 0.35; A2+A3-phases: NTRZ 51.5±33.7 no./h vs. TRZ 44.7±23.3 no./h, p = 0.068).

Conclusion: CAP rate was significantly decreased in older men on trazadone as compared to older men who did not use trazadone, suggesting that trazadone usage has a stabilising effect on sleep micro-structure.

Support: The National Heart, Lung, and Blood Institute (NHLBI) provides funding for the MrOS Sleep ancillary study "Outcomes of Sleep Disorders in Older Men" under the following grant numbers: R01 HL071194, R01 HL070848, R01 HL070847, R01 HL070842, R01 HL070841, R01 HL070837, R01 HL070838, and R01 HL070839.

0394

DIFFERENTIAL ROLE OF SLEEP PROBLEMS ON DEPRESSION AND SUICIDE IN COMMUNITY ADOLESCENTS

Kim, J.¹ Vander Stoep, A.² McCauley, E.² ¹Seattle University, Seattle, WA, ²University of Washington, Seattle, WA.

Introduction: Sleep changes during adolescence, including "eveningness" or a preference for staying up late, decreased sleep hours, increased daytime sleepiness and irregular sleep patterns, can contribute to adolescent sleep disturbances, such as insomnia, daytime fatigue, and other sleep problems. The goals of the study were; 1) to examine the proportion of adolescents who experienced each type of sleep disturbances based on six sleep problems; and 2) to examine the association between six types of sleep disturbances and concurrent depression, suicide ideation, and suicide attempt at $12^{\rm th}$ grade.

Methods: Using the data from the Developmental Pathways Project (DPP), a community-based study in adolescence, total 425 students were included. Sequential logistic regression analyses were performed to examine the association between each sleep item and depression, suicide ideation, and lifetime history of suicide attempt. Results: The most frequently reported sleep problems were 'overtired without good reason (49.9%)', 'sleeps less than most kids (46.8%)', and 'sleep more than most kids (46.7%)'. After adjusting for depressive symptoms at baseline, 12th grade reports of 'overtired without good reason' (OR = 1.63, 95% CI = 1.22 - 2.17), 'sleep less' (OR = 2.03, 95% CI = 1.51 - 2.74), 'trouble sleeping' (OR = 1.50 95% CI = 1.10-2.06, p < .05), 'nightmare' (OR = 1.51, 95% CI = 1.12-2.02, p < .01) were significantly associated with depression. For suicide ideation (SI) and suicide attempt, 'nightmare' (OR = 1.68, 95% CI = 1.15 - 2.48; OR = 2.43 95% CI = 1.30 - 4.53, respectively) was significantly and positively associated with SI and having history of suicide attempt.

Conclusion: 'Nightmare' has the strongest association with depression, suicide ideation, and suicide attempt. To disentangle the mechanism of the association between nightmares and mental health issues, longitudinal studies examining causal or bidirectional relationships of the mechanism are warranted.

Support: National Institutes of Mental Health and Drug Abuse, Nesholm Family Foundation, Seattle Children's Hospital Outcomes Research Steering Committee, Loeb Family Foundation, Seattle Children's Research Institute, University of Washington Office of the Provost, AETNA Foundation.

0395

"DOCTOR, I CAN'T SLEEP", HOW INTENSIVIST CANNOT RESTORE THEIR SLEEP DEBT

CHHOR, V.¹ LEGER, D.² PEPIN, E.³ ELBAZ, M.⁴

¹1Université Paris Descartes Department of Anesthesia and Intensive Care of Georges Pompidou European Hospital, Paris, FRANCE, ²Université Paris Descartes-APHP Hôtel-Dieu, PARIS, FRANCE, ³APHP Hôtel Dieu Centre du Sommeil, Paris, FRANCE, ⁴Université de paris Paris Descartes, Paris, FRANCE.

Introduction: Since 2001, intensivist physicians working in France have been organized in 24-hour shifts (in order to provide 24/7 support), followed by a mandatory rest period of at least 24 hours between shifts. The goal was to survey how intensivists cope with maintaining enough sleep throughout their 24-hour shifts.

Methods: During twelve months, the whole medical staff of our ICU, i.e. 29 individuals (11 senior physicians and 18 residents), agreed to participate in this study. They were allowed to settle into a typical work period of 7 days and 7 nights (i.e. 3 days before and 3 days following the 24-hour shift), separated by at least 5 days from any previous 24-hour shifts. A 24-hour shift typically started at 8:30am in the morning and finished 24 hours later. All physicians wore a wrist actigraphy device (MotionWatch 8) assessing total sleep time (TST) on a 24-hour period. We retained several criteria such as:

The reference TST, i.e. the mean of the TST during the first three days preceding the 24-hour shift

The sleep debt, through the comparison between the average of the TST during the 24-hour shift and the 24 (SD24) or 72 following hours (SD72) and the reference TST.

Results: Twenty nine actigraphy records from 29 individuals were analyzed. The reference TST preceding the shift was 377 min (IQR25-75: 346-396). Doctors participating in the study slept very little during a 24-hour shift: 181 min (IQR25-75: 134-260). Subsequently, we observed that intensivists were unable to completely recover their sleep debt, even after 72 hours. Average TST was significantly shorter at 72 hours compared to reference (343 min [IQR25-75: 304-367], Mann-Whitney test, p=0.015).

Conclusion: These findings raise the question of whether it is possible for intensivist doctors to maintain their energy and intensity in their jobs without sleeping enough. This remains an open question and we are currently unable to respond with only a simple study.

Support: No support

0396

PREVALENCE OF DELAYED SLEEP PHASE SYNDROME (DPSP) AMONG OMANI PEOPLE

Al-Abri, M. A.¹ Al-Kindi, T.¹

¹Sultan Qaboos University, Muscat, OMAN, ²Sultan Qaboos University, Muscat, OMAN.

Introduction: Delayed sleep phase syndrome (DSPS) is a circadian rhythm sleep disorder with a definition of delayed night sleep by two or more hours beyond the socially acceptable or conventional bedtime. The general reported prevalence of DSPS is 7% to 16%. However, there is no previous study which assess DSPS prevalence in Oman or nearby regions. This study aimed to assess the

prevalence of DSPS among Omani population and to establish a connection between demographics and DSPS.

Methods: This community-based study included 186 subjects aged from 18 to 64 who had one week of actigraphy records along with their demographical data in Sultan Qaboos University hospital.

Results: Among the 186, 19 (10.2%) subjects were identified of having DSPS in weekdays, and 15 (11.4%) subjects have DSPS in weekends. The results indicated that marital status had significant relation (P=0.02) more with unmarried group (62.50%, N=10). No significant relationship was found between DSPS and age, gender, BMI, education and employment status in either weekdays or weekends (P>0.05)

Conclusion: A consistent rates of DSPS was found among Omanis which are 10.2% in weekdays and 11.4% in weekends. Also, DSPS have significantly higher rates among unmarried subjects in week-days. Further studies in DSPS are needed to assess risk factors and possible health impacts.

Support: The research Council of Oman

0397

NEIGHBORHOOD PHYSICAL AND SOCIAL ENVIRONMENTS AND SLEEP AMONG CHILDREN AND ADOLESCENTS: A SYSTEMATIC REVIEW

Mayne, S. L. Mitchell, J. A. Virudachalam, S. Williamson, A. A. The Children's Hospital of Philadelphia, Philadelphia, PA.

Introduction: Understanding salient environmental determinants of pediatric sleep is essential for informing interventions and public health initiatives. Emerging evidence suggests the neighborhood environment can impact pediatric sleep. We are conducting a systematic review of studies examining associations of neighborhood physical and social environments with sleep among children and adolescents. Methods: We searched 6 databases (MEDLINE, PubMed, EMBASE, SCOPUS, Web of Science, PsychInfo) using search terms related to sleep, neighborhood environment, and pediatric populations to identify English-language articles with an abstract. We identified and screened 2,581 abstracts. Inclusion criteria included 1) assessing associations of ≥ 1 neighborhood-level factor with ≥ 1 sleep outcome and 2) including participants ≤ 18 years. We excluded review articles, protocols, qualitative and non-human studies. In total, 134 full-text articles were independently reviewed by 2 reviewers each to confirm eligibility. One reviewer abstracted preliminary data from included studies. Next steps include independent data abstraction by two reviewers using a standardized form, synthesis of results, and assessment of study quality according to the study design, sleep assessment method, sampling strategy, and control for confounding.

Results: Sixty-one articles met inclusion criteria. Fourteen articles included children aged 0-5 years, 38 included children aged 6-12 years, and 36 included adolescents aged 13-18 years (25 included multiple age groups). Twenty-two studies (36%) used objective sleep assessment methods (e.g. actigraphy). Seven studies (11%) examined sleep apnea/snoring. The most common neighborhood-level factors were safety/crime/community violence (n=28) and socioeconomic status (n=25), with fewer studies examining other exposures like noise (n=7) and social cohesion (n=4). Results on key associations and study quality are forthcoming.

Conclusion: A growing body of epidemiological data has emerged in recent years to provide insight into how the neighborhood environment can impact pediatric sleep. Preliminary results suggest few studies have examined associations of the built environment with sleep, with most studies focusing on school-aged children and adolescents. Support: This work was supported by: the Children's Hospital of Philadelphia's Possibilities Project (SLM); Sleep Research Society Foundation and K23HD094905 (AAW); NIH/NHLBI K01HL123612 (JAM)

0398

DEMOGRAPHIC DIFFERENCES IN THE DEGREE OF DISCREPANCY BETWEEN SLEEP DIARY AND ACTIGRAPHY MEASURES OF SLEEP

Scott, B.¹ Crawford, M.¹ Slavish, D.¹ Messman, B.¹ Wardle-Pinkston, S.² Dietch, J.³ Kelly, K.¹ Ruggero, C.¹ Taylor, D.² ¹Department of Psychology, University of North Texas, Denton, TX, ²Department of Psychology, University of Arizona, Tucson, AZ, ³War Related Illness and Injury Study Center, Palo Alto Veterans Affairs Health Care System, Palo Alto, CA.

Introduction: The accurate estimation of sleep is critical for understanding who is most at risk for sleep disorders and associated disease outcomes. Individuals who overestimate sleep disturbances may be at increased risk for insomnia. A few studies have shown demographic differences in the accuracy of sleep estimation when comparing subjective and objective measures; however, the previous literature is inconsistent and focuses primarily on older adults. We sought to replicate these studies in a large sample of nurses using 14 days of sleep diary and actigraphy measures.

Methods: Participants were 392 nurses (91.8% female; 77.8% white, mean age = 39.54) recruited for a larger study. Participants completed 14 days of actigraphy and sleep diaries to prospectively assess total sleep time (TST) and sleep efficiency (SE). Discrepancy between diary and actigraphy measures was calculated by subtracting actigraphy measures from diary measures. Linear regression was used to examine how age, race (0 = race other than white, 1 = white), gender (1 = male, 2 = female), ethnicity (1= non-Hispanic/Latinx, 2 = Hispanic/Latinx) predicted degree of sleep discrepancy.

Results: The average discrepancy between diary and actigraphy TST was 30.29 minutes (SD = 29.28), and the average discrepancy between diary and actigraphy SE was 4.16% (SD = 5.66). Race and ethnicity did not predict amount of TST or SE discrepancy. However, younger individuals had more discrepancy in both TST (b = -0.48, p < .001) and SE (b = -0.09, p < .001). Men also had a greater discrepancy in both TST (b = -10.90, p < .05) and SE (b = -2.56, p < .05).

Conclusion: Men and younger individuals had greater discrepancies between diary and actigraphy measures of sleep. This is in contrast to some previous research showing that elderly women tend to display greater discrepancies between subjective and objective measures of sleep. It is essential that future research explore the discrepancies between subjective and objective measures of sleep in larger and more demographically diverse samples. Establishing a better understanding of this relationship is crucial, as it may have significant implications for the diagnosis and treatment of insomnia.

Support: NIH/NIAID R01AI128359-01

0399

THE MSSA: A NOVEL INSTRUMENT TO ASSESS SLEEP AND SLEEP DISTURBANCES IN MILITARY MEN AND WOMEN

Gerwell, K.¹ Pruiksma, K. E.¹ Brock, M. S.² Peterson, A. L.¹ Carrizales, F. A.¹ Brundige, A.¹ Taylor, D. J.⁴ Vanecek, R.² Hansen, S.² Foster, S. N.² Young-McCaughan, S.¹ Straud, C. L.¹ Mysliwiec, V.¹

¹The University of Texas Health Science Center at San Antonio, San Antonio, TX, ²Wilford Hall Ambulatory Surgical Center, San Antonio, TX, ³The University of Texas Health Science Center at San Antonio, San Antonio, TX, ⁴University of Arizona, Tucson, AZ.

Introduction: Military personnel experience unique stressors (e.g., deployments, shift work, family separation) which can cause sleep disturbances. However, little is known regarding the general sleep quality of military personnel and how it changes throughout their service, what types of stressors precipitate sleep disturbances, and how this differs among military men and women. We present findings from a new self-report measure, the Military Service Sleep Assessment (MSSA), which was designed to assess (1) current primary problems with sleep, (2) sleep quality throughout military service (3) life events that may have impacted their sleep and (4) the specific events which most effected sleep.

Methods: A total of 69 military personnel (22 women and 47 men) completed the MSSA and 49 also completed a diagnostic polysomnogram (PSG). Chi-square tests were run to differences in responses between men and women.

Results: No significant differences were found between men and women. In general, sleep quality progressively decreased over a participant's military career from 3 to 2 on a 5-point Likert scale (1=low, 5=high). For those with at least one deployment (n=52), 73% reported that deployment and 54% reported that a redeployment (return from deployment) negatively impacted sleep quality for 3 months. Women reported that permanent change of station (PCS) negatively impacted their sleep more frequently than men (36% vs. 28%). The reported events that most significantly impacted sleep quality were deployment, military service other than deployment and trauma.

Conclusion: The MSSA is a novel instrument that can be used to increase understanding of sleep disturbances in military men and women which can inform prevention and treatment strategies. This measure is being used to systematically evaluate the factors which may precipitate or perpetuate sleep disturbances in military men and women such as military service-associated factors, training, deployment history, changing stations, and exposure to trauma or other stressful life events.

Support: This study is supported by the Defense Health Agency, Defense Medical Research and Development Program, Clinical Research Intramural Initiative for Military Women's Health.

0400

HABITUAL SLEEP, CIRCADIAN MISALIGNMENT, AND CARDIOVASCULAR RISK FACTORS AMONG LATE ADOLESCENTS

Ji, *X.*¹ *Wang*, *Y.*¹ *Saylor*, *J.*¹ *Patterson*, *F.*² *Ruggiero*, *L.*² ¹University of Delaware School of Nursing, Newark, DE, ²University of Delaware Department of Behavioral Health and Nutrition, Newark, DE.

Introduction: Emerging evidence suggests the potential role of sleep in cardiovascular disease (CVD) risk. Sleep variability and circadian misalignment may represent understudied sleep dimensions, particularly among late adolescents. This study investigated the associations of habitual sleep, circadian misalignment, night-to-night sleep variability with CVD risk factors among late adolescents.

Methods: Using a cross-sectional design, we enrolled 58 healthy, college students (19.22±1.06 years old). Participants completed a

7-day sleep diary, the Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale. Circadian misalignment was assessed using the weekend-weekday differences in sleep duration and midsleep time. Sleep variability was calculated as intra-individual standard deviation of sleep durations. The number of CVD risk factors (BMI, waist circumference, blood pressure, fasting glucose and lipid profile) above subclinical/clinical thresholds was used as a proxy of overall risk. Logistic and generalized linear regression tested the relationships.

Results: Forty-two participants (73%) had at least one elevated CVD risk factors and 19 (34%) were short sleepers (<7 h). On average, the midsleep shifted 54 minutes later on weekends and the intraindividual sleep variability was 1.31 hours. After controlling for age, gender and race, there was a trend towards higher overall CVD risk (β =0.45±0.22, p=0.05) with a greater weekend-weekday discrepancy in sleep duration. For each CVD risk factor, a greater discrepancy in weekend-weekday midsleep times (OR=2.29±0.82, p=0.02) was estimated to increase the odds of high blood pressure. Participants with greater discrepancy in weekedy-weekend sleep durations (OR=1.58±0.41, p=0.03) or excessive daytime sleepiness (OR=4.68±3.38, p=0.03) were more likely to have high BMI. Worse sleep quality (higher PSQI scores) was associated with high BMI (OR=1.36±0.19, p=0.03) and waist circumference (OR=1.40±0.24, p=0.04).

Conclusion: This study suggests that circadian misalignment, compared with other sleep characteristics, better predicts cardio-vascular risk among late adolescents. Future research is needed to examine the interaction among circadian misalignment, sleep variability and sleep duration on CVD risk.

Support: American Nurse Foundation 18A01422

0401

SLEEP CHARACTERISTIC DIFFERENCES DURING EARLY POSTPARTUM IN WHITE AND AFRICAN AMERICAN WOMEN

Kishman, E. E.¹ Sparks, J. R.¹ Liu, J.¹ Castleberry, L. A.¹ Cook, J. W.¹ Youngstedt, S. D.² Wang, X.¹ ¹University of South Carolina, Columbia, SC, ²Arizona State University, Phoenix, AZ.

Introduction: In the general population, poor sleep quality and shorter sleep duration is associated with several adverse health outcomes. African American adults are more likely to report poorer sleep quality and shorter total sleep duration compared to White adults. However, there is limited information comparing sleep characteristics in White and African American women during postpartum, when many women experience reduced sleep quality. The purpose of this study was to compare sleep quality at 6-8 weeks postpartum in White and African American women.

Methods: White (n=84) and African American (n=37) women, who gave birth to a singleton at \geq 37 weeks of gestation, completed the Pittsburg Sleep Quality Index (PSQI) and the Epworth Sleepiness Scale (ESS) at 6-8 weeks postpartum. The PSQI was used to assess global sleep quality, time in bed, and 7 components regarding sleep. The components included: Subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. The ESS total score was used to assess daytime sleepiness.

Results: The global PSQI score was higher for African American women (8.8 \pm 3.3, mean \pm SD), indicating poorer sleep quality than White women (7.1 \pm 3.0, p=.006). African American women had higher sleep latency and shorter sleep duration compared to

White women (p=.0179 and p<.0001, respectively). Time in bed was not statistically different for African American women compared to White women (485.6 \pm 143.7 and 530.8 \pm 85 minutes/ night, p=.08). No other components of the PSQI were significantly different between the two racial groups. African American women scored higher on the ESS than White women (8.9 \pm 3.1, 6.6 \pm 3.2, p=.0002) indicating greater daytime sleepiness.

Conclusion: These results suggest that African American women experience lower sleep quality and greater daytime sleepiness in early postpartum compared to White women.

Support: National Institute On Minority Health And Health Disparities of the National Institutes of Health under Award Number R21MD012740.

0402

IMPACT OF VARIOUS ACTIGRAPHIC EDITING APPROACHES ON SLEEP/WAKE OUTPUTS IN ADULTS WITH INSOMNIA AND HEALTHY SLEEPERS

Kline, C. E.¹ Egeler, M. E.¹ Kubala, A. G.¹ Patel, S. R.¹

Lehrer, H. M.¹ Duggan, K. A.² Hall, M. H.¹

¹University of Pittsburgh, Pittsburgh, PA, ²North Dakota State University, Fargo, ND.

Introduction: Actigraphy data can be edited using a variety of approaches. However, whether time-intensive manual editing provides different sleep/wake estimates compared to other approaches is unknown. The purpose of this study was to compare sleep/wake data obtained from a standardized editing approach that incorporates multiple inputs versus three other common approaches.

Methods: 72 adults (33.8±11.1 y, 74% female, 71% white) provided 1022 nights of data for analysis; 45 were healthy sleepers (678 nights) and 27 met DSM-5 criteria for insomnia. Participants wore an Actiwatch Spectrum on their nondominant wrist and completed a sleep diary for 3-24 nights. Each night's rest interval was set using four different approaches: (1) a standardized process based upon published guidelines (Patel et al., Sleep 2015) that incorporates a hierarchical order of multiple inputs (event marker, light, diary, activity; STANDARD); (2) software-provided automated algorithm (AUTO); (3) automated algorithm with incorporation of event markers (AUTOE); and (4) sleep diary (DIARY). We used linear mixed-effects models to evaluate whether sleep/wake parameters differed between the STANDARD and other editing approaches, accounting for patient status (healthy sleeper, insomnia) and the possibility that differences among editing approaches may be dependent on patient status.

Results: All results are expressed relative to the STANDARD approach. Bedtime was 36.1 ± 5.1 min earlier (P<.0001) and morning out-of-bed time was 13.6 ± 5.7 min later (P=.02) using the AUTO (P<.0001) approach. Time in bed was 42.3 ± 4.7 min longer with AUTO (P<.0001). Sleep onset latency was 11.7 ± 1.4 min and 2.8 ± 1.4 min longer for AUTO (P<.0001) and DIARY (P=.05), respectively. Sleep duration was 22.5 ± 4.4 min longer with AUTO (P<.0001). Wake after sleep onset was 6.8 ± 1.2 min greater with AUTO (P<.0001). Similar patterns were observed for all sleep/ wake measures among healthy sleepers and adults with insomnia.

Conclusion: A standardized approach to editing actigraphy data leads to different sleep/wake estimates compared to other common approaches, though the differences were often small in magnitude and not dependent upon sleep status. Most notably, reliance upon the automated algorithm yielded longer time in bed, sleep duration, sleep onset latency, and wake after sleep onset compared to the standardized approach. **Support:** NIH K23HL118318

0403

SLEEP VALUATION: A NOVEL CONSTRUCT AND QUESTIONNAIRE

Nielson, S. Simmons, Z. Kay, D. Brigham Young University, Provo, UT.

Introduction: Sleep valuation, the relative worth of one's own sleep, is an under-explored construct that could have implications on sleep health. This study sought to validate the Sleep Valuation Questionnaire (SVQ) and to explore demographic differences in sleep valuation.

Methods: Participants (N = 946) recruited through TurkPrime were stratified across age (18-99), race (50% White, 17% Black, and 8.33% for each American Indian, Asian, Pacific Islander, and other), Hispanic ethnicity (20%), gender (50/50 female/male), employment status (50% employed full time and 8.33% for each part time, homemaker, retired, student, temp worker, unemployed, and disabled), Participants completed a demographic survey, followed by the original 43 item SVQ completed twice. Iterated principal factoring with a Promax solution was used to identify the factor structure of the SVQ. Cronbach's alpha and correlation analyses were also used to help identify items with poor reliability. Total sleep valuation, the sum of valid items on the SVO, was used as the dependent variable in a multiple regression analysis. Age, gender, race, work status, socioeconomic status, educational level, marital status, and general health and mental health estimates served as independent variables.

Results: After removing items with weak factor loadings (<0.6), poor reliability, and weak face validity, the number of items were reduced to 10, which loaded on to 2 factors: Sleep Desire and Sleep Need. Those who cohabitate had lower SVQ scores than married individuals (p=0.04), full-time workers had higher SVQ scores than non-full-time workers (p=0.001), higher age was associated with lower SVQ scores (p<0.001), and higher general mental health was associated with lower SVQ scores (p<0.001).

Conclusion: This is the first study to explore how demographic variables relate to sleep valuation. The SVQ may help identify factors that contribute to sleep valuation and sleep valuation relates to sleep behavior, sleep health, and sleep treatment utilization. **Support:** None

0404

JEWISH-ARAB DISPARITIES IN SLEEP BEHAVIORS AND DIFFERENTIAL ETHNIC IMPACT ON DAYTIME FUNCTIONING, DRIVING SAFETY, AND HEALTH IN ISRAEL

Rosenberg, E.¹ Perlis, M. L.² Parthasarathy, S.³ Jean-Louis, G.⁴ Chakravorty, S.² Grandner, M. A.³

¹Ministry of Health, Jerusalem, ISRAEL, ²University of Pennsylvania, Philadelphia, PA, ³University of Arizona, Tucson, AZ, ⁴New York University, New York, NY.

Introduction: In Israel, those with Arabic as compared to Jewish ethnicity, exhibit poorer health and motor vehicle safety behaviors. Their ethnic differences in sleep duration and quality may modulate their vulnerabilities to these behaviors.

Methods: 7,230 Israeli individuals (N=5,880 Jewish and N=1350 Arabic) responded to the 2017 Israeli Bureau of Statistics population-based survey of households. Variables were self-reported. Outcomes included sleepiness, sleep medications, functional impairment, drowsy driving, overall health, 1-year health change, and obesity. Predictors included categorical sleep duration

(<=5, 6, 7, 8 [reference], or >=9 hours) and sleep disturbance in the past month (none [reference], mild [1/week], moderate [2-3/week], or severe [>3/week]). Covariates included age, sex, and financial status. Ethnicity (Jewish/Arabic) was treated as a predictor of sleep and behavioral outcomes.

Results: When compared to normal (8-hour) sleepers, Jewish as compared to Arabic individuals were more likely to to sleep <=5h (RRR=3.99, p<0.0005), 6h (RRR=4.65, p<0.0005), and 7h (RRR=3.34, p<0.0005), and were more likely to report severe sleep difficulties (RRR=1.49, p<0.0005) and sleepiness (oOR=1.52, p<0.0005). Yet, they were less likely to report functional impairment (oOR=0.65, p<0.0005), drowsy driving (OR=0.58, p<0.0005), worse health (oOR=0.51, p<0005), worsening health (oOR=0.70, p<0.0005), or obesity (OR=0.64, p<0.0005). Significant ethnicity by sleep duration interactions (p<0.05) characterized sleepiness, sleep medications, functional impairment, health, and health change. Moreover, significant ethnicity by sleep disturbance interactions (p<0.05) characterized the same outcomes, in addition to drowsy driving. Overall, the impact of sleep duration and sleep difficulties was generally greater among Arabs for all variables.

Conclusion: Despite Jewish individuals endorsing relatively shorter sleep and more severe sleep difficulties, Arabs seem to be more vulnerable to the health and functional outcomes. This finding may explain some of the discrepancies in the health and safety outcomes between these ethnic groups.

Support: Dr. Grandner is supported by R01MD011600

0405

SLEEP DURATION AND SLEEP DISTURBANCE RELATED TO OBESITY, HEALTH, MOTOR VEHICLE SAFETY, AND DAYTIME FUNCTIONING IN ISRAEL: DATA FROM THE 2017 ISRAEL SOCIAL SURVEY

Rosenberg, E.¹ Perlis, M. L.² Parthasarathy, S.³ Chakravorty, S.² Grandner, M. A.³

¹Ministry of Health, Jerusalem, ISRAEL, ²University of Pennsylvania, Philadelphia, PA, ³University of Arizona, Tucson, AZ.

Introduction: Previous studies suggest the Israeli population exhibits relatively short sleep duration and experiences sleep difficulties. This analysis evaluates the relationships between habitual sleep and outcomes of interest in this population.

Methods: Data were obtained from 7,230 Israeli individuals. The sample consisted a 2017 population-based survey of households, conducted by the Israeli Bureau of Statistics. All variables were self-reported. Outcomes of interest included drowsy driving, sleep medication use, functional impairment, sleepiness, overall health, 1-year health change, and obesity. Predictors included categories of sleep duration (<=5, 6, 7, 8 [reference], or >=9 hours) and sleep disturbance in the past month (none [reference], mild [1/week], moderate [2-3/week], or severe [>3/week]). Covariates included age, sex, ethnic group, and financial status. Binary and ordinal logistic regressions were employed to evaluate the relationship between them and post-hoc analyses evaluated the relationships between subgroups.

Results: Drowsy driving was associated with <=5h, 6h, and 7h sleep duration categories, and severe sleep disturbance. The use of sleep medication use was associated with <=5h and >=9h, and all levels of sleep disturbance. Functional impairment and sleepiness were both associated with <=5h, 6h, 7h, and >=9h, and all levels of sleep disturbance. Their reported overall health was linked to sleep duration of <=5h and >=9h, and all levels of sleep disturbance.

Worsening health was associated with \leq =5h and all levels of sleep disturbance. Obesity was associated with \leq =5h and severe sleep disturbance. In post-hoc analyses restricted to individuals with no sleep disturbance, habitual sleep duration was still statistically significantly related to drowsy driving, sleep medications, sleepiness, and health change.

Conclusion: Short sleep duration and sleep disturbance are associated with worse motor vehicle safety, health, and functioning in the Israeli population. Effects of sleep duration were generally maintained even for those without sleep disturbance. These results may help focus public health efforts on improving sleep health. **Support:** Dr. Grandner is supported by R01MD011600

0406

DAYTIME SLEEPINESS IN THE COMMUNITY: IMPLICATIONS FOR SLEEP, CIRCADIAN, AND PHYSICAL HEALTH

*Grandner, M. A.*¹ *Tubbs, A.*¹ *Jean-Louis, G.*² *Seixas, A.*² *Hale, L.*³ *Branas, C.*⁴ *Killgore, W. D.*¹ *Wills, C. C.*¹

¹University of Arizona, Tucson, AZ, ²New York University, New York, NY, ³Stony Brook University, Stony Brook, NY, ⁴Columbia University, New York, NY.

Introduction: Daytime sleepiness impacts performance and well-being. The present study used validated measures to explore associations of community-level daytime sleepiness with sleep health, preferred sleep phase, physical inactivity, and overall health. Methods: Data were from the Sleep and Healthy Activity, Diet, Environment, and Socialization (SHADES) study of N=1007 adults age 22-60 from the community. Daytime sleepiness was assessed with the Epworth Sleepiness Scale (ESS). Outcomes of interest included the Insomnia Severity Index (ISI), Pittsburgh Sleep Quality Index (PSQI), endorsement of a preference for an advanced or delayed sleep phase on the Sleep Disorders Symptom Check List (SDSCL), STOP-BANG sleep apnea questionnaire score, sedentary time assessed with the International Physical Activity Questionnaire (IPAQ), and the global health item on the SF-12, operationalized as excellent/good or fair/poor health. Through regression analyses, we assessed whether daytime sleepiness was independently associated with several sleep, circadian and physical health outcomes, adjusting for habitual sleep duration and sociodemographic factors like age, sex, education, and race/ ethnicity.

Results: Our adjusted models indicate that daytime sleepiness was associated with insomnia (B=0.57; 95%CI: 0.50, 0.65; p<0.0001), sleep quality (B=0.34; 95%CI: 0.29, 0.39; p<0.0001), advanced sleep phase (OR=1.06; 95%CI: 1.03, 1.09; p<0.0001), delayed sleep phase (OR=1.05; 95%CI: 1.02, 1.07; p=0.0003), STOP-BANG score (B=0.08; 95%CI: 0.07, 0.10; p<0.0001), sedentary minutes (B=6.12; 95%CI: 2.77, 9.47; p=0.0004), and overall poor health (OR=1.10; 95%CI: 1.07, 1.13; p<0.0001). After additional adjustment for habitual sleep duration, all relationships were maintained.

Conclusion: Daytime sleepiness is associated with more severe insomnia, preference for advanced or delayed sleep timing, worse sleep quality, and greater risk of sleep apnea. Moreover, daytime sleepiness was associated with greater sedentary time and worse overall health. Since these relationships are independent of sleep duration, they likely do not reflect an effect of sleep deprivation.

Support: This work was supported by a grant from Jazz Pharmaceuticals. The SHADES study was funded by R21ES022931. Dr. Grandner is supported by R01MD011600.

SLEEP, STRESS, CARDIOVASCULAR, AND PSYCHOLOGICAL HEALTH IN COLLEGE STUDENTS Paraeldo A. M.¹ Mulling, K. M.²

Reynolds, A. M.¹ Mullins, K. M.²

¹University of Virginia's College at Wise, Wise, VA, ²University of Virginia's College at Wise, Wise, VA.

Introduction: Epidemiological studies have long established that sleep factors, stress, and cardiovascular health are related. College students often struggle with the demands of college life, which leads to increased stress, symptoms of depression and anxiety, and poor sleep. The focus of the current study was to examine habitual sleep habits in college students, in association with psychological factors and physiological factors.

Methods: Participants included 51 undergraduate students (18 men, average age M=20.25 years, SD=1.78) who wore wrist actigraphs to measure their typical sleep habits. After one week, participants completed questionnaires about psychological symptoms (i.e., depression, anxiety, and stress; Depression Anxiety Stress Scale, DASS-21) and subjective physiological symptoms (i.e., fatigue; Multidimensional Assessment of Fatigue Scale, MAF). Blood pressure and heart rate were measured using a wrist cuff.

Results: Overall total sleep time was 6.59 hours and sleep efficiency was 82.55%. Pearson correlational analyses revealed a negative moderate association between sleep efficiency and diastolic blood pressure (r(49) = -.318, p = .024). Global PSQI scores were moderately associated with stress (r(49) = .419, p = .002). MAF Global Fatigue Index scores revealed positive associations with depression (r(49) = .344, p = .014), anxiety (r(49) = .474, p < .001), and stress (r(49) = .620 p < .001). Heart rate was positively associated with depressive symptoms r(49) = .296, p = .035), stress symptoms r(49) = .447, p = .001), and fatigue r(49) = .456, p = .001).

Conclusion: As expected, college students' sleep was short in duration and poor in efficiency. Sleep factors, cardiovascular factors, psychological factors, and stress were all related, demonstrating the importance of sleep on physiological and psychological health. More research should be conducted to further examine the relationships and directionality between sleep, psychological factors, and stress as there may be underlying mechanisms important for cardiovascular health.

Support: None.

0408

MYTHS ABOUT INFANT, CHILD, AND ADOLESCENT SLEEP: ADDRESSING FALSE BELIEFS THAT HINDER SLEEP HEALTH DURING THESE CRUCIAL DEVELOPMENTAL STAGES

Robbins, R.¹ Hale, L.² Beebe, D.³ Wolfson, A. R.⁴ Grandner, M. A.⁵ Mindell, J. A.⁶ Owens, J.⁷ Tapia, I.⁶ Byars, K. C.⁸ Gruber, R.⁹ Montgomery-Downs, H.¹⁰ Wise, M.¹¹ Carskadon, M. A.¹² ¹Brigham and Women's Hospital, Boston, MA, ²Population and Preventive Medicine, Stony Brook, NY, ³Cincinnati Children's Hospital Medical Center, Cincinnati, OH, ⁴Loyola University Maryland, Baltimore, MD, ⁵University of Arizona College of Medicine, Tucson, AZ, ⁶Children's Hospital of Philadelphia, Philadelphia, PA, ⁷Center for Pediatric Sleep Disorders, Boston, MA, ⁸Cincinnati Children's Hospital Medical Center, Cincinnati, OH, ⁹Douglas Mental Health University Institute, Montreal, QC, CANADA, ¹⁰West Virginia University, Morgantown, WV, ¹¹Methodist Le Bonheur Healthcare, Memphis, TN, ¹²Warren Alpert Medical School of Brown University, Providence, RI. **Introduction:** Sleep is vital for healthy development from infancy through adolescence. Despite its importance, false beliefs that conflict with scientific evidence (myths) may be common among caregivers and impair sleep health during these crucial stages.

Methods: Researchers compiled a list of potential myth statements using internet searches of popular press and scientific literature. We utilized a Delphi process with experts (n=12) from the fields of pediatric, sleep, and circadian research and clinical practice. Selection and refinement of myths by sleep experts proceeded in three phases, including: focus groups (Phase 1); email-based feedback to edit, add, or remove myths (Phase 2); and closed-ended questionnaires (Phase 3) where experts rated myths on two dimensions: (1) falseness and (2) public health significance using 5-point Likert scale: 1 ("not at all") to 5 ("extremely false/important").

Results: Thirty-two sleep myths were identified across three developmental categories: infant (14 myths), child (6 myths), and adolescent (12 myths). Mean expert ratings illuminated the most pressing myths in each developmental category: infant sleep ("Sleep training causes psychological harm, including reduced parent-child attachment:" falseness =4.7, s.d.=0.7; public health significance=4.0, s.d.=1.1); child sleep ("Heavy, loud snoring for my child means he's sleeping deeply:" falseness=4.8, s.d.=0.6; public health significance=4.7, s.d.=0.7), and teenager sleep ("Falling asleep in class means your teenager is lazy and not motivated:" falseness=4.8, s.d.=0.5; public health significance=4.3, s.d.=0.8).

Conclusion: The current study identified commonly-held myths about infant, child, and adolescent sleep that are not supported by (or worse, counter to) scientific evidence. If unchecked, these myths may hinder sleep at a critical developmental stage. Future research may include public health education to correct myths and promote healthy sleep among infants, children, and teenagers. **Support:** 5T32HL007901

0409

THE EFFICACY OF AFTERNOON-EVENING SLEEP FOLLOWING NIGHT SHIFTS ON SLEEP AND ALERTNESS IN NURSES WITH ROTATING SHIFT WORK SCHEDULE: REAL WORLD DATA

Kim, J.^{1,2} Han, S.³ Kim, S.⁴ Duffy, J.⁵

¹Department of Neurology, Dankook University College of Medicine, DankKook University Hospital, Cheonan, KOREA, REPUBLIC OF, ²Department of Neurology, Ewha Womans University Seoul hospital, Seoul, KOREA, REPUBLIC OF, ³Department of Neurology, Wonkwang University Sanbon Hospital, Sanbon, KOREA, REPUBLIC OF, ⁴Dankook University, Cheonan, KOREA, REPUBLIC OF, ⁵Division of Sleep and circadian disorders, Department of Medicine and Neurology, Brigham and Women's hospital, Boston, MA.

Introduction: The aim of this study was to investigate the efficacy of changing sleep timing to afternoon-evening following nightshifts in hospital nurses with three rapid rotating shift schedules. **Methods:** Hospital nurses with three rotating shift schedules were enrolled for a 1-month pre-intervention and a 1-month intervention study. During the Intervention, sleep timing following nightshifts was directed to afternoon-evening sleep for 8h time-in-bed (TIB) after 1 PM, and ad-lib sleep schedule for other shifts. Baseline and follow-up evaluation included sleep schedule, sleep duration, Epworth sleepiness scale (ESS), insomnia severity index (ISI) for each shift, Beck depression inventory (BDI), and Beck anxiety inventory (BAI). Sleep was assessed by sleep diary and actigraphy. Alertness during the night shift was evaluated using the Karolinska

sleepiness scale (KSS) in the beginning and at the end of the shift by texts sent to their cell phones. The participants were asked to give feedback and a willingness to continue this intervention.

Results: A total of 26 subjects (30.7 ± 8.5 years, 25 female) finished the study among 29 nurses who participated in the study. The shift work was 6.5 ± 8.0 years. The mean morningness-eveningness scale was $42.1\pm8.0(31-62)$. TIB following nightshifts were 379.9 ± 91.2 and 478.4 ± 48.7 min for preintervention and intervention, respectively (p=0.001). Total sleep time (TST) was 328.0 ± 91.0 vs. 361.0 ± 70.4 min, respectively following nightshifts (p=0.187, Cohen's drm = 0.467). BDI, BAI, ESS, and ISI were significantly improved after the intervention. 60.7% and 49% of the participants reported improved alertness, and work efficiency during the nightshift. 17.9% and 42.9% of the participants reported increased sleep duration, and improved sleep quality after nightshift, respectively. Only eight participants were willing to continue the afternoon-evening sleep schedule following night shifts. KSS was not different between pre-intervention and intervention.

Conclusion: The afternoon-evening sleep schedule modestly increased total sleep time following nightshift. The overall mood, sleepiness and insomnia scale improved after the intervention although the alertness assessed by KSS failed to show the difference. The individual difference should be considered for applying afternoon-evening sleep for rapid rotating shift schedules.

Support: 2018 Research award grants from the Korean sleep research society and NRF-2019R1A2C1090643 funded by the Korean national research foundation

0410

INSUFFICIENT SLEEP AMONG HEALTHCARE PROFESSIONALS

Jenkins, D.¹ Peprah, R.¹ Donley, T.¹ Sexias, A.¹ Khosrof, A.¹ Jean-Louis, G.¹

¹NYU Grossman School of Medicine, New York, NY, ²NYU Grossman School of Medicine, New York, NY.

Introduction: Insufficient sleep (IS) is a common problem among healthcare professionals, especially those who are shift workers. Evidence has shown that sleeping less than eight hours can lead to sleep debt. Sleep debt can have a negative impact on the mental, emotional and cognitive well-being of health care providers. In addition to sleep debt, having long shift hours reduces the opportunity for sleep because there is less time to recuperate.

Methods: We analyzed data gathered from healthcare workers (n=4,093) from the 2017 and 2018 National Health Interview Survey (NHIS), a nationally representative study of the US civilian non-institutionalized population. Sleep was categorized as short (≤ 6 hrs), normal/healthy (7-8 hrs), and long (≥ 9 hrs) sleep. Using STATA 15.0 for Windows, we report weighted frequencies and Chi square tests. Alpha of 0.05 was used for all significance levels.

Results: Of the sample, 18% were male and 82% were female. The mean age was 50.7 ± 17.5 . The majority of the sample was White (77%), 12% were Black and 9% were of other minority". The proportion of women who reported short sleep (31%) and normal (45%) were significantly higher than men (p<0.000). Healthcare workers under 30 had the highest proportions of short sleep compared to any other age groups (p<0.000). Lower numbers were associated with long sleep among those who worked directly with patients compared to workers who did not (p<0.000). Healthy sleep was significantly associated with not having trouble falling asleep, staying asleep, not taking sleep medications, and feeling rested 7 or more times in the past week (p<0.000).

Conclusion: Our study explores sleep patterns among healthcare professionals. Previous studies have shown that this population is more susceptible to insufficient sleep which leads to sleep debt. We found that the current data suggest that this association may have changed for healthcare professionals today.

Support: This study was supported by funding from the NIH: R01MD007716, R01HL142066, K01HL135452, and K07AG052685.

0411

ASSOCIATION BETWEEN HEALTHY DIETARY PATTERNS AND SELF-REPORTED SLEEP DISTURBANCES IN OLDER MEN

van Egmond, L. T. Tan, X. Benedict, C. Uppsala University, Uppsala, SWEDEN.

Introduction: Ageing is often accompanied by an increased prevalence of sleep problems. Healthy lifestyle choices, including diet, are especially important to mitigate such impairments. To date, little is known about how dietary patterns may link to measures of sleep quality in older subjects. Therefore, we investigated in a Swedish older male population, whether adherence to the Mediterranean Diet (MD) or the Healthy Diet Indicator (HDI; based on recommendations from the World Health Organization) is linked to a decrease in sleep disturbances.

Methods: We studied 970 men (age: 71 \pm 1yr) from the ULSAM dataset. Sleep initiation or maintenance problems were evaluated by self-reporting questionnaires. Dietary intake was recorded with a precoded seven-day food diary. To calculate the adherence scores, intake of dietary components was assessed. Traditional MD components included fat quality, vegetables, fruits, cereals, fish, meat, dairy, and alcohol. The HDI components included saturated-, polyunsaturated fatty acids, proteins, carbohydrates, sucrose, fiber, fruits and vegetables, cholesterol, and fish. Reasonable dietary reporting was calculated to identify possible under- or over-reporters.

Results: When adjusted for potential confounders, no associations between dietary scores and sleep parameters were found. In contrast, low consumption of dairy products —one of the dietary features of the MD —was associated with better subjective sleep initiation. This association was, however, not found in men with adequate reports of daily energy intake (~54% of the cohort).

Conclusion: Our findings do not suggest that older men can mitigate perceived sleep problems by adhering to the MD or HDI. Whether low consumption of dairy products can facilitate sleep initiation must be confirmed in future studies by utilizing objective measures of sleep, such as polysomnography. Finally, when investigating associations between dietary patterns and sleep, particular attention should be paid to the potential confounder of inadequate reporting of energy intake.

Support: This research was funded by Novo Nordisk Foundation (NNF14OC0009349), Swedish Brain Foundation, Swedish Research Council (2015-03100), Åke Wiberg Foundation (M17-0088), Fredrik and Ingrid Thuring Foundation (2017-00313) and the Swedish Medical Research Society.

0412

PARENTS SLEEP LONGER WHEN SCHOOL IS OUT FOR THE SUMMER: ASSOCIATIONS AMONG PARENTHOOD, GENDER, AND SEASON

Ruder, M. Rus, H. M. Raj, A. Gahan, L. O'Mullane, B. Danoff-Burg, S. Weaver, M. Raymann, R. SleepScore Labs, Carlsbad, CA.

Introduction: Seasonal effects in sleep are often attributed to day length; however, change in obligatory daily activities might also have an impact on sleep behavior. Longitudinal measurement using consumer sleep technology enables the observation of patterns in sleep behavior in the home environment. We analyzed the impact of parenthood and gender on total sleep time (TST) over the summer break period using data collected in the home.

Methods: Sleep data were collected using the SleepScore mobile application from October 2018 through October 2019, with the summer break period defined as June 25th - August 5th. U.S. age and gender matched samples of parents and non-parents were selected using Mahalanobis distance from a pool of users more likely to have school-aged children. The final samples included n=345 parents (38.7 +/- 4.5 years) and n=345 non-parents (37.8 +/- 4.7 years); both groups were 46% female. Only weeknights (n=34,323) were analyzed to maximize impact of school schedule. Linear regression and independent t-tests were used to analyze main and interaction effects for gender, parenthood, and summer break.

Results: Male gender, parenthood, and summer break were associated with decreased sleep duration (ps < .01). However, during summer break, parents exhibited an increase in TST, with mothers (+5.6 mins) having a greater increase than fathers (+1.1 mins). In contrast, adults without children showed a decrease in TST during summer break, with males having a greater reduction (-8.8 mins) than females (-6.5 mins).

Conclusion: These results suggest that parental status may play a part in seasonal sleep patterns. Contrary to the typical trend of shorter TST during summer, being a parent is associated with longer TST during summer break, with a greater increase for females. This change may be attributed to parents following a less rigid schedule when their children are not in school. **Support:** N/A

0413

THE INFLUENCE OF COMMUNITY ENVIRONMENT EXPOSURE AND INDIVIDUAL HEALTH BEHAVIOR ON INSUFFICIENT SLEEP

Shin, J. Grigsby-Toussaint, D. Brown University, Providence, RI.

Introduction: Short sleep duration is associated with the risk of various chronic diseases, and it has been hypothesized to influence health behaviors and environmental exposure such as green space and noise. However, little studies have explored this relationship, especially with the consideration of the integrated environment information. The purpose of this study is to examine the influence of environmental exposure and individuals' health behavior on sleep duration.

Methods: We examined aggregate national-level datasets regarding health information, noise, and green space. Sleep, health behavior, and socio-demographic variables were derived from 500 cities data, and the unit of analysis was the prevalence of each variable in the census tract. The insufficient sleep was calculated by the percentage of the population who had less than 7 hours of sleep in the census block. Spatial analysis was performed for green space and noise measurement; both values were calculated within the community activity space, which is a combined area with the census urban area and census place to examine the potential activity space. Multiple linear regression analysis was performed to model the relationship. **Results:** The final sample was 29,610 census tracts from 497 cities, and the average median age of each census tract was 36.02 ± 7.085 . 36.64% of insufficient sleep was reported from the entire sample.

The model (R²=0.804) indicate that green space (β =-.053, p<.001) and natural noise (β =-.029, p<.001) has negatively associated with insufficient sleep; while, smoker (β =.374, p<.001), binge drinker (β =.105, p<.001), no leisure physical activity time (β =.111, p<.001), and artificial noise (β =.034, p<.001) has positive association with insufficient sleep.

Conclusion: The results indicate that a better environment source in the community mitigates the insufficient sleep population as well as individual health behavior. Further studies are needed, however, to fully disentangle the association between sleep duration and sleep quality associated with other environmental exposure. **Support:** N/A

0414

CHRONOTYPE AND SLEEP AMONG OVARIAN CANCER SURVIVORS PARTICIPATING IN A LIFESTYLE INTERVENTION

Crane, T. E.¹ Skiba, M. B.² Donzella, S.² Thomson, C. A.² Parthasarathy, S.³

¹University of Arizona Biobehavioral Health Sciences, College of Nursing, Tucson, AZ, ²University of Arizona Mel and Enid Zuckerman College of Public Health, Tucson, AZ, ³University of Arizona College of Medicine, Tucson, AZ.

Introduction: Chronotype is defined as an individual's propensity to sleep at a specific time in a 24-hour cycle with late chronotype associated with poorer health outcomes including cancer. The role of chronotype on lifestyle behaviors remains relatively undefined in ovarian cancer. The Lifestyle Intervention for oVarian cancer Enhanced Survival study is testing whether 1205 women randomized to a diet and physical activity intervention for 24-months will have longer progression-free survival versus attention control. Here we determine the frequency and predictors of late versus early and mid chronotypes in disease-free ovarian cancer survivors.

Methods: 894 ovarian cancer survivors with baseline measures were included in analyses. Chronotypes were determined using self-reported time to bed (early- < 9 pm; mid- ≥ 9 pm - ≤ 12 am; late- >12 am) captured through the Pittsburgh Sleep Quality Index. Demographic, diet and physical activity data were captured with validated questionnaires and BMI measured in clinic. Descriptive statistics and logistic regression, adjusted for smoking status and race, were performed.

Results: 12.4% of women were late chronotype with significant differences between chronotypes observed for race, smoking history, sleep duration, and physical activity (p < 0.05). Late chronotype reported fewer hours of sleep per night (6.54 ± 1.51 hrs) compared to mid (7.10 ± 1.31 hrs) and early (7.74 ± 1.30 hrs) chronotype. Blacks had higher odds of being late chronotype, OR 4.28 (95% CI 2.16-8.46). Late chronotype were more likely to report a history of smoking and lower recreational activity and had a higher mean BMI of 29.1 \pm 6.0 kg/m² compared to mid and early chronotype 27.8 \pm 6.2 kg/m² and 27.4 \pm 5.4kg/m², respectively. No significant differences were observed for sleep or diet quality, age, education or employment status.

Conclusion: Results of this analysis are consistent with other community-based population studies with regard to chronotype and race. Ovarian cancer is aggressive and late chronotype are more likely to have other risk factors that elevate risk of recurrence (obesity, tobacco use and inactivity. Six-month data are being analyzed by treatment arm and will provide important insights as to the role of sleep phase and lifestyle behaviors in this vulnerable population.

Support: NCT00719303; NCI R01CA186700-01A1

METABOLIC DYSFUNCTION AND SLEEP DISRUPTION IN MODELS OF ALZHEIMER'S DISEASE

Carroll, C. M.¹ Stanley, M.² Macauley, S. L.¹ ¹Wake Forest School of Medicine, Winston Salem, NC, ²University of British Columbia, Vancouver, BC, CANADA.

Introduction: Metabolic perturbations and sleep disruptions are a cause and consequence of Alzheimer's disease pathophysiology. A bidirectional relationship exists where impairments in sleep and metabolism contribute to the development of Alzheimer's disease while the presence of Alzheimer's disease pathology leads to decreased cerebral metabolism, peripheral glucose intolerance, and disrupted sleep. While the effects of type 2 diabetes (T2D) and Alzheimer's disease on sleep have been explored separately, no previous studies have examined the effect of acute glycemic variability, a defining feature of T2D, on sleep in the context of Alzheimer's disease. The goal of this study is to determine how glycemic variability drives sleep disruptions by modifying the relationship between cerebral glucose metabolism and neuronal activity.

Methods: Biosensors are implanted bilaterally into the hippocampus of APP/PS1, a model of amyloid-beta (A β) overexpression, and P301S, a model of tau deposition and neurodegeneration, mice to measure ISF glucose and lactate, markers of cerebral metabolism and neuronal activity, respectively. Simultaneous cortical EEG/EMG recordings are used for sleep/wake scoring and analysis.

Results: Glycemic fluctuations cause a decoupling of the typical relationships between cerebral metabolism and neuronal activity, while also increasing arousal in 3-month-old, wildtype mice. The presence of AD-like pathology results in a similar, albeit muted cerebral metabolic response to peripheral glycemic variability, but a diminished effect on wakefulness, likely due to age- and pathology-dependent increases in overall time spent awake. Conversely, in aged, P301S mice, cerebral metabolic responsiveness is lost and a ceiling effect on wakefulness emerges, suggesting differential effects on sleep with tau accumulation. Moreover, aged mice show progressive disruptions to overall sleep quality and quantity, highlighting the synergism existing between AD-like pathology and glucose intolerance in sleep dysfunction. Lactate seems to be a common driver of disruption in this synergistic cycle.

Conclusion: This study represents a novel approach to defining the dynamic interplay between risk factors for AD and T2D and suggests a feedforward loop of disease progression where disrupted sleep can alter the relationship between neuronal activity, metabolism, and pathology.

Support: NIH/NIA 1K01AG050719. Harold and Mary Eagle Fund for Alzheimer's Research New Vision Award through Donors Cure Foundation

0416

POOR SLEEP QUALITY PREDICTS SERUM MARKERS OF NEURODEGENERATION AND COGNITIVE DEFICITS IN WARRIORS WITH MILD TRAUMATIC BRAIN INJURY

Werner, K.¹ Shahim, P.² Gill, J.² Nakase-Richardson, R.³ Kenney, K.¹

¹Uniformed Services University of Health Sciences, Bethesda, MD, ²National Institutes of Health, Bethesda, MD, ³University of South Florida, Tampa, FL.

Introduction: Increasing evidence links neurodegeneration to traumatic brain injury (TBI), and a separate body of literature links

neurodegeneration to sleep dysfunction, implicating increased toxin production and decreased glymphatic clearance. Sleep disorders affect 50% of TBI patients, yet the sleep-neurodegeneration connection in these patients remains unexplored. We hypothesized that warfighters with TBI and sleep dysfunction would have increased neuronal injury, revealing potential mechanistic underpinnings for TBI outcomes. We measured plasma biomarkers, cognitive function and sleep surveys for correlation analysis.

Methods: In a retrospective cross-sectional study of warfighters (n=113 chronic mild TBI patients), the Pittsburgh sleep quality index (PSQI) was compared with amyloid β 42 ($A\beta$ 42), neurofilament light (NFL), tau, and phospho-tau (threonine 181) isolated from plasma and exosomes. Executive function was tested with the categorical fluency test. Exosomes were precipitated from plasma. Proteins were measured with the Single Molecule Array (Quanterix). Linear models were adjusted for age, ApoE, and number of TBIs.

Results: Poor sleepers with TBI (PSQI>8) had elevated NFL compared to good sleepers in plasma (p=0.007) and exosomes (p=0.00017), and PSQI directly correlated with NFL (plasma: Beta=0.23, p=0.0079; exosomes: Beta=2.19, p=0.0013) stronger than any other marker of neurodegeneration. Poor sleepers also showed higher obstructive sleep apnea (OSA) risk compared to good sleepers by STOP-BANG scores (3.6, SD=1.6 vs 2.8, SD=1.74; p=0.0014) as well as decreased categorical fluency (20.7, SD=4.1) (18.3, SD=4.6, p=.0067). Plasma tau and Aβ42 also correlated with PSQI (Beta=0.64, p=0.028, and Beta=0.40, p=0.049 respectively).

Conclusion: This is the first reported data correlating markers of neuronal injury and cognitive deficits with sleep complaints and OSA risk in patients with TBI - possibly identifying treatable pathophysiological mediators of TBI neurodegeneration. Limitations include a small sample size, lack of objective sleep measures, and inability to establish directionality due to cross-sectional design. Prospective trials will be required to further explore our proposed hypothesis. If confirmed, these findings would call for targeting sleep disorders in the TBI population to mitigate risk of neurodegeneration.

Support: This work was supported by grant funding from: Department of Defense, Chronic Effects of Neurotrauma Consortium (CENC) Award W81XWH-13-2-0095 and Department of Veterans Affairs CENC Award I01 CX001135.

0417

ASSOCIATIONS BETWEEN REST-ACTIVITY PATTERNS AND RESTING-STATE NETWORKS IN OLDER ADULTS

Moon, C.¹ Cole, R. A.² Xiao, Q.³ Voss, M. W.⁴ ¹University of Iowa, College of Nursing, Iowa City, IA, ²University of Iowa, Carver College of Medicine, Iowa City, IA, ³University of Iowa, Department of Health and Human Physiology, Iowa City, IA, ⁴University of Iowa, Department of Psychological and Brain Sciences, Iowa City, IA.

Introduction: Resting-state functional connectivity is coherent brain activity in a task-free state that strongly correlates to taskevoked sensory, motor, and higher-order cognitive systems. Certain networks show decreased functional connectivity with aging. Aging is associated with changes in circadian rhythms and sleep-wake cycles. Limited research has been conducted on how circadian activity and sleep are related to markers of functional brain aging. The purpose of this study was to explore whether rest-activity patterns and shorter sleep duration are related to functional connectivity of specific resting-state networks in older adults. **Methods:** A total of 124 cognitively normal participants (mean age (SD) = 67.2 (5.7), 42% men) underwent 3.0 T MRI and week-long wrist actigraphy protocols. Rest-activity pattern was analyzed using an extended cosine model calculating acrophase (time of peak activity) and pseudo-F statistics of goodness-of-fit (a measure of overall rhythmicity). We used resting-state fMRI scans to measure functional connectivity in association and sensory networks as defined by the Schaefer 17 network functional atlas. Multiple linear regression analysis was used to investigate how rest-activity pattern parameters and sleep duration are associated with resting-state functional connectivity, adjusting for age, sex, and sleep apnea.

Results: We found that the average acrophase was 2:30 PM (SD = 54 min), and delayed acrophase (average vs. delayed [+1SD]) was associated with lower functional connectivity of the right-lateralized default mode network A (p=0.02), and higher pseudo-F statistics was associated with higher functional connectivity in networks including left dorsal attention B (p=0.001), right somatomotor A (p = 0.05), and somatomotor B (both p=0.02). Longer sleep duration was associated with higher right executive control B (p=0.03).

Conclusion: The overall rhythmicity of diurnal rest-activity patterns and longer sleep duration are associated with some restingstate functional networks. Further investigation is needed to understand the mechanisms between circadian rhythm and brain function.

Support: National Institute of Health, U of Iowa Aging Mind Brain Initiative, Center on Aging

0418

EFFECT OF ACUTE ADMINISTRATION OF DORA-12 ON SLEEP IMPAIRMENT IN THE AGED PS19 MOUSE MODEL OF TAUOPATHY

Kam, K. Vetter, M. Berryman, N. Varga, A. Icahn School of Medicine at Mount Sinai, New York, NY.

Introduction: Aged PS19 mice (MAPT P301S), a mouse model of tauopathy and neurodegeneration, display reduced NREM and REM sleep starting around 8-9 months before death around 12 months. Here, we tested the acute effect of a dual orexin receptor antagonist (DORA-12) on sleep in 11 mice (5 male, 6 female) at 10.3 ± 1.8 months.

Methods: Two consecutive 24-hour recordings (12/12hr L:D cycle) were scored semi-automatically for non-REM sleep, REM sleep, and wake in mice implanted with EEG/EMG. Mice were treated with either vehicle (day 1) or 100mg/kg of DORA-12 (day 2) by oral gavage at both ZT0 and ZT9.

Results: After the first dose at ZT0, both latency to the first NREM sleep episode (paired t-test p=0.002) and to the first REM sleep episode (paired t-test p=0.005) was significantly shorter with DORA-12 (NREM: 20.8±17.8 min.; REM: 23.5±21.2 min.) compared to vehicle (NREM: 49.2±22.3 min.; REM: 127.0±93.3 min.). There was no difference in NREM or REM sleep latency observed after the second dose at ZT9. DORA-12 treatment increased NREM duration across the 24hr period (DORA-12: 664±52 min.; Veh: 601±54 min., paired t-test p=0.007) and also after the 2nd dose (DORA-12: 311±65 min.; Veh: 263±84 min., paired t-test p=0.009). DORA-12 treatment also increased REM duration across 24hrs (DORA-12: 61±30 min.; Veh: 48±29 min., paired t-test p=0.014) but not after the 2nd dose alone (DORA-12: 22±14 min.; Veh: 20±15 min., paired t-test p=0.388). Notably in both vehicle and DORA-12 conditions, we observed apparent dream enactment behavior including mastication, paw grasp, and fore limb extension during REM in 3 of 11 PS19 mice (all male), not typically observed in younger PS19 or age-matched non-transgenic mice, suggestive of a possible REM behavior disorder (RBD) phenotype. Wake-like behaviors occurred during theta-dominant EEG but with an EMG amplitude >4SD the preceding NREM sleep baseline for at least > 1sec.

Conclusion: In aged PS19 mice, DORA-12 was found to decrease the latency to NREM and REM after the first dose while also increasing NREM and REM duration across the entire 24hr recording period. We also capture a heretofore undescribed RBDlike phenotype in aged PS19 tauopathy mice. **Support:** Merck MISP

0419

IL-6 MODERATES THE ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA SEVERITY AND INCIDENT ALZHEIMER'S DISEASE: THE FRAMINGHAM HEART STUDY

Baril, A.¹ Beiser, A. S.¹ Redline, S.² McGrath, E. R.³ Aparicio, H. J.¹ Gottlieb, D. J.³ Seshadri, S.⁴ Himali, J. J.⁴ Pase, M. P.⁵

¹The Framingham Heart Study, Boston University School of Medicine, Boston, MA, ²Brigham & Women's Hospital, Harvard Medical School, Boston, MA, ³Harvard Medical School, Boston, MA, ⁴Glenn Biggs Institute for Alzheimer's & Neurodegenerative Diseases, University of Texas Health Sciences Center, San Antonio, TX, ⁵The University of Melbourne, Melbourne, AUSTRALIA.

Introduction: Both sleep disturbances and inflammation are potential risk factors for Alzheimer's disease (AD). However, it is unknown how inflammation and sleep interact together to influence the risk of developing AD dementia. Our objective was to evaluate whether interleukin-6 (IL-6) levels interact with sleep disturbances when predicting incident clinical AD.

Methods: We studied participants in the Framingham Heart Study Offspring cohort who completed in-home overnight polysomnography. Sleep characteristics were continuous and included sleep duration, wake after sleep onset (WASO), and apneahypopnea index (AHI). Participants were stratified into quartiles of IL-6 levels. Surveillance for incident AD dementia occurred over a mean follow-up of 13.4 ± 5.4 years. Using Cox proportional hazards regression models, we tested the interaction of sleep measures by IL-6 quartiles on incident AD dementia. All analyses adjusted for age and sex and P<0.05 was considered significant.

Results: The final sample included 291 dementia-free participants at baseline (age 67.5 \pm 4.9 years, 51.6% men). Approximately one quarter of participants had obstructive sleep apnea (OSA; AHI>15) at baseline (median:6.2, Q1:2,3, Q3:14.3). We observed 33 cases of incident AD dementia during follow-up. Although no interaction was observed for either sleep duration or WASO with IL-6 levels, there was a significant interaction of AHI with IL-6 in predicting AD dementia (p=0.002). In the lowest IL-6 quartile, higher AHI was associated with an elevated risk of AD dementia (hazard ratio, 4.15 [95%CI, 1.42, 12.1], p=0.01) whereas no association between AHI and incident AD was observed in other IL-6 quartiles.

Conclusion: Our findings suggest that the pro-inflammatory cytokine IL-6 moderates the association between OSA and incident AD risk. The association between increasing OSA severity and incident AD was only observed in those with lower IL-6

levels, suggesting that this association might be especially apparent when no other confounding risk factors such as inflammation are present.

Support: The Framingham Heart Study is supported by contracts from the National Heart, Lung and Blood Institute, grants from the National Institute on Aging, and grants from the National Institute of Neurological Disorders and Stroke.

0420

FIBRE-SPECIFIC WHITE MATTER NEURODEGENERATION IS ASSOCIATED WITH LONG SLEEP DURATION AFTER STROKE

Gottlieb, E. W. Egorova, N. Khlif, M. S. Khan, W. Werden, E. Pase, M. P. Howard, M. Brodtmann, A.

University of Melbourne, Florey Institute of Neuroscience, Melbourne, AUSTRALIA.

Introduction: Long sleep duration in aging populations has recently been proposed as a key modifiable risk factor and sequela of stroke. It is unclear whether the pathogenesis of post-stroke sleep-wake dysfunction is due to focal infarction to regional sleep-wake hubs in the brain, or to accelerated whole-brain neurodegeneration. We utilise a novel technique known as whole-brain fixel-based analyses (FBA) to characterize the first fibre-specific white-matter markers of long sleep duration after stroke.

Methods: We included 98 radiologically-confirmed ischemic stroke participants (67 male; mean age = 68) and 40 age-matched controls with no history of neurodegenerative disease imaged 3-months post-stroke. Sleep-wake was measured for one week using BodyMedia's SenseWear armband. Diffusion-weighted MRI (DWI) were acquired using echoplanar imaging and preprocessed using MRtrix3. FBA were employed to identify tracts with altered white-matter fibre-density and fibre-bundle cross-section (FDC) in the long sleep duration (>8 hr, n=20) and normal sleep duration groups (between >6 hr and <8 hr, n=59) compared to controls. Statistical comparisons of FDC between groups were performed at each FDC fixel by a general linear model controlling for age, sex, and intracranial volume.

Results: Stroke participants with long sleep duration exhibited significant FDC reductions of up to 40% within the cortico-ponto-cerebellar tract when compared to healthy controls (family-wise-error-corrected p=<0.05). Bilateral pontine degeneration was observed at the decussation of the superior cerebellar peduncles. Stroke participants with normal sleep duration exhibited diffuse whole-brain degeneration most apparent along the corpus collosum and cingulum; however, the distribution was less extensive relative to long sleepers (i.e., no cortico-cerebellar projections) and percentage effect reductions did not exceed 20%.

Conclusion: Long sleep duration after stroke is associated with cortico-ponto-cerebellar degeneration when compared to controls or stroke-participants with normal sleep duration. Excessively long sleep may contribute to post-stroke neurodegeneration beyond the effects of direct infarction and may be a modifiable pharmacological target for abating brain volume loss after stroke.

Support: This work was supported by the National Health and Medical Research Council project grant (APP1020526), the Brain Foundation, Wicking Trust, Collie-Trust, and Sidney and Fiona Myer Family Foundation. NE was supported by the Australian Research Council DECRA award DE180100893.

0421

DECREASED ACTIGRAPHIC DAYTIME ACTIVITY IS ASSOCIATED WITH LOWER MEMORY PERFORMANCE IN COGNITIVELY-UNIMPAIRED INDIVIDUALS WITH AUTOSOMAL DOMINANT ALZHEIMER'S DISEASE

Pardilla-Delgado, E.¹ Ramirez Gomez, L.¹ Baena, A. Y.² Montes, M. I.³ Bocanegra, Y.² Martinez, J. E.¹ Lopera, F.² Quiroz, Y. T.¹

¹Massachusetts General Hospital, Boston, MA, ²Grupo de Neurociencias de Antioquia, Universidad de Antioquia, Medellin, COLOMBIA, ³Universidad de Antioquia, Medellin, COLOMBIA.

Introduction: Alzheimer's disease (AD) impacts brain regions that control circadian regulation systems such as wakefulness and daytime physical activity. Recent evidence shows that AD pathology is damaging for wake-promoting neurons. Whether early changes in wakefulness and daytime activity occur during asymptomatic stages of familial AD (fAD) remains unknown. In this study, we aimed to investigate whether daytime activity differs between cognitively-unimpaired carriers of early-onset fAD and agematched non-carrier family members. Further, we examined the associations between daytime activity and memory performance.

Methods: A total of 25 members of the large Colombian kindred with the Presenilin1 (*PSEN1*) E280A mutation were included in the study (9 mutation carriers and 16 non-carriers, mean age=38.2). *PSEN1* mutation carriers develop dementia before the age of 50. All subjects underwent wrist actigraphy for 7-14 days to measure daytime activity (average activity per minute and per epoch), and completed the CERAD Word List Learning and the Free and Cued Selective Reminding Test (FCSRT).

Results: Compared to non-carriers, mutation carriers had less average daytime activity (Mann-Whitney U Test p=.04). Higher average daytime activity was associated with better memory recall in both the CERAD word list delayed recall (r=.47, p=.05) and the FCRST delayed total recall (r=.53, p=.02). No associations with age were observed.

Conclusion: Our results suggest that cognitively-unimpaired mutation carriers have reduced daytime activity, years before the onset of dementia. Reduced daytime activity in carriers is also associated with lower memory performance. Our preliminary findings add to the growing evidence that circadian dysfunction is present in early AD, and may play an important role in subsequent memory impairment. Future research with large samples is needed to further examine sleep and circadian dysfunction in asymptomatic individuals at genetic risk for AD. **Support:** NIA 5R01AG054671-03 to YTQ

0422

APOCALYPSE TAU: THE RELATIONSHIP BETWEEN INFLAMMAGING AND LOCAL SLEEP DISRUPTION IN OLDER ADULTS IS MEDIATED BY TAU BURDEN

Dave, A.¹ Sprecher, K. E.² Lui, K. K.¹ Chappel-Farley, M. G.³ Chen, I. Y.¹ Blennow, K.⁴ Zetterberg, H.⁴ Riedner, B. A.² Bendlin, B. B.⁵ Mander, B. A.¹ Benca, R. M.¹

¹Department of Psychiatry and Human Behavior, University of California, Irvine, CA, ²University of Wisconsin School of Medicine and Public Health, Department of Psychiatry, Madison, WI, ³Department of Neurobiology and Behavior, University of California, Irvine, CA, ⁴Department of Psychiatry and Neurochemistry, Institute of Neuroscience and Physiology, the Sahlgrenska Academy at the University of Gothenburg, Mölndal, SWEDEN, ⁵University of Wisconsin School of Medicine and Public Health, Department of Medicine, Madison, WI.

Introduction: Chronic inflammation in aging is independently associated with tau burden and sleep disruption, though the mechanism linking inflammation with sleep disruption remains unknown. Recent evidence associates tau burden with deficits in local expression of sleep spindles and slow wave activity (SWA). Here we test the hypothesis that age-related central inflammation disrupts local sleep by influencing tau pathology.

Methods: Cognitively asymptomatic older adults from the Wisconsin Alzheimer's Disease Research Center underwent overnight polysomnography with high-density electroencephalography (hdEEG; 256 channels) at the University of Wisconsin-Madison (n=33, 61.9±6.7 years, 23 female). EEG data were subjected to multitaper spectral analysis (0.5-40Hz) to yield topographic maps of SWA (SWA1:0.5-1Hz, SWA2:1-4.5Hz) and spindle (sigma1:11-13Hz; sigma2:13-16Hz) power during NREM sleep. Cerebrospinal fluid assay-based measurements of YKL-40 (indicating glial activation), phosphorylated tau (P_{tau}), and total tau (T_{tau}), were correlated with SWA and sigma topographical power employing Holm-Bonferroni correction. Multiple linear regression models were implemented controlling for age, apnea-hypopnea index (AHI), and sex at significant derivations. Finally, Sobel testing was employed to assess whether tau burden mediated YKL-40-sleep associations.

Results: Age was associated with YKL-40 (r=0.53, p=0.002), and YKL-40 was associated with both P_{tau} (r=0.66, p<0.001) and T_{tau} (r=0.68, p<0.001). Correlations between sigma2 activity and both P_{tau} and T_{tau} were detected at 14 derivations, 12 of which remained significant after controlling for age, sex, and AHI. YKL-40 was associated with sigma2 power (r=-0.39, p=0.025) across derivations expressing peak significance with tau. Sobel mediation analyses indicated that both P_{tau} (t=-2.15, p=0.031) and T_{tau} (t=-2.36, p=0.018) mediated the relationship between YKL-40 and sigma2 activity at these derivations. SWA was not associated with T_{tau} , P_{tau} , or YKL-40.

Conclusion: These results suggest that age-related increases in central glial activation may disrupt local expression of fast spindles by increasing tau burden, highlighting a potential role for chronic inflammation in sleep deficits observed in aging and Alzheimer's disease.

Support: Supported by R56 AG052698, P50AG033514

0423

OLDER AGE MODIFIES THE ASSOCIATION BETWEEN COMBINED SLEEP DISORDERED BREATHING AND SLEEP DURATION WITH NEUROCOGNITIVE DECLINE IN HISPANIC/LATINO ADULTS

Kaur, S.¹ Tarraf, W.² Wu, B.³ Daviglus, M.⁴ Shah, N.⁵ Sotres-Alvarez, D.⁶ Gallo, L.⁷ Wohlgemuth, W.⁸ Redline, S.⁹ Gonzales, H.³ Ramos, A.¹

¹University of Miami Miller School of Medicine, Miami, FL, ²Wayne State University, Detroit, MI, ³University of California San Diego, San Diego, CA, ⁴University of Illinois Chicago, Chicago, IL, ⁵Mount Sinai Icahn School of Medicine, New York, NY, ⁶University of North Carolina at Chapel Hill, Chapel Hill, NC, ⁷San Diego State University, San Diego, CA, ⁸Miami VA Healthcare System, Miami, FL, ⁹Brigham & Women's Hospital, Harvard Medical School, Boston, MA.

Introduction: We aimed to determine if age or sex modifies associations between sleep-disordered breathing (SDB), sleep duration and severe phenotypes of combined SDB/sleep duration with 7-year neurocognitive change (NC) in a diverse sample of U.S. Hispanic/Latinos.

Methods: We analyzed data of 5,235 adults 50-80 years of age from SOL-INCA, an ancillary to the Hispanic Community Health Study/Study of Latinos that determines the risk factors for NC. The main outcome was NC after a mean follow-up of 7-years on measures of memory (SEVLT sum and SEVLT recall), language (word fluency), processing speed (DSS) and a cognitive impairment screener. We evaluated the effect of baseline SDB (AHI \ge 15), sleepiness (Epworth Sleepiness Scale, ESS \ge 10), self-reported sleep duration (i.e. <6 hours, 6-9 hours, \ge 9 hours), age and sex on NC. Survey linear regression models with interaction terms were used to examine the relationship between SDB, sleep duration, combinations of SDB and sleep duration phenotypes and NC. Depression, vascular risk, sleep medication, and study site were entered into all models as covariates.

Results: Overall, the mean age was 56.0 years, 54.8% females, 62.2% completed high school, 17.3% had SDB, 6.6% had short sleep,and 14.8% had long sleep. Sleep duration and SDB were not associated with NC. There was a significant interaction between agexSDB+sleep duration on delayed recall ($F_{10,599}$ = 2.40, *p*=0.01) and processing speed ($F_{10,597}$ = 2.55, *p*=0.01). Combined SDB + short sleep was associated with decline in processing speed (β =-0.6, 95% CI= [-1.2, -0.1], and combined SDB+long sleep was associated with decline in verbal memory (β =-0.9, 95% CI=[-1.7, -0.2] in adults aged ≥ 65 years. There was no association in participants aged <65 years and no sex differences.

Conclusion: Age, but not sex, modified the association between SDB and sleep duration with decline on processing speed and verbal memory. Sleep interventions tailored for older adults may be useful in slowing or preventing neurocognitive decline.

Support: This work is supported by National Institute on Aging (R01AG048642, RF1AG054548, R01AG061022, and R21AG056952).

0424

EFFECT OF CHRONIC INTERMITTENT HYPOXIA ON GLOBAL CEREBRAL METABOLIC RATE OF OXYGEN CONSUMPTION IN RATS

Xu, J. Geng, E. Brake, L. Wiemken, A. Keenan, B. Kubin, L. Schwab, R.

University of Pennsylvania, Philadelphia, PA.

Introduction: Patients with obstructive sleep apnea (OSA) commonly exhibit grey and white matter loss, which may be related to hypoxic damage in the brain during sleep. Our preliminary data demonstrated lower values of cerebral metabolic rate of oxygen (CMRO₂) consumption in apneics versus controls. As such, reduced CMRO₂ may be an important contributor to the neurologic consequences of OSA. Here we report a rodent model for chronic intermittent hypoxia (CIH) to quantify effects on CMRO₂ consumption. We hypothesized that increased severity of CIH results in decreased CMRO₂ levels.

Methods: Three groups of rats were subject to varying levels of hypoxia: sham (21% oxygen; n = 19), moderate (11% oxygen; n = 14), and severe (6% oxygen; n = 21). To deliver hypoxia, rats were exposed to three-minute cycles of oxygen between 21% and condition-specific nadir O₂ for 12 hours daily during their sleep cycle. CMRO₂ values were measured with MRI techniques, performed on anesthetized rats before and after 3 months exposure to CIH.

Results: Rats from the three hypoxia groups did not differ significantly in CMRO₂ values at baseline (0 months). After 3 months of exposure to hypoxic conditions, there was a trending difference (p=0.0726) in percent change from baseline between severely hypoxic (-35.3%) and sham (+12.3%) rats. Moderately hypoxic rats demonstrated an intermediate decrease from baseline after 3 months (-19.0%).

Conclusion: Our findings suggest that increased severity of intermittent hypoxia yields a dose-response decrease in brain oxygen consumption. Our data add to the growing body of evidence on the relationship between obstructive sleep apnea and hypoxic damage in the brain, suggesting that CMRO₂ levels may be an indicator of the neurologic consequences of OSA.

Support: Funded by NIH P01 HL094307

0425

SEVERITY OF INSOMNIA SYMPTOMS DIFFER BY COGNITIVE STATUS IN ADULTS WITH DOWN SYNDROME

Desai, S.¹ Chen, I. Y.² Doran, E.¹ Hom, C.² Nguyen, D. D.¹ Benca, R. M.² Lott, I. T.¹ Mander, B. A.²

¹Department of Pediatrics, Irvine, CA, ²Department of Psychiatry and Human Behavior, Irvine, CA.

Introduction: Sleep is disturbed in Down syndrome (DS), with sleep apnea and insomnia prevalent throughout life. Sleep disturbance increases dementia risk and is more prevalent in dementia in non-DS populations. However, relationships between sleep and clinical status in DS remains unclear. We examined informant-reported sleep in adults with DS, with or without a consensus diagnosis of dementia, and related the severity of sleep disturbances to measures of adaptive behavior.

Methods: Insomnia (selected from Children's Sleep Habits Questionnaire), daytime sleepiness (modified ESS), sleep apnea risk (modified STOP-BANG), and adaptive behavior (Vineland Adaptive Behavior Scales; VABS-3) questionnaires were collected from informants for 47 DS adults (52.1±6.6 years) enrolled in a Alzheimer's disease biomarker study. Participants' clinical statuses were categorized as cognitively unaffected (clinically significant impairment absent; n=38, 51.0±6.2 years), or as having definite dementia (clinically significant decline present; n=9, 56.6±6.4 years) using a standard consensus diagnosis procedure. Age was compared between groups using an independent samples t-test. ANCOVA was used to compare insomnia, daytime sleepiness, sleep apnea risk, and adaptive behavior measures across groups, while controlling for age. Partial correlation analyses examined associations between sleep measures and VABS-3 measures while controlling for clinical status. Results: Participants categorized as definite dementia were older (t=-2.381, p=0.022). ANCOVA determined that insomnia symptoms, but not daytime sleepiness or apnea risk, were more severe in definite dementia participants (F=5.567, p=0.023), even when controlling for age. VABS-3 subscale scores differed by clinical status (all save play and leisure scores p<0.017). Partial correlation analyses adjusting for clinical status indicated that insomnia symptom severity worsened with lower adaptive functioning (e.g., daily living skills—coping r=-0.41, p=0.007; socialization r=-0.33, p=0.024) regardless of clinical status.

Conclusion: These findings indicate that insomnia may be related to functional impairment and dementia in DS adults, and raises the possibility that insomnia treatments may influence dementia course and clinical symptomatology in DS. **Support:** NIH U01AG051412

0426

TIME RESTRICTED FEEDING CONSOLIDATES SLEEP IN THE BACHD MOUSE MODEL OF HUNTINGTON'S DISEASE

Nichols, I. S.¹ Chiem, E.¹ Tahara, Y.¹ Anderson, S.¹ Trotter, D.² Whittaker, D.¹ Ghiani, C.¹ Colwell, C.¹ Paul, K.¹ ¹University of California, Los Angeles, Los Angeles, CA, ²Morgan State University, Baltimore, MD.

Introduction: Disturbances in the daily sleep-wake cycle are common in individuals with neurodegenerative disorders. Huntington's disease (HD) is a genetic neurodegenerative disorder in which patients exhibit a variety of impairments that include, poor motor function, disrupted circadian rhythms, and sleep abnormalities such as difficulty initiating sleep at bedtime and more frequent nighttime arousals. In the BACHD mouse model time restricted feeding (TRF) has been successful at improving motor functions and circadian rhythms. The BACHD mouse model has a bacterial artificial chromosome that expresses the full-length human mutant huntingtin gene.

Methods: In order to determine the effects of TRF on sleep-wake architecture, EEG/EMG polysomnographic records were examined in mice between 3-4 months old bearing the BAC knock-in of a human genetic mutation of HD and WT litter mates, first during ad libitum (ad lib) feeding then during an 18 hour fasting protocol. TRF protocol consisted of 6 hours of food access limited between ZT15-ZT21 and 18 hours of fasting.

Results: A two-way ANOVA revealed that TRF significantly decreased the amount of total sleep (p=0.04) and NREM sleep (p=0.04) in the dark phase in both WT and BACHD mice. TRF did not significantly affect sleep in the light phase, however trends suggest that BACHD mice have more sleep in the light phase under TRF than ad lib.

Conclusion: This data suggests that TRF improves sleep by consolidating sleep to the light phase and wake to the dark phase. In conclusion, TRF may be a promising tool that can improve the negative effects of neurodegenerative diseases on sleep-wake processes.

Support: These experiments were supported by R01-NS078410 and UCLA start-up funds.

0427

BICUCULINE INCREASED THE CATAPLEXY IN THE MALE TAIEP RATS: AN ANIMAL MODEL OF NARCOLEPSY WITH AN INHERENT TUBULOPATHY

Cortes, C.¹ Eguibar, J. R.¹ Ibarra-Hernandez, J. M.¹ ¹Benemerita Universidad Autonoma de Puebla, Puebla, MEXICO, ²Benemerita Universidad Autonoma de Puebla, Puebla, MEXICO.

Introduction: Narcolepsy is a hypersomnolence that is characterized by sleep fragmentation, sleep paralysis, hypnagogic hallucinations and cataplexy that is characterized by atonia induced by strong emotions. The amygdala is the trigger for cataplexy through GABAergic mechanisms. *Taiep* is a myelin mutant with TUBB4A tubulopathy which showed spontaneous episodes of atonia or induced by manipulations from the tail or the thorax. EEG recordings during immobility episodes (IE's) had a cerebral cortex desynchronized associated to theta rhythm in the hippocampus. The aim of this sturdy was to analyze the effects of bicuculine administration on IE's and sleep-wake pattern on adult male *taiep* rats.

Methods: We used 6 *taiep* male rats at 9 months of age. The subjects (Ss) lived in individual acrylic cages with water and food pellets available *ad libitum*, under a 12:12 light-dark cycle (lights on at 0700), with controlled temperature and humidity recording room. All Ss were implanted to record EEG, EMG and EOG to characterize EI's. We evaluated a basal 24 h EEG recording and then after bicuculine i.p. administration of 0.5, 1 y 1.5 mg/Kg every 48h. We measured the number, mean duration and latency to the first IE's.

Results: The duration of IE's increased 527% with 1 mg/Kg and reach 700% with 1.5 mg/Kg of bicuculine (P<0.01) with respect to saline-treated control group. Importantly, the frequency of IEs did not differ among the groups and did not affect the number of awake, slow wave or rapid eye movements sleep phases.

Conclusion: Bicuculine, a specific GABA antagonist, modify the duration of IES but not their frequency supporting a role of GABAergic mechanism on IE's. It is relevant because sodium oxybate, an indirect GABA agonist, reduced cataplexy and improved sleep quality on narcoleptic patients.

Support: CONACYT grants 243333 and 243247 to CC and JRE, respectively and from VIEP-BUAP 2019 to CA in Neuroendocrinología BUAP-CA-288.

0428

TAIEP RATS HAD NORMAL LEVELS OF OREXIN NEURONS IN THE LATERAL HYPOTHALAMUS BUT THEIR CATAPLEXY ATTACKS ARE SENSIBLE TO SPECIFIC OREXIN B AGONIST

Eguibar, J. R.¹ Cortes, C.¹ Espinoza, K.¹ De Ovando, C. I.¹ ¹Benemerita Universidad Autonoma de Puebla, Puebla, MEXICO, ²Benemerita Universidad Autonoma de Puebla, Puebla, MEXICO.

Introduction: Narcolepsy is characterized by sleep fragmentation, sleep paralysis, hypnagogic hallucinations and cataplexy. The cataplexy is a sudden loss of muscle tone triggered by emotions in humans, as well as in animal models. It is stablished that most of the patients had a significant decrease of orexin neurons in the lateral hypothalamus. *Taiep* rats had a mutation in tubulin TUBB4A and suffer immobility episodes (IE's) that had a desynchronized activity in the cortex associated with theta rhythm in the hippocampus similar to narcolepsy patients. The aim of this study was to analyze the effects of central administration of an orexin B agonist and determination by immunohistochemistry of the number of orexin neurons on adult male *taiep* rats.

Methods: We used 14 male *taiep* rats of 9 months of age. The subjects (Ss) lived 3-4 in acrylic cages with water and food pellets ad libitum, under a 12:12 light-dark cycle (lights on at 0700), whit-controlled temperature and humidity in the recording room. All Ss were implanted to record EEG, EMG and EOG to characterize immobility episodes (IE's) in a control 8 h recording and after i.c.v. administration of [Ala 11, D-Leu 15]-orexin B with 1, 3 and 10 nmol/1 μ L. We measured the number, mean duration and latency to the first IE's.

Results: The administration of [Ala 11, D-Leu 15]-orexin B significantly reduced the number of IE's (P<0.01), from 4.28 ± 1.5 IE's to just 0.25 ± 0.17 with 10 nmol/1 µL dose, but did not change the amount of awakening, slow wave or rapid eye movement sleep. Importantly, the number or orexins neurons were similar between *taiep* and Sprague-Dawley rats.

Conclusion: The myelin mutant *taiep* rats is a model of narcolepsy with cataplexy with normal number of orexin neurons in the lateral hypothalamus. Additionally, a specific orexin B agonist reduced IE's without any effect the sleep pattern. This could be useful for the design of new treatments.

Support: CONACYT grants 243333 and 243247 to CC and JRE, respectively. Grants from VIEP-BUAP 2019 and CA in Neuroendocrinología BUAP-CA-288.

0429

CHRONIC SLEEP DISTURBANCE CAUSES COGNITIVE IMPAIRMENT AND ALZHEIMER'S DISEASE-LIKE NEUROPATHOLOGY

Le, W.

Sichuan Academy of Medical Sciences · Sichuan Provincial Hospital, Chengdu, CHINA.

Introduction: Sleep disturbance is among the most common clinical problem and possesses a significant concern for the geriatric population. Recently, increasing evidence has indicated that disturbed sleep may not only affect neuropsychological functions, but also contribute to the cognitive impairment and, therefore, significantly increase dementia risk.

Methods: In the present study, we examined the potential impacts of chronic sleep deprivation (SD) on learning-memory and AD-related pathologies in $A\beta PP^{swe}/PS1^{\Delta E9}$ transgenic (TG) mice and their wild-type (WT) littermates.

Results: Our results indicated that mice (both TG and WT) exposed to 2-month SD showed an altered amyloid- β protein precursor processing, elevated level of phosphorylated tau protein, and impaired cognitive performance as compared to non-sleep deprivation (NSD) controls. Moreover, the SD-treated TG mice exhibited more amyloid- β 1-42 production and developed more senile plaques in the cortex and hippocampus than NSD-treated TG mice. In addition, SD caused a striking neuronal mitochondrial damage, caspase cascade activation, and neuronal apoptosis in the hippocampus of both TG and WT mice. More importantly, all these behavioral, neuropathological, and biochemical changes induced by chronic SD were long lasting and were irreversible during a 3-month normal housing condition.

Conclusion: Collectively, these results indicate that chronic SD impairs learning and memory, exacerbates AD pathologies, and aggravates the mitochondria-mediated neuronal apoptosis in a long-lasting manner.

Support: Our findings provide important experimental evidence to prove that chronic sleep disturbance is a risk factor for AD.

0430

META-ANALYSIS ON THE EFFECTS OF CAFFEINE ON NEURODEGENERATIVE COGNITIVE DECLINE

Taylor, E. Killgore, W. D.

University of Arizona, Tucson, AZ.

Introduction: Mild cognitive impairment (MCI), Alzheimer's disease(AD), and dementia are common forms of neurodegenerative cognitive decline in aging populations. Alertness, attention, and sleep patterns are often impaired in dementia and MCI and can affect ongoing cognition. Given the current lack of treatment options, it is important to identify protective factors. Caffeine is a commonly consumed substance which has been demonstrated in previous observational studies to have a protective effect on the onset of MCI and the progression of MCI to AD.

Methods: A meta-analysis of longitudinal prospective cohort studies published up to December 2017 was conducted comparing highest vs lowest reported category of caffeine consumption on neurodegenerative outcomes. Three databases were searched including PubMed, EMBASE, and Web of Science. Two investigators independently extracted data and assessed study quality. 13 studies were selected including 94880 participants. The effect size was reported as RRs with ORs and HRs treated as approximations of the RRs.

Results: A meta-analysis conducted using random effects showed a pooled RR of .84, 95% CI (0.75, 0.93) indicating a moderate protective effect in higher levels of caffeine consumption compared to lower levels. By outcome, AD had a RR of 1.14 with 95% CI (0.69, 1.90); dementia had a RR of 0.81 (0.72, 0.92); cognitive decline had a RR of 0.81 (0.55, 1.18); and MCI had a RR of 0.78 (0.65, 0.93). Conclusion: Overall this meta-analysis suggests that compared with the lowest category, the highest caffeine intake category is inversely related to the incidence of age-related cognitive disorders, with this relationship being most apparent for dementia and MCI. Given that caffeine is well accepted and consumed widely in a variety of forms, caffeine in moderate doses, may prove beneficial in sustaining cognitive functioning. Further work will examine the hypothesis that increased alertness and attention with caffeine may sustain cognition through use dependent plasticity or circadian modulation.

Support: None

TWO-WAY COMMUNICATION BETWEEN DREAMERS AND EXPERIMENTERS

Konkoly, K. R. Paller, K. A.

Northwestern University Psychology Department, Evanston, IL.

Introduction: Dreams are emblematic of human sleep, but they have yet to be adequately explained. In part, this is due to the limited options available for peering into dream experiences. Mapping neural measures onto dreams is problematic when those dreams are recounted after waking. Retrospective dream reports are subject to distortion and rapid forgetting.

Methods: Here, we describe a method to overcome these obstacles through two-way communication between dreamers and experimenters. To demonstrate proof-of-concept, we presented softly spoken math problems to participants during lucid REM sleep, and they provided answers using covert physiological signals such as eye movements. We confirmed REM sleep using standard polysomnographic methods.

Results: Thus far, 3 out of 8 participants who had lucid dreams correctly answered problems during REM sleep.

Conclusion: Results document that sleeping individuals can have sufficient abilities for veridical perceptual analysis, maintaining information, computing simple answers using working memory, and expressing volitional replies. Dreamers can thus be capable of interacting and exchanging information with other individuals. In this way, the mental content experienced by the dreamer can be interrogated to characterize the phenomenological experiences and cognitive abilities of dreaming.

Support: Mind Science Foundation, National Science Foundation

0432

A DEEP LEARNING APPROACH FOR AUTOMATED SLEEP-WAKE SCORING IN PRE-CLINICAL ANIMAL MODELS

Svetnik, V. Wang, T. Xu, Y. Hansen, B. J. Fox, S. V. Merck & Co., Inc., Kenilworth, NJ.

Introduction: Experimental investigation of sleep-wake dynamics in animals is an important part of pharmaceutical development. It typically involves recording of electroencephalogram, electromyogram, locomotor activity, and electrooculogram. Visual identification, or scoring, of the sleep-wake states from these recordings is time-consuming. We sought to develop software for automated sleep-wake scoring capable of processing large databases of multichannel signal recordings in a range of animal species.

Methods: We used a large historical database of signal recordings and scores in non-human primates, dogs, mice, and rats, to develop a deep Convolutional Neural Network (CNN) classification algorithm for automatically scoring sleep-wake states. We compared the performance of the CNN algorithm with that of a widely used Machine Learning algorithm, Random Forest (RF).

Results: In non-human primates and dogs, CNN accuracy in sleep-wake scoring of data was significantly higher than RF accuracy: 0.75 versus 0.66 for non-human primates and 0.73 versus 0.64 for dogs. In rodents, the difference between CNN and RF was smaller: 0.83 versus 0.81 for mice and 0.78 versus 0.77 for rats. The variability of CNN accuracy was lower than that of RF for non-human primates, dogs and mice, but similar for rats.

Conclusion: We recommend use of CNN for sleep-wake scoring in non-human primates and dogs, and RF for sleep-wake scoring in rodents. **Support:** Merck Sharp & Dohme Corp., a subsidiary of Merck & Co., Inc., Kenilworth, NJ, USA

0433

TARGETING LIGHT SENSITIVITY PARAMETERS TO OPTIMIZE CIRCADIAN PHASE PREDICTIONS

Stone, J. E. McGlashan, E. M. Cain, S. W. Phillips, A. J. Turner Institute for Brain and Mental Health, School of Psychological Sciences, Monash University, Melbourne, AUSTRALIA.

Introduction: Existing models of the human circadian clock accurately predict phase at group-level but not at individual-level. Interindividual variability in light sensitivity is not currently accounted for in these models and may be a practical approach to improving individual-level predictions. Using the gold-standard predictive model, we (i) identified whether varying light sensitivity parameters produces meaningful changes in predicted phase in field conditions; and (ii) tested whether optimizing parameters can significantly improve accuracy of circadian phase prediction.

Methods: Healthy participants (n=12, 7 women, aged 18-26) underwent continuous light and activity monitoring for 3 weeks (Actiwatch Spectrum). Salivary dim light melatonin onset (DLMO) was measured each week. A model of the human circadian clock and its response to light was used to predict the three weekly DLMO times using the individual's light data. A sensitivity analysis was performed varying three model parameters within physiological ranges: (i) amplitude of the light response [p]; (ii) advance vs. delay bias of the light response [K]; and (iii) intrinsic circadian period [tau]. These parameters were then fitted using least squares estimation to obtain optimal predictions of DLMO for each individual. Accuracy was compared between optimized parameters and default parameters.

Results: The default model predicted DLMO with mean absolute error of 1.02h. Sensitivity analysis showed the average range of variation in predicted DLMOs across participants was 0.65h for p, 4.28h for K and 3.26h for tau. Fitting parameters independently, we found mean absolute error of 0.85h for p, 0.71h for K and 0.75h for tau. Fitting p and K together reduced mean absolute error to 0.57h.

Conclusion: Light sensitivity parameters capture similar or greater variability in phase as intrinsic circadian period, indicating they are a viable option for individualising circadian phase predictions. Future prospective work is needed using measures of light sensitivity to validate this approach. **Support:** N/A

0434

CLINICAL APPLICATION OF COMPUTER AIDED CLOUD SLEEP SCORING SYSTEM

Lin, W.¹ Kuo, P.² Liu, M.² Li, C.² Lin, C.^{1,3} Liang, S.^{2,4} ¹Sleep Medicine Center, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University (NCKU), Tainan, TAIWAN, ²Department of Computer Science and Information Engineering, National Cheng Kung University (NCKU), Tainan, TAIWAN, ³Department of Otolaryngology, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University (NCKU), Tainan, TAIWAN, ⁴MOST Artificial Intelligence Biomedical Research Center, National Cheng Kung University (NCKU), Tainan, TAIWAN.

Introduction: According to a survey by World Sleep Society, 45% of the population suffered from sleep disorders. The best way to diagnose these patients is to use Polysomnography (PSG), recording

their physiological signals throughout the night. Mostly, sleep technologists manually score sleep stages. Manual scoring is quite subjective and time-consuming. Although the technologist's judgments are based on scoring standards of the American Academy of Sleep Medicine, fine-tuning scoring results because of different considerations in different sleep centers may be happened. In order to assess the consistency of scoring standards in sleep technologists, we tried to establish a cloud sleep scoring system and evaluate its feasibility in 4 sleep centers in southern Taiwan.

Methods: We constructed a computer-aided cloud sleep scoring system. Each sleep technologist could score the same test data of PSG online without being restricted by places and hardware equipment. After comparing scoring results of all participants, the scoring system could provide the following reports, including an overall agreement, agreement of each sleep stage and each sleep index. Besides, multi-person scoring results of each epoch with displaying physiological signals were analyzed.

Results: Seven sleep technologists from 4 hospitals in Tainan, Taiwan joined this study. Standard deviations (SDs) of each sleep stage included 2.64 in Wake stage, 6.90 in N1, 8.31 in N2, 6.87 in N3, 1.38 in REM, respectively. SDs of sleep indexes were 2.64 in sleep efficiency, 2.14 in sleep onset time, 8.35 in wake after sleep onset time, 10.03 in total sleep time, individually. The overall agreement was 89.6%. The satisfaction of this scoring system operation was 85.7%.

Conclusion: With the cloud sleep scoring system assistance, it was feasible to evaluate the scoring consistency among sleep technologists in different sleep centers.

Support: This work is supported by the Ministry of Science and Technology, Taiwan. (MOST 108-2634-F-006-012)

0435

AUTOSCORING OF SLEEP AND ASSOCIATED EVENTS VERSUS A REFERENCE SCORER COMPETING WITH THREE ADDITIONAL MANUAL SCORINGS: A CLINICAL VALIDATION STUDY

Anderer, P.¹ Ross, M.¹ Cerny, A.¹ Moreau, A.² ¹Philips Austria GmbH, Vienna, AUSTRIA, ²Philips Neuro, Eugene, OR.

Introduction: Manual scoring of polysomnographic (PSG) data is a time-consuming and tedious process with noticeable interrater variability. Autoscoring may overcome these limitations if it delivers valid results. The goal of this study was to validate a comprehensive autoscoring system in a clinically representative population.

Methods: The validation data consisted of 97 PSGs in patients with sleep-related breathing disorder, scored manually by a reference scorer and three further experts. The Somnolyzer autoscoring system combined pattern recognition for events such as spindles, k-complexes, slow-waves, eye-movements, apneas, hypopneas, desaturations and leg movements with an artificial intelligence classifier consisting of a bidirectional long short-term memory recurrent neural network (RNN) architecture. Intra-class correlation coefficients (ICC) for absolute agreement were determined for the commonly used metrics in sleep medicine to compare both, the three human expert scorings and the autoscoring versus the reference scoring.

Results: ICC coefficients for autoscoring and the three manual scorings versus the reference scoring were for sleep efficiency: .95, .83, .91, .93; N1(%): .71, .44, .39, .56; N2(%): .87, .63, .55, .45; N3(%): .80, .62, .44, .32; R(%): .92, .89, .91, .88; arousal index: .88,

.81, .22, .78; PLMI: .97, .88, .86, .91; AHI: .91, .89, .87, .78; OA: .94, .89, .91, .90; CA: .96, .96, .96, .82; MA: .93, .77, .43, .41. Thus, the ICCs between autoscoring and the reference scoring were equal or higher than the ICCs between any of the three manual scorings and the reference scoring for all endpoints.

Conclusion: Autoscoring of PSGs based on artificial intelligence outperformed even the best of three human expert scorers. Since the autoscoring performs pattern recognition in real-time, the final autoscoring results are available immediately after the end of the recording.

Support: All authors are employees of Philips

0436

DEEP LEARNING FOR SCORING SLEEP BASED ON SIGNALS AVAILABLE IN HOME SLEEP APNEA TEST STUDIES: CARDIORESPIRATORY SLEEP STAGING *Anderer, P.¹ Ross, M.¹ Cerny, A.¹ Radha, M.^{2,3} Fonseca, P.^{2,3}* ¹Philips Austria GmbH, Vienna, AUSTRIA, ²Royal Philips,

Eindhoven, NETHERLANDS, ³Eindhoven University of Technology, Eindhoven, NETHERLANDS.

Introduction: Typically, neurological signals are not recorded in home sleep apnea testing (HSAT) and thus standard sleep scoring is not applicable. The respiratory event index is calculated using total recording time rather than total sleep time (TST) resulting in a risk of underestimating sleep apnea severity. The objective of the study was to evaluate if artificial intelligence approaches can provide sleep scoring based on cardiorespiratory signals (CReSS) with reasonable accuracy.

Methods: Supervised deep learning for scoring sleep was trained with 472 and tested in 116 polysomnographies (PSG), scored independently by two experts and by a consensus scorer. The resulting bidirectional long short-term memory recurrent neural network (RNN) was integrated in the Somnolyzer system and validated in 97 PSGs of patients with obstructive sleep apnea (OSA) which had been scored independently by four human experts. Cohen's kappa agreement for four stages (W, L: N1+N2, D: N3, R) was determined as compared to a consensus scoring.

Results: Epoch-by-epoch comparison between CReSS autoscoring and manual consensus scoring resulted in Cohen's kappa of 0.68 (W: 0.74, L: 0.63, D: 0.54, R: 0.79). The intra-class correlation coefficient (ICC) between TST derived from CReSS and from neurological scoring was 0.86 (95%-CI: 0.79-0.90), while the ICC between subjective TST from sleep questionnaire and the objective TST was only 0.65 (95%-CI: 0.45-0.77). REM-related OSA had a prevalence of 16% and was detected with an accuracy of 95%.

Conclusion: With a kappa of 0.68, the cardiorespiratory-based RNN classifier is far above previously published values and reflects a substantial agreement with the manual consensus scoring in patients with sleep-disordered breathing. Thus, applying CReSS allows a more accurate determination of the OSA-severity and even a detection of REM related OSA in HSAT studies. **Support:** All authors are employees of Philips

0437

CHARACTERIZING THE IMPACT OF EEG REFERENCING ON SLEEP SPINDLE AND SLOW OSCILLATION ANALYSES

He, M.¹ Prerau, M. J.² Dimitrov, T. S.²

¹Health Sciences and Technology, Massachusetts Institute of Technology, Boston, MA, ²Division of Sleep and Circadian Disorders Brigham and Women's Hospital, Boston, MA.

Introduction: The impact of EEG referencing on sleep oscillations, such as spindles and slow oscillations, is largely overlooked across studies. While it is recognized that a topographic head plot of EEG activity does not reflect the true location of the underlying cortical activity, spatial distributions, as well as spectral properties and morphology of EEG oscillations can change dramatically as a function of referencing scheme. It is therefore vital to understand the impact of referencing when drawing inferences about the nature of EEG sleep oscillations. In this study, we use MRI structural data to construct subject-specific forward models of EEG signals. Using these models, we can simulate cortical activity and observe its true representation on the scalp. In particular, we simulate spindles and slow wave oscillations and examine how referencing affects topography, spectral power, and phase of oscillations.

Methods: High-density EEG (Brain Vision, 64-channel) polysomnography was performed on 9 healthy young subjects. 3T structural MRI scans were acquired and forward models were built in MNE-Python using 3-shell Boundary Element Models (BEM) based on individual anatomical details processed with Freesurfer. Simulations of various sleep spindle and slow oscillation dynamics were projected to the sensor space. Different referencing schemes (common average, Laplacian, linked-mastoid) were then applied to the experimental and simulated data and analyzed for effects on time-frequency characteristics of sleep oscillations.

Results: Analyses of experimental data showed distinct referencebased differences in topographical distribution of spectral power and phase of oscillations. Simulated data revealed many scenarios in which the spatial distribution of activity the EEG sensor space poorly represented the true location of the underlying source activity. Moreover, there were alterations to the spatial spread and envelope form of sleep spindle events under different referencing schemes despite from identical source activities.

Conclusion: This study shows that spindle and slow oscillation activity is highly variable across referencing schemes and that EEG topographical plots on the scalp may poorly represent cortical activity locations. It is thus vital to consider the choice of referencing when quantifying characteristics of sleep EEG oscillations. **Support:** This work was supported by R01 NS-096177.

0438

AUTOMATIC NIGHTTIME AGITATION AND SLEEP DISRUPTION DETECTION USING A WEARABLE ANKLE DEVICE AND MACHINE LEARNING

Kumar, R.¹ Feltch, C.² Richards, K.³ Morrison, J.³ Rangel, A.³ Janney, R.³ Shayesteh, S.³ Allen, R.⁴ Banerjee, N.¹

¹University of Maryland, Baltimore County, Catonsville, MD, ²Tanzen Medical, Inc., Baltimore, MD, ³University of Texas, Austin, Austin, TX, ⁴Johns Hopkins University, Baltimore, MD.

Introduction: Nighttime agitation behavior such as wandering and restlessness during awake and sleep in people with Alzheimer's disease (AD) is expensive to manage and adversely affects sleep. Nighttime agitation is mostly noted by subjective caregiver reports. An automated process for this assessment would improve clinical management. Here we report on the RestEaZeTM system that uses an ankle band and machine learning to automatically classify sleep status and nighttime agitation behaviors in older adults with AD. **Methods:** We collected data on 7 adults (mean: 81 years, SD: 10.6) with AD. They wore the RestEaZeTM ankle band with a 3-axis accelerometer, a 3-axis gyroscope, and three textile capacitive sensors. A trained Research Assistant (RA) continuously observed for wandering, restlessness, wake, and sleep between 5pm and 7am using

the Cohen Mansfield Agitation Inventory (CMAI). We merged, and band-pass filtered the data and divided it into 10-second non-overlapping windows. CMAI labels and time-series features (scaled using StandardScaler) extracted from the RestEaZeTM data were used to train a Random Forest binary classifier. The significant features were extracted based on the impact on the p-value for the classifier. We used the Synthetic Minority Oversampling Technique (SMOTE) to balance the dataset and performed 5-fold cross-validation with a 67-33 train-test split.

Results: We report the sensitivity, specificity, accuracy, and Areaunder-the Curve (AUC) for the ROC curve for the classifiers: (1) Sleep/ Awake: sensitivity=0.95, specificity=0.87, accuracy=0.92, AUC=0.97; (2) Wandering/Non-Wandering: sensitivity=0.85, specificity=0.99, accuracy=0.98, AUC=0.99; and (3) Restless/Non-Restless: sensitivity=0.84, specificity=0.84, accuracy=0.84, AUC=0.92. The significant features were related to the intensity of movements.

Conclusion: Our preliminary results show the feasibility of using RestEaZeTM for quantitatively measuring nighttime agitation. These can provide clinically useful objective measures of agitation that can be automatically transmitted to clinical or research records with minimal staff time requirements.

Support: The authors acknowledge the funding support from the National Institute on Aging under award R01AG051588 and Arbor Pharmaceuticals for support for Horizant and the matching placebo.

0439

NONLINEAR DYNAMICS FORECASTING FOR PERSONALIZE PROGNOSIS OF OBSTRUCTIVE SLEEP APNEA ONSETS

Le, T.

North Dakota State University, FARGO, ND.

Introduction: The emphasis on disease prevention, early detection, and preventive treatments will revolutionize the way sleep clinicians evaluate their patients. Obstructive Sleep Apnea (OSA) is one of the most prevalent sleep disorders with approximately 100 millions patients been diagnosed worldwide. The effectiveness of sleep disorder therapies can be enhanced by providing personalized and real-time prediction of OSA episode onsets. Previous attempts at OSA prediction are limited to capturing the nonlinear, nonstationary dynamics of the underlying physiological processes. Methods: This paper reports an investigation into heart rate dynamics aiming to predict in real time the onsets of OSA episode before the clinical symptoms appear. The method includes (a) a representation of a transition state space network to characterize dynamic transition of apneic states (b) a Dirichlet-Process Mixture-Gaussian-Process prognostic method for estimating the distribution of the time estimate the remaining time until the onset of an impending OSA episode by considering the stochastic evolution of the normal states to an anomalous (apnea)

Results: The approach was tested using three datasets including (1) 20 records from 14 OSA subjects in benchmark ECG apnea databases (Physionet.org), (2) records of eight subjects from previous work. The average prediction accuracy (\mathbb{R}^2) is reported as 0.75%, with 87% of observations within the 95% confidence interval. Estimated risk indicators at 1 to 3 min till apnea onset are reported as 85.8 %, 80.2 %, and 75.5 %, respectively.

Conclusion: The present prognosis approach can be integrated with wearable devices to facilitate individualized treatments and timely prevention therapies.

Support: N/A

VALIDATION STUDY OF NEURAL NETWORK ALGORITHM FOR AUTOMATED SLEEP STAGE SCORING: STAGENET

Choi, J.¹ Moon, J.²

¹Department of Otorhinolaryngology-Head and Neck Surgery, Soonchunhyang University College of Medicine, Bucheon Hospital, Bucheon, KOREA, REPUBLIC OF, ²Department of Biostatistics, Clinical Trial Center, Soonchunhyang University College of Medicine, Bucheon Hospital, Bucheon, KOREA, REPUBLIC OF.

Introduction: Polysomnography (level 1) is an important test for evaluating sleep status or disorder. In general, measured raw data of polysomnography is manually scored by sleep expert. However, manual scoring is a time-consuming and labor-intensive work. The purpose of this study was to verify the accuracy of automated sleep stage scoring based on the neural network algorithm compared to the manual sleep stage scoring.

Methods: A total 604 polysomnography data set of subjects (Male: Female = 409: 195) aged 19 to 60 years were finally included in the study. The performance of proposed model was evaluated with kappa and bootstrapped point-estimated of median percent agreement with 95% percentile bootstrap confidence interval with R=1000. The proposed model is trained with 484 data set and validated with 48 data set. For test, 72 data set are randomly selected. **Results:** The proposed model showed good concordance rates in stage W (94%), N1 (83.9%), N2 (89%), N3 (92%) and R (93%) between automated neural network algorithm and manual scoring. The average kappa value was 0.85. In bootstrap method, high overall agreements of automated neural network algorithm were found in stage W (98%), N1 (94%), N2 (92%), N3 (99%), R (98%) and total (96%).

Conclusion: Automated sleep stage scoring using proposed model - StageNet may be a reliable method for sleep stage classification. **Support:**

0441

AN AUTOMATIC SLEEP SCORING SYSTEM BASED ON ENSEMBLE CONVOLUTIONAL NEURAL NETWORK AND SPECTROGRAM OF SLEEP PHYSIOLOGICAL SIGNAL

Kuo, C. Chen, G.

Feng Chia University, Taichung, TAIWAN.

Introduction: Manual sleep stage scoring is time consuming and subjective. Therefore, several studies focused on developing automated sleep scoring algorithms. The previously reported the automatic sleep scoring have been develop usually using small dataset, which less than 100 subjects. In this study, an automatic sleep scoring system based on ensemble convolutional neural network (ensemble-CNN) and spectrogram of sleep physiological signal was proposed and evaluated using a large dataset with sleep disorder.

Methods: The spectrograms were computed from each 30-s EEG and EOG of 994 subjects from PhysioNet 2018 challenge dataset, using the continuous wavelet transform, which were fed into an ensemble-CNN classification for training. The ensemble-CNN contained five pretrained models, ResNet-101, Inception-v4, DenseNet-201, Xception, and NASNet models, because these models' architectures are different which can learn different features from the spectrograms to obtain high accuracy. The probabilities of five models were averaged to decide the sleep stage for each spectrogram. After classifying sleep stage, a smoothing process

was used for sleep continuity. Moreover, the total 80% data from PhysioNet dataset were randomly assigned to the training set, and the remaining data were assigned to the testing set.

Results: To validate the robustness of the proposed system, the validation procedure was repeated five times. The performance measures were averaged over the five runs. The overall agreement and kappa coefficient of the proposed method are 82% and 0.73, respectively. The sensitivity of the sleep stages of Wake, N1, N2, N3, and REM are 90.0%, 48.6%, 84.9%, 84.2%, and 81.9%, respectively.

Conclusion: The performance of the proposed method was achieved expert level, and it was noted that the ensemble-CNN is a promising solution for automatic sleep stage scoring. This method can assist clinical staff in reducing the time required for sleep stage scoring in the future.

Support: This work was supported by the Ministry of Science and Technology, Taiwan. (MOST 106-2218-E-035-013-MY2, 108-2221-E-035-064, and 108-2634-F-006-012).

0442

EXPERIMENTING AUTOMATIC SLEEP ANALYSIS APPLICATION IN A CLINICAL CONTEXT

BOUCHEQUET, P.¹ LEGER, D.² LEBRUN, M.³ ELBAZ, M.⁴ ¹Université de Paris, Paris Descartes, Paris, FRANCE, ²Université de Paris, Paris Descartes, PARIS, FRANCE, ³Université de Paris- Paris Descartes, Paris, FRANCE, ⁴Université de Paris Paris Descartes, Paris, FRANCE.

Introduction: Multiplication of publications describing groundbreaking automatic sleep analysis processes and algorithms push for real-life experimentation in clinical context, outside of controlled research environments.

Methods: Various automatic sleep analysis processes from the literature were implemented and orchestrated in a streamlined workflow. Artificial Intelligence algorithms using regular statistical learning or deep learning were re-trained on our own data after repeating the ad-hoc pre-processing steps described in the corresponding articles. For this, we used polysomnographic records previously taped in our clinic, subject to adequate legal authorizations and agreements: 500 nights from single patients with various pathologies. Those trained models were then applied to newly recorded polysomnographies through a platform developed and hosted on premise. For each polysomnography, a standardized and automatized report were generated and transmitted to the clinician in charge of the analysis. This report contains algorithms outputs, including automatic staging and related statistics such as hypnodensity, quantitative electroencephalography (EEG) analysis, spindles detection and automatic diagnosis. Aggregated record statistics are displayed next to our database statistics for benchmarking purposes.

Results: For sleep staging, we not only reproduced the results of the selected literature but obtained better metrics: a 0.76 Kappa agreement vs 0.69 in the literature. This may be due to our larger training database or the quality of physiologic signals in our data. Clinicians showed interest in the automatic staging part of the analysis. They noticed algorithm errors are mostly focused on ambiguous epochs, just like visual scoring. However, they found help into automated output and explanatory variables (hypnodensity) to score those ambiguous epochs.

Conclusion: Automatic sleep analysis algorithms used as decision helping tools shows real potential and should be generalized, as long as underlying processes are published and understood by users and clinicians.

Support: Banque Publique d'Investissement.

INTRA WEEK SLEEP PATTERNS ANALYZED USING **CONSUMER SLEEP TRACKER DATA**

Gahan, L. Ruder, M. Raj, A. O'Mullane, B. Raymann, R. J. SleepScore Labs, Carlsbad, CA.

Introduction: Big data collected using consumer sleep technology can provide objectively measured insights on sleep behavior in the real-life environment. It has the advantage over self-report data of being less prone to bias. Here we used a non-contact bio-motion sensor to remotely capture objective sleep data. We analyzed 168432 nights of sleep data to test if differences between weekday versus weekend sleep behavior, known from self-report, would still hold using objective data in a large population.

Methods: Sleep data was acquired using the SleepScore Max remote sleep sensor and included 168432 nights (2730 users, mean age: 46.6 +/- 11.8 years, 33% female, all resident in the USA). Analysis was restricted to those of working age; adults between 20-65. Any sleep which ended from Monday to Friday was considered weekday sleep, and any ending on Saturday or Sunday as weekend sleep. Data records were inspected and cleaned before analyzing. Descriptive statistics and independent t-tests were used to analyze the data.

Results: Total Sleep Time, Time In Bed and Sleep Onset Latencies were longer during weekend (TST: + 20.6 mins, TIB: +22.9 mins, SOL: +1.1 min, all p <0.001), resulting in a slightly poorer Sleep Efficiency (-.016%, p<0.01) for weekend nights. Time to bed and final awakening were both delayed in weekends as compared to weekdays (Time to bed +30.0 mins, and final awakening +53.4 mins, both p<0.001).

Conclusion: This big data analysis confirms the earlier observed difference in sleep and sleep behavior between weekdays and weekends. This should be considered for optimizing (automated) sleep interventions, that may not normally take the weekend effect into consideration.

Support:

0444

EXAMINING SLEEP DURATION AND SOCIAL JETLAG FROM A POPULAR WEARABLE SLEEP TRACKER IN FRANCE AND CANADA

ELBAZ, M.¹ ROBBINS, R.² Bouchequet, P.³ Czeisler, C.² LEGER. D.⁵

¹APHP Hôtel Dieu Centre du Sommeil et de la Vigilance, Paris, FRANCE, ²Harvard Medical School, Boston, MA, ³Université de Paris Paris Descartes, Paris, FRANCE, ⁴Harvard Medical School, Boston, MA, ⁵Université de Paris Paris-Descartes, PARIS, FRANCE.

Introduction: Many population estimates of sleep duration and quality rely primarily on self-reported data. Passive and ubiquitous digital tracking and wearable devices may provide more accurate estimates of sleep duration and quality.

Our objective was to identify trends in sleep duration and social jetlag using data from a popular mobile sleep application (app) in France and Canada 'iSommeil.'

Methods: We examined sleep using 8,207 nights from iSommeil, a popular sleep-tracking app in France and Canada. In this analysis, we explored sleep data collected from this app from 2,126 users. We examine sleep parameters by sex and between week and weekend. Specifically, we explore social jetlag, as calculated by the midpoint of sleep during the weekend, subtracted from the midpoint of sleep during the week.

Results: Women represented 1,254 (59.7%) of the sample and men represented 857 (40.3%) of the sample. Among women, 16.4% of the sample averaged <6 hours of sleep; 51.9% averaged 6-7.99 hours of sleep, and 31.7% averaged >=8 hours of sleep. Among men, 17.4% averaged <6 hours of sleep; 58.4% averaged 6-7.99 hours of sleep, and 24.2% averaged >=8 hours of sleep. Social jetlag scores among all users averaged 31.4, yet the average for men was 27.1 while that for women was 36.4.

Conclusion: Our study of data from a popular sleep tracker in France and Canada showed that sleep duration of 6-7.99 hours was most observed among the majority of participants. Our results also showed that women had higher social jetlag scores than men. Future research may compare sleep measures obtained via wearable sleep trackers with validated research-grade measures of sleep. Support:

0445

A NOVEL ALGORITHM FOR THE ESTIMATION OF SLEEP STATES BASED ON BREATHING AND MOVEMENT

Dietz-Terjung, S.¹ Martin, A.² Schöbel, C.²

¹Departement of Telemedicine and Sleep Medicine, Ruhrlandklinik, University Medicine Essen, Essen, Germany, Essen, GERMANY, ²Departement of Telemedicine and Sleep Medicine, Ruhrlandklinik, University Medicine Essen, Essen, Germany, Ruhrlandklinik, GERMANY.

Introduction: We tested the diagnostic accuracy of the novel Nox BodySleep[™] algorithm (Nox Medical, Iceland) for the estimation of sleep states from polygraphy (PG) sleep recordings based on features extracted from actigraphy and respiratory inductance plethysmography (RIP) belts. The algorithm automatically classifies epochs into three states, Wake, REM sleep and NonREM sleep. Validation was performed against polysomnography (PSG) in a sleep laboratory collective including patients with sleep disordered breathing (SBAS) and sleep related movements disorders. Methods: Patients received PSG according to clinical routine. The recording was evaluated by the novel algorithm and the results were evaluated by descriptive statistics methods (IBM SPSS Statistics 25.0).

Results: We found a good Spearman correlation (r=0.8) and a bias of 11 minutes for the estimation of Total Sleep Time. Sleep Efficiency was also valued with a good Spearman correlation (r=0.7) and a bias of 1.6%. Wake phases were estimated with a F1 score of 0.64 while REM and Non-REM phases were evaluated with a F1 score of 0.73 and 0.82, respectively. Additionally, an overall accuracy of 0.8 and a Cohens kappa of 0.7 were found. Patients with sleep related movement disorders showed a slighly weaker correlation as patients with SBAS.

Conclusion: The algorithm shows a good diagnostic accuracy for the estimation of sleep states and significant sleep parameters. After validation on a larger patient collective, it could be used in the ambulatory and telemedical field to allow investigations comparable to the accuracy of a PSG. Support: No support.

0446

A NOVEL SYSTEM FOR ENABLING HIGH-DENSITY EEG **RECORDINGS IN A MOUSE**

Thankachan, S.¹ Gerashchenko, L.² Gerashchenko, D.¹

¹Harvard Medical School, West Roxbury, MA, ²Neurotargeting Systems Inc., Chestnut Hill, MA.

Introduction: Recent advances in micro-electromechanical system (MEMS) technology have promoted the development of microelectrode arrays (MEA) that allow high resolution recordings in neuroscience research. However, applying MEA in studies in freely moving mice remains very challenging due to the large number of electrical connections required in this type of studies. The use of commutators for a large number of connections is not practical, and headmounts/loggers placed on the animal head are too heavy for small animals such as mice. Therefore, there is a need for a better compact system for using MEA in mice. Herein, we designed such a system and successfully recorded high-density-EEG in freely moving mice.

Methods: We designed a system in which forty flexible ultrathin wires are connected to the headstage enclosed in a container held close to the mouse. The container also houses a logger and battery connected to the headstage. This recording system allows minimizing weighted pressure on the animal using a counterbalance, so that the animal can freely move in the cage.

Results: We tested the system using a signal generator and mouse EEG arrays (NeuroNexus). When potentials produced by the signal generator were recorded via the wires, recorded traces were indistinguishable from the traces that were recorded when the signal generator was connected directly to the logger. We then implanted mice with EEG electrode arrays under surgical anesthesia. The high-density EEG recordings were performed one and four weeks after the surgery. High-quality EEG signals were observed in all the channels of the 32-channel logger (SpikeGadgets) in freely moving mice.

Conclusion: We successfully developed and tested a novel system for enabling high-density EEG recordings in freely moving mice. We expect that this system will be useful for recording biopotentials from different types of MEA in freely moving mice.

Support: NIH 1R43OD023231 (LG), NIH 1RF1AG061774 (DG), and NIH 5R21NS106406 (DG)

0447

RESTNET: A ROBUST END-TO-END DEEP LEARNING APPROACH TO SLEEP STAGING OF SELF APPLIED SOMNOGRAPHY STUDIES

Jónsson, S. Æ. Gunnlaugsson, E. Finssonn, E.

Loftsdóttir, D. L. Ólafsdóttir, G. H. Helgadóttir, H. Ágústsson, J. S. Nox Research, Nox Medical, Reykjavík, ICELAND.

Introduction: Sleep stage classifications are of central importance when diagnosing various sleep-related diseases. Performing a full PSG recording can be time-consuming and expensive, and often requires an overnight stay at a sleep clinic. Furthermore, the manual sleep staging process is tedious and subject to scorer variability.

Here we present an end-to-end deep learning approach to robustly classify sleep stages from Self Applied Somnography (SAS) studies with frontal EEG and EOG signals. This setup allows patients to self-administer EEG and EOG leads in a home sleep study, which reduces cost and is more convenient for the patients. However, self-administration of the leads increases the risk of loose electrodes, which the algorithm must be robust to. The model structure was inspired by ResNet (He, Zhang, Ren, Sun, 2015), which has been highly successful in image recognition tasks. The ResTNet is comprised of the characteristic Residual blocks with an added Temporal component.

Methods: The ResTNet classifies sleep stages from the raw signals using convolutional neural network (CNN) layers, which avoids manual feature extraction, residual blocks, and a gated recurrent unit (GRU). This significantly reduces sleep stage prediction time and allows the model to learn more complex relations as the size of the training data increases.

The model was developed and validated on over 400 manually scored sleep studies using the novel SAS setup. In developing the model, we used data augmentation techniques to simulate loose electrodes and distorted signals to increase model robustness with regards to missing signals and low quality data.

Results: The study shows that applying the robust ResTNet model to SAS studies gives accuracy > 0.80 and F1-score > 0.80. It outperforms our previous model which used hand-crafted features and achieves similar performance to a human scorer.

Conclusion: The ResTNet is fast, gives accurate predictions, and is robust to loose electrodes. The end-to-end model furthermore promises better performance with more data. Combined with the simplicity of the SAS setup, it is an attractive option for large-scale sleep studies.

Support: This work was supported by the Icelandic Centre for Research RANNÍS (175256-0611).

0448

SCALED-UP SLEEP APNEA ENDOTYPING USING POLYSOMNOGRAPHY FOR CLINICAL USE

Finnsson, E.¹ Jónsson, S. Æ.¹ Ólafsdóttir, G. H.¹ Loftsdóttir, D. L.¹ Helgadóttir, H.¹ Ágústsson, J. S.¹ Wellman, D. A.² Sands, S. A.² ¹Nox Research, Nox Medical, Reykjavík, ICELAND, ²Division of Sleep and Circadian Disorders, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA.

Introduction: Sleep apnea is caused by several key endophenotypic traits namely pharyngeal collapsibility, poor muscle compensation, ventilatory instability (high loop gain), and arousability from sleep (low arousal threshold). Already, measures of these traits have shown promise for predicting outcomes of therapies (oral appliances, surgery, hypoglossal nerve stimulation, CPAP, or pharmaceuticals) and thus may be an integral part of future precision sleep medicine with treatments administered based on underlying pathophysiology. However, currently, the novel methods developed for endotyping from polysomnography are computationally expensive and can only be performed by the original authors or their collaborators due to the need for technological expertise. Here we present a cloud-based method for endotyping sleep apnea from polysomnography for use in the clinical arena.

Methods: For cloud-based use, we optimized the Phenotype Using Polysomnography ('PUP') method of Sands et.al. (2015-2018) by performing the following: Code was translated from MATLAB to Python; efficient methods were developed to detect breaths, calculate normalized ventilation (moving time-average), and model ventilatory drive (intended ventilation). The new implementation ('PUP.py') was validated by comparing the measured traits against the original values. **Results:** 38 manually scored clinical polysomnographic studies were endophenotyped using the two implementations. Results of the new implementation ('PUP.py') were strongly correlated with the original (p<10⁻⁶ for all): collapsibility and compensation (ventilation at eupneic drive 'Vpassive': r=0.98; ventilation at arousal threshold, r=0.97), loop gain (r=0.96), and arousal threshold (r=0.92).

Conclusion: We successfully implemented the original method by Sands et.al. to scale up sleep apnea endotyping and make it available to a broader audience.

Support: This work was supported by the Icelandic Centre for Research RANNÍS, the European Union's Horizon 2020 SME Instrument (733461), and the American Heart Association (15SDG25890059).

0449

CHARACTERIZING SPINDLE ACTIVITY AS A TIME-FREQUENCY PHENOMENON

Dimitrov, T. S.¹ He, M.² Prerau, M. J.¹

¹Brigham and Women's Hospital, Boston, MA, ²Health Sciences and Technology, Massachusetts Institute of Technology, Boston, MA.

Introduction: Spindles are currently defined clinically based on observed patterns in the EEG waveform trace, with automated methods seeking to replicate visual scoring by experts. Recent work suggests that sleep spindles may be more readily observed as time-frequency peaks in the EEG spectrogram. This study compares spectral peaks in the multitaper spectrogram to expert and automatic detection scoring, characterizes the variability of spindles across a night, and investigates topographical and temporal clustering of spindles within individual EEG records.

Methods: We compared spectral peaks, expert scoring, and automatic detection in two datasets (DREAMS, and a high-density control study). Peaks were identified using multitaper spectral estimation and the peak prominence of the normalized power spectrum for each channel. Spatiotemporal variability analysis was performed using cluster and pattern recognition algorithms including penalized sorting of channel activation order, 2D-cross correlation, PCA and UMAP cluster analysis, and the seqNMF method.

Results: Spectral peaks were shown to be highly robust to and easily differentiated from broadband noise, occuring at rates (10-16 per min) far exceeding spindle rates reported in literature (~2.5 per min). Expert scoring and automated scoring failed to capture clear spectral peaks in the time-frequency domain, indicating an underreporting of the phenomenology. No apparent clustering or patterns of sleep spindle-like activity was observed using the proposed methods, suggesting high variability of spatiotemporal evolution of spindles.

Conclusion: These results suggest that the difficulty of time-domain visual scoring of spindles causes an artificially low estimate of the underlying phenomenology, which is mirrored in the assumptions implicit in the thresholds of automated scorers. This work shows that spindles are highly variable in their spatiotemporal evolution, suggesting that there is no optimal single electrode for analysis and casting doubt on the presence of a single cortical generation mechanism. We must therefore revisit the concept of the spindle using the time-frequency domain to more robustly characterize underlying phenomenology.

Support: National Institute Of Neurological Disorders And Stroke Grant R01 NS-096177

0450

SCREENING METHOD FOR OBSTRUCTIVE SLEEP APNEA SYNDROME BY PATIENTS' EXHALANT WITH TD-GC-MS SYSTEM

Yao, D.¹ Chieng, L.¹ Chiang, R.^{2,3,4}

¹National Tsing Hua University, Hsinchu, TAIWAN, ²China Medical University, Taichung, TAIWAN, ³International Sleep Science Technology Association (ISSTA), Berlin, GERMANY, ⁴Innovative Medical and Health Technology Center (IMHTC) Asia-Pacific Branch, Taipei, TAIWAN. **Introduction:** Human exhaled breath test is getting more important for non-invasive health monitoring and detecting method nowadays. The diagnosis of obstructive sleep apnea syndrome (OSAS) is often difficult to be confirmed from the daytime presentation and usually need the overnight polysomnography. The methods for OSAS screening are therefore potentials for the clinical practice in the near future.

Methods: In this research, a method of thermal desorption (TD) tendon with gas chromatography-mass spectrometry (GC-MS) system has been developed for screeing of OSAS patients. We detected the volatile organic compounds (VOCs) from the special designed experimental bags which collected exhaled gas. Then we compared the VOCs from normal control and OSAS group in order to find out the biomarkers which could be used to screen OSAS patients. Furthermore, the Reliable Number(N) was used to see how often the VOC identified in all the experients in OSAS group and was defined as the times of a single VOC identified devided by the times of total experiment in a single OSAS patient.

Results: While the reliable number been set as ≥50%, we found 8 VOC markers, including Pentane and Cyclopentyl acetylene, appeared more often in OSAS patients. When we raise N to ≥70%, we have only 3 markers remaining.

Conclusion: Based on this result, we utilize the artificial intelligence method, deep learning, to figure out whether the peak intensity of different biomarkers are related to the severity of OSAS. **Support:** Thanks for Da-Jeng Yao Lab's support

0451

FULLY AUTOMATIC DETECTION OF SLEEP DISORDERED BREATHING EVENTS

Thybo, J.¹ Olesen, A. N.¹ Olsen, M.¹ Leary, E.¹ Arnal, P.² Sørensen, H. B.³ Jennum, P.⁴ Mignot, E.¹ ¹Stanford, Palo Alto, CA, ²Dreem, Paris, FRANCE, ³DTU, Lyngby, DENMARK, ⁴Rigshospitalet, Glostrup, DENMARK.

Introduction: Evaluation of sleep apnea involves manual annotation of Polysomnography (PSG) file, a time-consuming process subject to interscorer variations. The DOSED algorithm has been shown to be helpful in detecting Central Sleep Apnea (CSA), Obstructive Sleep Apnea (OSA), and Hypopnea when merged into a single event type. This work uses a modified version of DOSED capable of detecting each event type separately.

Methods: The network consists of 3 blocks of 1D convolutional layers followed by 6 blocks of 2D convolutional layers. The network has 2 classification layers, one determines the probability of each class, and the other determines the start and duration time of the event with the highest probability. Four channels from nasal and mouth airflow and position of abdomen and thorax are used as input to the model. The model was trained using 2800 PSG from 4 different cohorts (MESA, MROS, SSC, WSC) and tested on 70 PSG, which have been scored by six technicians (Stanford, U Penn, St Louis).

Results: On an event by event basis, model F1-scores versus a weighted consensus score based on 6 technicians were 0.60 for OSA, 0.43 for CSA, and 0.34 for Hypopnea. Average F1-scores for the 6 technicians were 0.48 (std 0.04) for OSA, 0.29 (std 0.145) for CSA, and 0.54 (std 0.183) for Hypopnea, indicating that the model functions better on an event-by-event basis than an average technician. Correlations between indices/hr for central apnea, obstructive apnea, and hypopnea indicate excellent correlations for apneas, but poor correlation for hypopnea. We are now adding the snoring channel to explore if predictions can be improved.

Conclusion: The result shows that deep learning-based models can detect respiratory events with an accuracy similar to technicians. The poor agreement between technicians from different universities indicates that we need better definitions of hypopnea. **Support:** .

0452

MORLET WAVELET TRANSFORMS AND NEURAL

NETWORKS IN POLYSOMNOGRAPHY ANALYSIS

Stretch, R. Zeidler, M.

University of California Los Angeles, Los Angeles, CA.

Introduction: Manually scoring polysomnograms is both timeconsuming and labor-intensive. It also increases variability in care. Generating features for use as components within larger models is an important part of building highly accurate auto-scoring systems. In this study, we examined the use of time-frequency data representations in combination with a convolutional neural networks (CNN).

Methods: We used just six (6) pre-scored polysomnograms from the MrOS dataset in this analysis. Only one electroencephalography (EEG) and one electrooculography (EOG) channel were extracted from each polysomnogram and split into 30 second epochs. Visual representations of each epoch in the timefrequency domain were generated using Morlet wavelets, then divided into training and validation sets in a 4:5 distribution. We then re-trained a ResNet-50 CNN using transfer learning to classify sleep stage based on the time-frequency representations. Results: A total of 4971 epochs were generated. Of those, 1242 epochs formed the validation set. Performance was high for identifying Stage W with an accuracy of 94.2% (295/313 epochs). However, performance for other stages was considerably lower. Stage N3 was predicted correctly in 68.0% of cases (138/203 epochs), although in 60/75 cases of misclassification the predicted class was Stage N2. Similarly, Stage N2 was predicted correctly in 62.0% of cases (183/295 epochs), and in 63/112 cases of misclassification the predicted class was Stage N3. Accuracy for Stage REM was 64.9%. Stage N1 prediction was poor (22.0% accuracy), likely due to insufficient representation in the sample (< 10% of epochs).

Conclusion: This exploratory analysis of the use of time-frequency representations in conjunction with a CNN demonstrates some promise, especially with respect to prediction of Stage W using this technique. Inclusion of additional data channels and larger sample size would likely improve accuracy.

Support: RS - ASPIRE Fellowship (sponsored by the American Thoracic Association).

LONGITUDINAL RELATIONSHIP BETWEEN INSOMNIA AND WORK PRODUCTIVITY IN JAPANESE CITY GOVERNMENT EMPLOYEES

Kadotani, H.¹ Ito, K.² Matsuda, A.¹ Nishikawa, K.³ Sumi, Y.⁴ Matsuo, M.⁴

¹Shiga University of Medical Science, Otsu, JAPAN, ²Department of Anesthesiology, Shiga University of Medical Science, Otsu, JAPAN, ³Shiga CBT center, Hikone, JAPAN, ⁴Department of Psychiatry, Shiga University of Medical Science, Otsu, JAPAN.

Introduction: "Presenteeism" refers to the decrease in productivity in employees who are present but not functioning at full capacity due to illness or other medical conditions. It is reported that the cost of presenteeism to businesses is 10 times higher than absenteeism (away from work due to illness or disability). Relative presenteeism is a ratio of actual performance to the performance of most workers at the same job. We analyzed effects of insomnia and depression two years before on presenteeism in a Japanese working population.

Methods: Questionnaire survey was conducted as a part of a cohort study named "Night in Japan Home Sleep Monitoring Study (NinJaSleep Study)" in 2016 and 2018. Participants were the city government employees in a rural city in Shiga prefecture, Japan. Presenteeism, insomnia and depression were analyzed by WHO-HPQ (Health and Work Performance Questionnaire), ISI (insomnia severity index) and PHQ-9 (Patient Depression Questionnaire), respectively. Pearson correlation coefficient analyses were performed to determine the strength of the association between two variables. Logistic regression was performed to estimate the odds ratio (OR) and 95% confidence interval (CI) of poor relative presenteeism (the lowest tertile of the relative presenteeism scores) after 2-year follow up.

Results: 1143 subjects (participation rate: 61.7%, 36.7% male, 44.5 ± 11.4 years, BMI: 22.3 ± 3.30) participated in both 2016 and 2018. Participants with poor productivity (poor relative presenteeism) in 2018 was significantly associated with ISI in 2016 (OR: 1.050, 95%CI: 1.010-1.090, p=0.013) but not with PHQ-9 in 2016 (OR: 1.008, 95%CI: 0.972-1.045, p=0.664) after adjusting for age, gender and BMI. Positive correlation was found between the total score of ISI and item 3 of PHQ-9 which asks insomnia or hypersomnia symptom (r=0.6122, P<0.0001).

Conclusion: Insomnia may be an independent risk factor for poor presenteeism. ISI may be useful to predict poor productivity in the future.

Support: Supported in part by a research grant from Investigator-Initiated Studies Program of Merck Sharp & Dohme Corp. / MSD K.K. The opinions expressed in this paper are those of the authors and do not necessarily represent those of Merck Sharp & Dohme Corp. / MSD K.K."

0454

SLEEP DISRUPTIVE COGNITIONS IN DEPLOYMENT-RELATED INSOMNIA

Howell, M. Mellman, T. Howard University, Washington, DC.

Introduction: Sleep disturbance is common following military deployment, and insomnia is associated with many adverse psychiatric and medical outcomes. Deployment to a threatening environment can engender nocturnal vigilance, which may be a salient feature of

sleep disturbance in formerly deployed Veterans. Cognitive behavioral therapy for insomnia (CBTI) is an effective treatment; however, CBTI emphasizes targeting dysfunctional beliefs about sleep (DBAS) and generalized worries rather than vigilance. The goal of the present study was to investigate the role of vigilance, in relation to other potential types of sleep-interfering cognitions in formerly deployed Veterans with sleep disturbance.

Methods: Thirty-nine formerly deployed Veterans with disturbed sleep completed measures prior to an intervention. Insomnia was measured with the Insomnia Severity Index (ISI) and measures derived from actigraphy and morning sleep diaries administered for one week. Measures for sleep interfering cognitions included Dysfunctional Beliefs about Sleep (DBAS), the Penn State Worry Questionnaire (PSWQ), a measure of generalized worry, and the Fear of Loss of Vigilance (FLV) subscale of the Fear of Sleep Inventory (FOSI).

Results: All of the measures of sleep-interfering cognitions were significantly associated with ISI score. Generalized worries (PSWQ scores) were strongly and significantly correlated with both FLV and DBAS, which were not significantly correlated with each other. FOSI FLV explained 7.1% more variance in ISI score than DBAS alone (p = .04) while DBAS explained 23.1% additional variance in ISI score over FOSI FLV alone (p < .001).

Conclusion: It may be important to target both nocturnal vigilance and dysfunctional beliefs about sleep in the treatment of insomnia in formerly deployed Veterans.

Support: Supported by W81XWH-14-1-0066 from the Congressionally Directed Peer-Reviewed Medical Research Program of the Department of Defense.

0455

ROLE OF INTERACTION BETWEEN ANTERIOR INSULA RESPONSE TO SLEEP-RELATED PICTURES AND STRESS LEVELS ON SLEEP DISTURBANCE

Lee, M.¹ Lee, K.¹ Lee, H.¹ Kim, N.² Jeon, J.¹ Jeon, S.³ Oh, S.⁴ Kim, S.⁵ Kim, S.³ Lee, Y.¹

¹Seoul National University Hospital, Seoul, KOREA, REPUBLIC OF, ²Neuroscience Research Institute, Gachon University, Incheon, KOREA, REPUBLIC OF, ³Department of Psychiatry, Sungkyunkwan University College of Medicine, Samsung Medical Center, Seoul, KOREA, REPUBLIC OF, ⁴Department of Psychiatry, Dongguk University Ilsan Hospital, Ilsan, KOREA, REPUBLIC OF, ⁵Department of Neurology, Gangneung, Gangneung Asan Hospital, Gangneung, KOREA, REPUBLIC OF.

Introduction: Literature suggests that stress may play an important role in sleep disturbance. Individuals with higher stress levels often showed hyperarousal to stressful events, possibly leading to sleep disturbance. Hyperarousal is also one of features of stress-related sleep disturbance. Here, we examined the extent to which stress levels interact with neural activity in response to sleep-related information to predict sleep disturbance.

Methods: Forty eight healthy adults (26 females, age = 35.7 ± 10.5) without sleep disorders based on nocturnal polysomnography participated in this study. They were viewing sleep-related pictures (e.g., bedroom and sunset) and non-sleep related, neutral pictures (e.g., kitchen and landscape) during fMRI scanning. They also completed questionnaires assessing stress levels and sleep disturbance using Life Experience Survey (LES) and Pittsburgh Sleep Quality Index (PSQI), respectively. Activity in response to

B. Clinical Sleep Science and Practice

sleep-related pictures compared to neutral pictures was extracted from our region-of-interest (ROI), the anterior insula, and entered into our moderation models. The SPSS macro PROCESS (Hayes, 2013) was used to conduct moderation analyses. Given a significant correlation between age and PSQI scores, age was included as a covariate.

Results: Our moderation analyses showed that interactions between stress levels and anterior insula response to sleep-related pictures significantly predicted sleep disturbance. Simple slope analyses showed that at higher anterior insula response, higher stress levels were associated with greater sleep disturbance, but at lower anterior insula response, stress was not significantly associated with sleep disturbance. These results indicate that individuals with high levels of stress were more likely to experience sleep disturbance if they showed greater anterior insula response to sleep-related pictures (i.e., hyperarousal in response to sleep-related information).

Conclusion: The current findings suggest that interactions between stress levels and neural substrates of hyperarousal, particularly the anterior insula, may play a critical role in sleep disturbance.

Support: Brain Research Program through the National Research Foundation of Korea funded by the Ministry of Science, ICT & Future Planning (Study No.: 2016M3C7A1904338 and NRF-2018R1D1A1B07049704).

0456

NATURAL HISTORY OF INSOMNIA: SLEEP REACTIVITY PREDICTS NEW-ONSET ACUTE INSOMNIA

Vargas, I.¹ Drake, C.² Muench, A.³ Boyle, J.⁴ Morales, K.³ Grandner, M.⁵ Ellis, J.⁶ Perlis, M.³

¹University of Arkansas, Fayetteville, AR, ²Henry Ford Hospital, Novi, MI, ³University of Pennsylvania, Philadelphia, PA,

⁴Philadelphia College of Osteopathic Medicine, Philadelphia, PA, ⁵University of Arizona, Tucson, AZ, ⁶Northumbria University, Newcastle, UNITED KINGDOM.

Introduction: Greater vulnerability to stress-related sleep disturbance (i.e., sleep reactivity) is a risk factor for chronic insomnia (CI). What has not been investigated is whether greater sleep reactivity, as assessed by the Ford Insomnia Response to Stress Test (FIRST), predicts the onset of acute insomnia (AI), and more, whether greater sleep reactivity predicts the transition from AI to CI.

Methods: A national cohort of 1,222 good sleeper subjects (68% female; mean age=53.2 years) were prospectively assessed to estimate the incidence of AI and CI. The FIRST was completed at baseline and sleep diaries were completed on a daily basis for a period of one year. Subjects were categorized based on their FIRST scores (high, FIRST>16; low, FIRST≤16). Subjects were also grouped based on whether they developed AI (two consecutive weeks with a frequency of \geq 3 nights per week of sleep initiation or maintenance problems) or maintained good sleep (GS; n=896). For those subjects that transitioned to AI (n=326), they were also grouped based on whether or not they developed CI (insomnia \geq 3 nights/week for at least three months; n=23). Chi-square analyses were performed to determine if higher FIRST scores at baseline predicted the incidence of AI or CI.

Results: 32.5% of subjects in the high FIRST group met criteria for AI at some point during the one-year interval, whereas 22.5% of subjects in the low FIRST group experienced AI (χ^2 =15.2, p<.001). In contrast, FIRST did not predict CI status (low FIRST, 8.5% CI, high FIRST, 5.6% CI; χ^2 =1.1, p=.30).

Conclusion: Greater sleep reactivity predicted incident AI but not the onset of CI. While these findings suggest that sleep reactivity

may be a predisposing factor for AI, data are not consistent with previous findings showing FIRST scores are predictive of the development of CI. It's possible that the present study was underpowered to detect these differences, given that the incidence of CI was low (less than 2% of the total sample). Additional analyses are ongoing to evaluate the temporal association between stressful life events and AI in subjects with high and low FIRST scores. **Support:** Perlis: NIH R01AG041783, K24AG055602

0457

INSOMNIA AND CAUSE-SPECIFIC MORTALITY IN MEN AND WOMEN

He, F.¹ Fernandez-Mendoza, J.¹ Vgontzas, A. N.¹ Calhoun, S. L.¹ Liao, D.¹ Bixler, E. O.¹

¹Penn State College of Medicine, Hershey, PA, ²Penn State College of Medicine, Hershey, PA.

Introduction: The association of insomnia with an increased risk of mortality has remained inconsistent across studies, which contrasts with accumulating evidence linking this prevalent and chronic sleep disorder with cardiovascular, cerebrovascular, oncologic, and psychiatric morbidity. The higher prevalence of insomnia in women compared to men may be an important contributor to the different survival rates reported in large, population-based studies.

Methods: The Penn State Adult Cohort is a random, general population sample of 1,741 adults ($48.8\pm13.6y$, 52.2% women) who were studied in the sleep laboratory and followed-up for their cause of death up to December 31, 2018. Insomnia was defined as a chronic complaint lasting at least 1 year (n=199). We assessed the risk of all-cause mortality (n=664) and the two most common causes of death: cardiovascular/cerebrovascular (n=275) and cancer (n=161). Cox proportional hazard models adjusted for age, race, sex, education, smoking, alcohol, BMI, AHI, cognitive impairment, mental health problems and physical health problems, including hypertension, diabetes, heart disease, stroke and cancer at baseline.

Results: The risk of all-cause mortality associated with insomnia was significantly increased in men (HR=1.84, 95%CI=1.18-2.87) but not in women (HR=0.80, 95%CI=0.57-1.13; p for sex-interaction<0.01). Insomnia was significantly associated with an increased risk of cardiovascular/cerebrovascular mortality in men (HR=2.11, 95%CI=1.14-3.99), but not in women (HR=0.98, 95%CI=0.59-1.63; p for sex-interaction=0.06). Insomnia was not significantly associated with an increased risk of cancer mortality either in men (HR=1.41, 95% CI=0.56-3.56) or in women (HR=0.90, 95% CI=0.45-1.80), after adjusting for hypertension, diabetes, heart disease, stroke and cancer at baseline.

Conclusion: Men with chronic insomnia are at an increased risk of mortality, particularly that of cardiovascular/cerebrovascular origin. There is a need for translational studies focused on sex-differences that can disentangle the biological and behavioral mechanisms underlying women's resilience.

Support: American Heart Association (14SDG19830018), National Institutes of Health (R01HL51931, R01HL40916)

0458

SMOKING AND CAFFEINE CONSUMPTION DIFFER BETWEEN INSOMNIA PHENOTYPES BASED ON OBJECTIVE SLEEP DURATION

Vgontzas, A. N.¹ Fernandez-Mendoza, J.¹ Puzino, K.¹ Calhoun, S. L.¹ Krishnamurthy, V. B.¹ Basta, M.² Bixler, E. O.¹

¹Penn State College of Medicine, Hershey, PA, ²University of Crete, Heraklion, GREECE.

Introduction: The insomnia with short sleep phenotype (ISS), in contrast to the normal sleep phenotype (INS), is characterized by physiological hyperarousal including activation of the stress system and cardiometabolic morbidity. The aim of this study was to assess whether these two insomnia phenotypes differ in terms of the use of two common stimulants (i.e., caffeine and nicotine).

Methods: Data from the Penn State Adult Cohort (N=1741) was used in this study (52.2% women, 48.8±13.6 years). A 1-night, 8-hour, polysomnography (PSG) was used to classify subjects into normal (≥6h) and short (<6h) sleep duration groups. Self-reported sleep difficulty was defined based on three levels of severity as normal sleep (n=1022), poor sleep (n=520) and insomnia (n=199). Self-reported heavy caffeine use was defined as ≥3 cups daily and heavy smoking as ≥20 cigarettes daily. Multinomial logistic regression analyses were conducted adjusting for covariates such as age, gender, and race.

Results: Compared to normal sleepers, ISS (OR=0.55, 95% CI=0.31-0.97, p=0.04), but not INS (OR=0.92, 95% CI=0.52-1.64, p=0.77), was associated with significantly less heavy caffeine use. In contrast, INS (OR=2.20, 95% CI=1.10-4.40, p=0.03), but not ISS (OR=0.95, 95% CI=0.41-2.17, p=0.90), was associated with significantly more heavy smoking.

Conclusion: These results indicate that the use of common stimulants (i.e., smoking cigarettes and drinking caffeine) is higher in the INS phenotype than the ISS phenotype. Individuals with the ISS phenotype may be using less caffeine and tobacco to avoid further stimulation of the already hyperaroused physiologic system, which may result in worsening of their insomnia. In the INS phenotype, changes in health behaviors should be an important part of a multidimensional approach to treatment.

Support: American Heart Association (14SDG19830018), National Institutes of Health (R01HL51931, R01HL40916)

0459

IS IT HAVING A BABY OR ME? DIFFERENTIATING INSOMNIA DISORDER AND SLEEP DISRUPTION FROM PREGNANCY TO 2 YEARS POSTPARTUM

Quin, N.^{1,2} Lee, J.¹ Pinnington, D. M.^{1,2} Shen, L.¹ Manber, R.³ Bei, B.^{1,2}

¹Monash University, Melbourne, AUSTRALIA, ²Royal Women's Hospital, Melbourne, AUSTRALIA, ³Stanford University, Stanford, CA.

Introduction: Insomnia Disorder (Insomnia) diagnosis requires sleep complaints to persist despite "sleep-conducive conditions and adequate sleep opportunity". Women experience significant sleep disruption during pregnancy and postpartum periods due to physiological changes and night-time infant care, but not all women with sleep complaints meet Insomnia criteria. This study examined sleep and mental health correlates of Insomnia Disorder and sleep complaints in the context of a randomised controlled trial for improving maternal sleep.

Methods: 163 generally healthy first-time mothers (age $M\pm SD=33.4\pm3.5$) with singleton pregnancy repeated the following assessments at 28-30 and 35-36 weeks' gestation, and 1.5, 3, 6, 12, and 24 months postpartum: the Insomnia module of the Duke Structured Interview for Sleep Disorders, PROMIS Sleep-Related Impairment, Depression, and Anxiety Short Forms. We compared

clinical features when DSM-5 Insomnia criteria (less the 3-month criteria) were (1) met (Insomnia), (2) not met only because of the sleep condition/opportunity criteria (Sleep Disruption), and (3) not met due to low symptom/distress (Low Complaint).

Results: 944 interviews and 1009 questionnaire were collected across 7 time-points. Proportions of women meeting Insomnia criteria were 16.0% and 19.8% during early and late third trimester, and ranged 5.3-11.7% during the 5 postpartum time-points. If the sleep condition/opportunity criteria were not considered, rates of "Insomnia" would have been 2-4 times higher at 21.4-40.4% across all time-points. Mixed effects models, controlling for intervention allocation, showed that compared with Insomnia, Sleep Disruption had comparable depression (p=.68) and anxiety (p=.23), and somewhat lower sleep-related impairment (p=.06). These symptoms were lowest for Low Complaint.

Conclusion: Both Insomnia and Sleep Disruption were associated with significant daytime impairment, depression, and anxiety. Assessing sleep complaints without considering sleep condition/opportunity can result in over-diagnosis of perinatal Insomnia in these women with primarily sleep disruption; these women may have limited benefits from Insomnia-specific treatment. Interventions for maternal sleep should carefully differentiate between Insomnia and other sleep concerns (e.g., sleep disruption/opportunity, sleepiness/fatigue) and appropriately address each.

Support: Australasian Sleep Association, Monash University, Royal Women's Hospital Foundation. National Health and Medical Research Council, Department of Education and Training.

0460

INSOMNIA IN 400 WOMEN: POLYSOMNOGRAPHY, IMMUNE PARAMETERS, DEPRESSION AND ANXIETY

Akerstedt, T.¹ Ghilotti, F.² Schwarz, J.³ Theorell-Haglöw, J.⁴ Lindberg, E.⁴

¹Karolinska institute, Stockholm, SWEDEN, ²Karolinska Institute, Stockholm, SWEDEN, ³Stockholm University, Stockholm, SWEDEN, ⁴Uppsala University, Uppsala, SWEDEN.

Introduction: Insomnia disorder has a very weak link to polysomnography (PSG) and so does sleep problems in the general population. The reason for this is not clear. One possibility is that the perception of disturbed sleep may be related to immune activation or anxiety/depression, without impairment of objective sleep. **Methods:** 400 women participated, constituting a representative sample of the city of Uppsala with oversampling of snorers. Insomniacs (N=41) were compared with normal sleepers in terms of polysomnography (PSG), immune parameters, anxiety and depression

Results: The results (after adjustment for age and BMI) show that C-reactive protein (CRP) reached a higher level ($4.4\pm.5$) in insomniacs (vs $2.3\pm.2$ for normal sleepers) (p=.003) and lower subjective health (p=.000), while anxiety (p=.000) and depression (p=.000) (Hospital Anxiety and Depression Scale (HAD)) showed higher levels. PSG sleep continuity variables lacked association with insomnia, as did all sleep stage variables except for REM%, with a lower level in the insomnia group (p=.021). Interleukin 6 and Tumor Necrosis Factor alpha were not related to insomnia. CRP levels did not correlate significantly with anxiety or depression, but with subjective health (r=-.21, p=.000). A logistic regression analysis (excluding the variable subjective health) with insomnia as outcome (0/1) yielded as predictors CRP (OR=1.14, Ci= 1.05;

1.24, p=.000), depression (OR=1.21, Ci=1.06;1.38, p=.000) and anxiety (OR=1.15, Ci=1.02;1.30, p=.021).

Conclusion: It was concluded that increased CRP levels may be part of the subjective experience of insomnia. **Support:** No external funding

0461

BLUNTED BAROREFLEX SENSITIVITY AND REDUCED MORNING PARASYMPATHETIC ACTIVITY IN SLEEP-ONSET INSOMNIA

Tsai, H.¹ Kuo, T.^{1,2} Yang, C.^{1,2}

¹Institute of Brain Science, National Yang-Ming University, Taipei, TAIWAN, ²Sleep Research Center, National Yang-Ming University, Taipei, TAIWAN.

Introduction: Insomnia is a risk factor for hypertension and cardiovascular events, and this association is strongest for sleep-onset insomnia. However, little is known about insomnia on cardiovascular modulation, especially soon after morning awakening, the peak period of time for cardiovascular incidents. This study explored morning cardiovascular function in individuals with sleeponset insomnia by analysing heart rate variability, blood pressure variability, and baroreflex sensitivity.

Methods: Sleep structure of the participants (15 good sleepers and 13 individuals with sleep-onset insomnia) was measured by laboratory polysomnography, followed by continuous recordings of the participant's blood pressure and heart rate for 10 min in the morning.

Results: When compared to the good sleepers, the insomnia group showed significant reductions in total sleep time, a longer sleep-onset latency, and reduced sleep efficiency. The sleep structure, including durations of sleep stages, numbers of awakenings and arousal index did not differ between the groups. After morning awakening (averaged time: 12.33 ± 10.48 min), the shorter R-R intervals, lower total power, and lower high-frequency power of heart rate variability were observed among individuals with sleep-onset insomnia, compared with good sleepers. Elevated slopes of systolic and diastolic blood pressure, as well as lower baroreflex sensitivity, were also shown in the insomnia group. Indices of sympathetic activity, including low-frequency power of blood pressure variability, did not differ between the groups.

Conclusion: Weak vagal activity and blunted baroreflex sensitivity were evident among sleep-onset insomnia. These findings indicate difficulty in initiating sleep, without significant sleep fragmentation, can independently affect morning cardiovascular function. This study provides a possible link between sleep-onset insomnia and risk of cardiovascular events. **Support:** N/A

0462

IDENTIFYING AND CHARACTERIZING INSOMNIA SYMPTOM GROUPS ACROSS THE DEPLOYMENT CYCLE IN CURRENT ARMY SOLDIERS

Miller, K. E.¹ Boland, E. M.^{1,2} Klingaman, E. A.³ Gehrman, P. R.^{2,1} ¹VISN ⁴ MIRECC, Cpl. Michael J. Crescenz VA Medical Center, Philadelphia, PA, ²Department of Psychiatry, Perelman School of Medicine at the University of Pennsylvania, Philadelphia, PA, ³Department of Veterans Affairs VISN ⁵ MIRECC, Baltimore, MD. **Introduction:** Most research conducted on insomnia and its development in military personnel focuses on cross-sectional data, precluding examination of the course of sleep changes over time. The present study characterized Army Soldiers based on insomnia symptom status trajectory from pre to post-deployment and explored baseline factors predictive of these trajectories in a sample of 7,245 soldiers across 3 Brigade Combat Teams.

Methods: Data were analyzed from the Army Study to Assess Risk and Resilience in Service members (STARRS)-All-Army Study (AAS) Pre Post Deployment Study, using surveys that captured 1-2 months pre-deployment, during deployment, and 6-months post-deployment. Insomnia symptom status was defined at each timepoint as insomnia symptoms that interfered with one or more domains of functioning at least some of the time in the past month. Theoretically-derived variables linked to sleep disturbance were selected as predictors of insomnia symptom trajectory and evaluated using a general linear selection model.

Results: Four trajectories characterized the majority of the sample: 'good sleepers' (no insomnia symptoms across time; 44.4%), 'nonremitting new onset insomnia' (no pre-deployment insomnia, developed insomnia symptoms during deployment that remained at 6 months; 22.8%), 'deployment-only insomnia symptoms' (no predeployment insomnia, developed insomnia during deployment but recovered by follow-up; 12.8%), and 'chronic insomnia' (insomnia both pre- and post-deployment; 7.4%). Several pre-deployment factors predicted insomnia trajectory, the strongest of which were past six-month attention deficit disorder symptoms, number of lifetime exposures to potentially traumatic events, and past month depression symptoms.

Conclusion: Insomnia is one of the most common reasons that military personnel seek behavioral health treatment and is associated with poorer military readiness. Better characterization and identification of insomnia symptoms over time can improve intervention during post-deployment transitions, particularly for those with new onset insomnia that does not remit.

Support: Cooperative agreement U01MH087981 (Department of the Army; U.S. Department of Health and Human Services, National Institutes of Health, National Institute of Mental Health); U.S. Department of Veterans Affairs, Clinical Science Research and Development-IK2CX001874-PI:Katherine E. Miller, IK2CX001501-PI:Elaine M. Boland; Rehabilitation Research and Development-1IK2RX001836-PI:Elizabeth A. Klingaman. The views expressed here are those of the authors and do not represent the Department of Veterans Affairs.

0463

CLUSTER ANALYSIS IN PERIMENOPAUSAL AND MENOPAUSAL WOMEN WITH INSOMNIA

Srisawart, P.¹ Wang, L.² Bena, J.² Drerup, M.¹ Mehra, R.¹ Barwick, F.⁴ Moul, D.¹

¹Sleep Disorders Center, Neurological Institute, Cleveland Clinic, Cleveland, OH, ²Lerner Research Institute, Cleveland Clinic, Cleveland, OH, ³Sleep Disorders Center, Neurological Institute, Cleveland Clinic, Cleveland, OH, ⁴Department of Psychiatry & Behavioral Sciences, Stanford School of Medicine, Stanford, CA.

Introduction: Women in perimenopause or menopause report higher rates of insomnia, with depression, pain and sleep apnea common comorbidities. Identifying clinically relevant subtypes of women with similar symptom patterns might help target treatment more precisely and optimize outcomes more successfully.

Methods: Participants were woman >50 years with insomnia (ISI>10) who were recruited from 12,108 patients visiting the Cleveland Clinic Sleep Disorders Center between 2008-2012. Patients completed questionnaires at initial clinic visit, and comorbidity data was extracted from medical records. K-method cluster analysis of cross-sectional data with PAM (portioning around medoids) was performed to identify clusters of patients based on insomnia (ISI), depression (PHQ2), and pain (EQ5D) symptoms as well as presence or absence of diabetes or sleep disorders (OSA, RLS). Silhouette widths and visualization using factoextra in R identified the optimal number of clusters. Characteristics of each cluster were compared using Pearson chi-square, Kruskal-Wallis or ANOVA models in SAS.

Results: Sample comprised 374 women. Average age was 60.5 years and 81.6% were White. A three-cluster solution was the most plausible. Clusters with mild (N=155, ISI=14.1±1.9), moderate (N=131, ISI=19.7±1.6) and severe (N=88, ISI=25.4±1.9) insomnia showed significant differences in characteristic. Clusters differed on depression level (PHQ2≥4 mild 19%, moderate 38%, severe 60%), and pain (ED5D=3 mild 3%, moderate 12%, severe 23%). Although the mild insomnia cluster reported better overall health, it showed higher rates of OSA compared to the moderate insomnia cluster, along with significantly older age and higher BMI.

Conclusion: Perimenopausal and menopausal women divided into three clusters with mild, moderate and severe insomnia, with levels of reported depression and pain symptoms increasing with insomnia symptoms. Clusters also differed on age, BMI and prevalence of OSA, suggesting that specific symptom clusters might indicate more precise and targeted treatment of common comorbid conditions during menopause transition. **Support:**

Suppor

0464

SLEEP CONTINUITY BY NEIGHBORHOOD: ARE THERE DIFFERENCES?

Giller, J.¹ Muench, A.¹ Grandner, M. A.² D'Antonio, B.¹ Perlis, M. L.¹

¹Behavioral Sleep Medicine Program, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA, ²Sleep and Health Research Program, Dept of Psychiatry, University of Arizona, Tuscon, AZ, ³Behavioral Sleep Medicine Program, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA.

Introduction: To our knowledge, no prior work has been conducted on whether sleep continuity disturbance (e.g., SL; WASO; EMA, etc.) varies by neighborhood. While differences on these metrics have been found by, e.g., race and socioeconomic status, it may also be that sleep continuity disturbance varies relative to where one lives and works. Accordingly, an analysis was undertaken to assess whether regional differences exist with respect to sleep continuity disturbance (SCD).

Methods: The study utilized a cross-sectional group design in an archival/community dataset that was collected in the Philadelphia area (www.sleeplessinphilly.com). This dataset (n = 2840) was comprised of adults between 18 and 89 years of age with self-reported sleep complaints (63.4% female; 36.6% male; average age 38). Study subjects who endorsed >30 minutes on >3 days/week on SL, WASO & EMA were categorized by zip code and into four regional groups: Center City (n=258); South Philadelphia (n=103); North

& Northeast Philadelphia (n=400) and West Philadelphia (n=345). Contingency analyses and ANOVAS were used to assess for regional group differences.

Results: It was found that SCD rates significantly differed by region. Differences in percent endorsement by region were as follows, SL:, 64.1% (Northeast/North), 63.5 (South), 56.3% (West Philadelphia), & 48.7% (Center City); WASO: 45% (Northeast/North), 40% (South), 36.5% (West Philly), & 32.4% (Center City); EMA: 46.4% (South); 43.7 (Northeast/North); 43.7 (West Philly); 43.1 (Center City).

Conclusion: The Northeast/North region of Philadelphia had the highest rates, and center city had the lowest rates of SCD for the early part of the night (SL & WASO). Early morning SCD was most common for "South Philly and least common for Center City. Analyses are on-going in relation to other regional differences (demographic, income, crime stats, etc.) and those found to vary by region will be assessed for their predictive value.

Support: No support was provided for this abstract

0465

ASSOCIATION BETWEEN PHYSICAL OR SEXUAL ABUSE AND SLEEP REACTIVITY IN PATIENTS WITH INSOMNIA

Drake, C. L.¹ Cheng, P.¹ Kalmbach, D.¹ Roth, T.¹ Sagong, C.² Arnett, L.²

¹Henry Ford Health System, Detroit, MI, ²Henry Ford Health System, Detoirt, MI.

Introduction: Physical and sexual abuse are common and have demonstrated associations with insomnia. A common factor predisposing individuals to the development of insomnia is sleep reactivity, yet no studies have determined the relationship between sleep reactivity and physical and/or sexual abuse.

Methods: Patients with DSM-5 insomnia disorder (N = 658; 519 F; mean age = 45.03) participated in an online randomized controlled trial of behavioral treatment of insomnia. Participants completed the Insomnia Severity Index (ISI), Ford Insomnia Response to Stress Test (FIRST), and demographic information at baseline. Abuse history was assessed with a single 4-choice item asking participants if they had a history of physical or sexual abuse. One-way analysis of variance was used to determine the level of sleep reactivity in each of the 4 abuse groups (none, physical, sexual, both) controlling for insomnia (ISI). Post-hoc analyses also compared differences in self-reported difficulties falling and staying asleep.

Results: Compared to those with no abuse history (n = 465), patients who reported both physical and sexual abuse (n = 50) had significantly elevated FIRST scores (p < .001). Results remained significant after controlling for severity of insomnia, age, and gender. Post-hoc analyses showed group differences in sleep onset latency but not wake after sleep onset.

Conclusion: Combined physical and sexual abuse are associated with higher levels of sleep reactivity to stress, and that the effects may be most prominent for difficulties falling asleep and not sleep maintenance. Prospective studies are needed to determine the potential effects of abuse on sleep reactivity, and whether sensitization of the sleep system to stress constitutes an etiological pathway in the development of insomnia disorder in survivors of abuse. Future work should also investigate potential overlapping biological markers of abuse and sleep reactivity.

Support: This study was funded by the Robert Wood Johnson Foundation (PI: Drake).

0466

EVOLUTION OF NONRESTORATIVE SLEEP AND ITS INTERACTIONS WITH INSOMNIA SYMPTOMS IN A LONGITUDINAL SURVEY OF THE AMERICAN POPULATION

Ohayon, M. M. Stanford University, Palo Alto, CA.

Introduction: Nonrestorative sleep (NRS) is one of the sleep disturbances that is under-appreciated. Some studies have set its prevalence at around 10% of the general population but definitions are heterogenous. Despite its frequency, studies that paid attention to that symptom are disparate and have often taken many routes. Our aim is to examine its prevalence, its incidence and its predictive factors.

Methods: The initial study was carried with 15,929 individuals from 15 US States. The longitudinal study was carried on in eight of these states. A total of 12,218 subjects were interviewed by phone during the first wave (W1) and 10,930 at the second wave (W2) three years apart. The analyses were carried on the subjects who participated in both interviews (N=10,930). NRS was assessed using a series of five questions. The global score determined the presence/absence of NRS.

Results: A total of 14.7% (CI95%: 14%-15.4%) reported NRS at W1. At follow-up, 13.1% (CI95%: 12.5%-13.7%) reported NRS. The incidence per year was 2.3%. NRS was chronic in 28.9% of cases. NRS occurred alone (i.e. without any other insomnia symptoms) in 5% of the sample at W1 and 3.6% at W2. 22.2% of those with NRS alone at W1 reported other insomnia symptoms at W2. Sleep duration was at least 6h30 in 81.6% of NRS alone cases at W1 and 76.5% at W2. Daytime repercussions were reported by 66.2% of NRS alone at W1 and 52.8% at W2. NRS alone (RR: 2.4) or in combination with insomnia symptoms (RR: 3.4) was one of the strongest predictors for developing a Major Depressive Disorder at W2.

Conclusion: NRS is a sleep disturbance that has some unique features that distinguish it from insomnia symptoms. Nonetheless, it can have a profound impact on daily life and can lead to further difficulties in other areas if not addressed properly.

Support: Arrillaga Foundation

0467 BENEFITS OF CBT-I FOR WOMEN VETERANS WITH AND WITHOUT PTSD

Carlson, G. C.¹ Kelly, M. R.² Josephson, K.² Mitchell, M.² Fiorentino, L.³ McGowan, S.^{4,5} Culver, N.⁴ Kay, M.⁴ Alessi, C.^{2,6} Washington, D. L.^{1,6} Yano, E.^{1,7} Martin, J. L.^{2,6}

¹HSR&D Center for the Study Healthcare Innovation, Implementation and Policy, VA Greater Los Angeles Healthcare System, Los Angeles, CA, ²Geriatric Research, Education and Clinical Center, VA Greater Los Angeles Healthcare System, Los Angeles, CA, ³University of California, San Diego, San Diego, CA, ⁴Department of Mental Health, VA Greater Los Angeles Healthcare System, Los Angeles, CA, ⁵Department of Psychiatry, David Geffen School of Medicine at University of California, Los Angeles, Los Angeles, CA, ⁶Department of Medicine, David Geffen School of Medicine at University of California, Los Angeles, Los Angeles, CA, ⁷Department of Health Policy & Management, Fielding School of Public Health, University of California, Los Angeles, Los Angeles, CA. **Introduction:** A quarter of women Veterans (WVs) receiving VA healthcare meet diagnostic criteria for both insomnia disorder and posttraumatic stress disorder (PTSD). Cognitive Behavioral Therapy for Insomnia (CBT-I) is effective at improving sleep among individuals with comorbid psychiatric conditions; however, no studies have examined the impact of CBT-I in women with insomnia plus PTSD. The current analyses examined changes in sleep symptoms, quality of life (QoL), and mental health symptoms from pre- to post-CBT-I in WVs with and without PTSD.

Methods: This was a secondary analysis of 75 WVs with insomnia (32 with probable PTSD), who received CBT-I within a behavioral sleep intervention study (NCT02076165). Measures completed at baseline, posttreatment, and 3-month follow-up included: insomnia severity (Insomnia Severity Index, ISI), sleep quality (Pittsburgh Sleep Quality Index, PSQI), PTSD symptoms (PTSD Checklist-5, PCL-5; probable PTSD=total score \geq 33), depressive symptoms (Patient Health Qestionnaire-9, PHQ-9), and mental and physical quality of life (Short Form Health Survey, SF-12). One sample *T*-tests examined changes in ISI, PSQI, PHQ-9, PCL-5, and SF-12 from baseline to posttreatment and baseline to follow-up. Two samples *T*-tests compared change scores in ISI, PSQI, PHQ-9, and SF-12 between participants with and without PTSD.

Results: There were significant improvements in ISI ($p \le .001$), PSQI ($p \le .001$), PHQ-9 ($p \le .001$), PCL-5 (p = .001), and SF-12 mental ($p \le .001$) and physical (p = .03) from baseline to posttreatment and 3-month follow-up ($p \le .001$ -.01). There were no significant change score differences between WVs with and without PTSD from baseline to posttreatment (p = .06-.98) or 3-month follow-up (p = .09-.93). **Conclusion:** CBT-I appears to be an effective treatment to improve insomnia symptoms among WVs with and without PTSD, and may reduce psychiatric symptoms as well. These findings suggest WVs with comorbid insomnia and PTSD benefit from CBT-I. The appropriate sequencing of CBT-I and PTSD treatments remains potentially important, but unstudied.

Support: VA/HSR&D IIR-HX002300; NIH/NHLBI K24HL14305; VA Office of Academic Affiliations through the Advanced Fellowship Programs in HSR&D and Women's Health

0468

A MULTIPLE DOSE, PLACEBO-CONTROLLED, RANDOMIZED DOUBLE-BLIND, MULTICENTER STUDY TO INVESTIGATE TRIPROLIDINE IN THE TREATMENT OF TEMPORARY SLEEP DISTURBANCE

Reyner, L.¹ Miller, J. E.² Shea, T.³

¹Awake Ltd., Woodhouse Eaves, UNITED KINGDOM, ²SRxA, McLean, VA, ³RB, Parsippany, NJ.

Introduction: American Association of Sleep Medicine guidelines states that the primary goals of the treatment of insomnia are to improve sleep quality and related daytime function. While H1 antihistamines have sedative effects, they are associated with residual daytime sleepiness and an effective dose range for hypnotic effect has hitherto not been established. Triprolidine a first generation antihistamine used to treat allergic rhinitis and the common cold has a mean half-life of 3.2 hours. We evaluated the effect of two doses of triprolodine compared with placebo on sleep onset latency and daytime sleepiness to determine the optimum dose in subjects with temporary sleep disturbance.

Methods: Multicenter, placebo-controlled, parallel group, double blind, multiple dose, randomized study of 178 patients aged 18 years or above with a primary diagnosis of temporary sleep disturbance. Patients were randomized to one of three groups.

Group 1: 2 x placebo tablets; Group 2: 1 x placebo tablet + 1 x 2.5mg triprolidine tablet; Group 3: 2 x 2.5mg triprolodine tablets, taken 20 minutes before intended sleep on three consecutive evenings. Efficacy was measured objectively using the Sleep Disturbance Index using a wrist actimeter and subjectively using the Loughborough Sleep Log and Karolinska Sleepiness Scale.

Results: Both doses were statistically significantly superior to placebo in terms of quality and duration of sleep and sleep interruptions. No hangover effects or daytime sleepiness were observed with either dose compared to placebo. Patients on the 2.5 mg dose awoke more refreshed than the 5 mg dose. No serious adverse effects observed in any group and anticholinergic events i.e. dry mouth were very low.

Conclusion: Tripolidine is effective and safe in the treatment of temporary sleep disturbance. The optimum dose is 2.5mg.

Support: The study was sponsored by Boots Healthcare International.

0469

PERCEPTIONS OF AN EDUCATION ONLY CONTROL GROUP IN THE OSTEOARTHRITIS AND THERAPY FOR SLEEP (OATS) STUDY: AN ONGOING STATEWIDE, TELEPHONE-DELIVERED CBT FOR INSOMNIA (CBT-I) RANDOMIZED TRIAL

*McCurry, S. M.*¹ *Vitiello, M. V.*¹ *Pike, K. C.*¹ *Thakral, M.*² *Morin, C. M.*³ *Von Korff, M.*⁴

¹University of Washington, Seattle, WA, ²University of Massachusetts Boston, Boston, MA, ³Université Laval, Quebec City, QC, CANADA, ⁴Kaiser Permanente Washington Health Research Institute, Seattle, WA.

Introduction: Comorbid osteoarthritis (OA) and insomnia is common in older adults. CBT-I is efficacious for improving sleep in older persons with OA but not widely accessible. We examined treatment process data from OATS, a large ongoing clinical trial of telephone-delivered CBT-I.

Methods: 327 Kaiser Permanente Washington members aged 60+ with OA, Insomnia Severity Index scores of 11+, and Brief Pain Inventory scores of 9+ were randomized to individual CBT-I vs. education only control (EOC). Six telephone sessions of CBT-I or EOC were offered over eight weeks. Participants rated their perceptions of treatment (credibility, acceptability, suitability, perceived effectiveness, adherence, and therapeutic relationship) on a 7-point Likert scale after session 1 and at 2-month post-test.

Results: Participants (mean age=70.2 years, 74.6% female) were randomized to the two treatment arms (CBT-I=163, EOC=164). Participants did not differ significantly across arms by age, gender, education, or by sleep, pain, or mood (depression, fatigue) outcome measures at baseline. CBT-I had significantly (p=.03) more white participants (90% CBT-I, 78% EOC). Study retention was 82% and 88% at post-test for CBT-I versus EOC, respectively. There was no difference in number of sessions attended (median=6). CBT-I sessions were somewhat longer than EOC (24.2 vs. 22.8 minutes; p=.005). Most participants in both groups at both time points gave high rankings (5+77) points) on all six treatment perception ratings (CBT-I range: 75.9-99.3%; EOC range: 69.0-97.9%). Average summed treatment perception ratings improved between Session 1 and post-test for both conditions (mean=5.9 and 6.1, respectively, for CBT-I; mean=5.6 and 5.8, respectively, for EOC).

Conclusion: The OATS EOC group was credible and acceptable to participants, resulting in equivalently high levels of participation

and retention compared to CBT-I. Findings suggest the ongoing trial has adequately controlled for nonspecific participant treatment effects that might confound interpretation of efficacy outcomes.

Support: This work was supported by PHS grant 5R01AG053221.

0470

A RANDOMIZED CONTROLLED TRIAL OF DIGITAL COGNITIVE BEHAVIORAL THERAPY FOR INSOMNIA IN PREGNANT WOMEN

Kalmbach, D. A.¹ Cuamatzi-Castelan, A.¹ Tonnu, C. V.¹ Roth, T.³ Sangha, R.³ Swanson, L. M.⁴ O'Brien, L. M.⁴ Drake, C. L.¹ ¹Henry Ford Health System, Novi, MI, ²Henry Ford Health System, Novi, MI, ³Henry Ford Health System, Detroit, MI, ⁴University of Michigan, Ann Arbor, MI.

Introduction: . Over half of pregnant women experience clinical insomnia, which is linked to perinatal depression and cognitiveemotional dysregulation. Despite high rates of prenatal insomnia and known maternal consequences, efficacious insomnia treatment options for this population are woefully scant. Early evidence from randomized controlled trials (RCTs) support the efficacy of face-to-face cognitive-behavioral therapy for insomnia (CBTI) for prenatal insomnia. Yet, as many patients are unable to access this specialist-driven care, a critical need exists to increase its accessibility. This RCT examined the efficacy digital CBTI via mobile health app in pregnant women with insomnia.

Methods: Single-site RCT. Ninety-one pregnant women (29.03±4.16 years) nearing/entering the third trimester who screened positive for clinical insomnia on the Insomnia Severity Index (ISI) were randomized to digital CBTI or digital sleep education control. Blinded assessments were performed before treatment, after treatment (during pregnancy), and 6 weeks after childbirth. The ISI, Pittsburgh Sleep Quality Index (PSQI), Edinburgh Postnatal Depression Scale (EPDS), and Pre-Sleep Arousal Scale's Cognitive factor (PSAS-C) served as study outcomes.

Results: From pre to posttreatment, CBTI patients reported reductions in ISI (-4.91 points, p<.001) and PSQI (-2.98 points, p<.001) and increases in nightly sleep duration by 32 minutes. Sleep symptoms did not change during pregnancy in the control group. After childbirth, CBTI patients, relative to controls, slept longer by 40 minutes per night (p=.01) and reported better sleep maintenance. No pre or postnatal treatment effects on depression or cognitive arousal were observed.

Conclusion: Digital CBTI improves sleep quality and increases sleep duration during pregnancy and after childbirth. Digitally delivered interventions have potential to improve the health of new and expectant mothers, but CBTI likely needs to be tailored for perinatal patients to optimize outcomes.

Support: This study was funded by the American Academy of Sleep Medicine (198-FP-18, PI: Kalmbach).

0471

24-MONTH POST MARKETING SAFETY SURVEILLANCE DATA OF A NOVEL CONTINUOUS RELEASE AND ABSORPTION MELATONIN (CRA-MELATONIN) DELIVERY SYSTEM, CONFIRMS FAVORABLE SAFETY PROFILE

Brodner, D.¹ Corsino, P.²

¹The Center for Sinus, Allergy, & Sleep Wellness, Boynton Beach, FL, ²Physician's Seal, Boca Raton, FL.

Introduction: The awareness of sleep disorders having negative health consequences, including hypertension, diabetes, obesity, depression, heart attack and stroke, has sharply escalated in recent years. Traditional treatments, including benzodiazepenes, non-benzodiazepenes, anti-depressants and non-prescription first-generation antihistamines, come with limitations in efficacy, safety and tolerability. The search for non-drug alternatives continues. The novel, well tolerated CRA-melatonin was shown in a randomized, crossover, pharmacokinetic (PK) study versus the leading marketed melatonin to achieve quick uptake and then continuous release and absorption for up to 7 hours. No safety or tolerability issues were observed. The Remfresh Safety Update at 24 months (REMSU24), a real-world safety surveillance study was conducted to confirm the previously observed safety profile of CRA-melatonin.

Methods: An independent call center with pharmacovigilancetrained health care personnel in accordance with FDA guidelines on properly reporting events, was retained to receive and record customer questions, product issues and adverse events (AEs). The study was conducted from March 9, 2017 to March 9, 2019.

Results: An estimated 320,255 patients used CRA-melatonin during the surveillance period. There were no serious AEs. The self-reporting rates of non-serious AEs were low with only 51 events recorded, a 0.016% event rate. The two most frequent AEs, headaches and nightmares are known comorbidities of insomnia. As background, there had been no treatment emergent adverse events for CRA-melatonin in the PK trial.

Conclusion: CRA-melatonin's extended 7-hour PK profile may be an effective and well-tolerated baseline therapy to improve sleep. These results confirm the favorable safety and tolerability trend observed in the PK study. In REMSU24, the scatter of reported adverse events could not be separated from what could be expected in the untreated general population.

Support: This study was supported by Physician's Seal LLC

0472

DO THE SUBJECTIVE EFFECTS OF HYPNOTIC MEDICATIONS RESULT IN UNBLINDING OF TREATMENT ASSIGNMENT IN HYPNOTIC RANDOMIZED CLINICAL TRIALS?

McCall, W. V.¹ Benca, R. M.² Rumble, M.³ Krystal, A. D.⁴ ¹Department of Psychiatry and Health Behavior, Medical College of Georgia; Augusta University, Augusta, GA, ²Department of Psychiatry and Human Behavior, University of California, Irvine, Irvine, CA, ³Department of Psychiatry, University of Wisconsin School of Medicine and Public Health, Madison, Madison, WI, ⁴Langley Porter Psychiatric Hospital and Clinics and Department of Psychiatry, University of California, San Francisco, San Francisco, CA.

Introduction: Some hypnotic medications have obvious subjective effects, including therapeutic effects (i.e., anti-insomnia effects), and side effects (i.e., feelings of impairment/intoxication). Information is lacking regarding whether the subjective effects of hypnotics result in unblinding of treatment assignment (active drug versus placebo) in hypnotic randomized clinic trials (RCTs), thus undermining internal validity of study results. In response, we now report the 'best guesses' of clinical trial participants, versus study coordinators, versus study physicians in the study Reducing Suicidal Ideation Through Insomnia Treatment (REST-IT).

Methods: Eighty-nine of the 103 participants in the REST-IT study completed their 'best guess' regarding which randomized treatment they had been assigned. REST-IT was a blinded RCT, comparing zolpidem controlled release (CR) versus placebo at bedtime given

over 8 weeks in adults with major depressive disorder who also had insomnia and suicidal ideation, and who also received open label fluoxetine. At the conclusion of study participation, the study participants, the study coordinators and the study physician each independently recorded their 'best guess' regarding which treatment arm the patient had been assigned. The study physicians and the study coordinators had access to the participants' Insomnia Severity Index scores when the 'best guess' was made.

Results: Patients guessed correctly 58.4% of the time, coordinators 53.9% of the time, and physicians 49.4% of the time. The percentage guessed correctly did not differ significantly between groups. Physicians most often guessed placebo (56.2%) while study coordinators most often guessed zolpidem-CR (55.1%). Agreement between pairs of study participants with the study coordinators and the study physician was moderately high, with all kappa values 0.49-0.57, and all kappa differences between zolpidem and placebo with p-values >0.8.

Conclusion: The blind was maintained in this RCT of zolpidem-CR versus placebo, especially for the physicians. Our results may not apply to other hypnotics or other RCT designs.

Support: NIMH MH095776, MH095780, MH95778

0473

EFFECTIVENESS AND SAFETY OF LEMBOREXANT IN SUBJECTS PREVIOUSLY TREATED WITH PLACEBO FOR 6 MONTHS IN SUNRISE-2

Yardley, J.¹ Inoue, Y.² Pinner, K.¹ Perdomo, C.³ Filippov, G.³ Kubota, N.⁴ Moline, M.³

¹Eisai Ltd., Hatfield, UNITED KINGDOM, ²Tokyo Medical University, Tokyo, JAPAN, ³Eisai Inc., Woodcliff Lake, NJ, ⁴Eisai Co., Ltd., Tokyo, JAPAN.

Introduction: In SUNRISE-2 (NCT02952820; E2006-G000-303), while lemborexant (LEM) provided significant benefit versus placebo (PBO) on sleep-diary measurements over 6mo, some improvement was noted in PBO subjects. We report outcomes from PBO subjects rerandomized to LEM during the last 6mo of SUNRISE-2. Methods: SUNRISE-2 was a randomized, double-blind, global phase 3 study in adults (≥18y) with insomnia disorder. Subjects received PBO or LEM (5mg [LEM5]; 10mg [LEM10]) for 6mo. PBO subjects were rerandomized to LEM for another 6mo; LEM subjects continued assigned treatment. Changes from 6mo baseline (calculated after PBO completion) in subjective sleep onset latency (sSOL), subjective sleep efficiency (sSE), and subjects.

Results: At study baseline for PBO subjects (n=318), median sSOL (min) was 55.9, mean (SD) sSE (%) and sWASO (min) were 61.3 (17.8) and 132.5 (80.2), respectively. The 6mo baseline values for rerandomized PBO-LEM5 (n=133) and PBO-LEM10 (n=125) subjects, respectively, were: median sSOL, 31.2, 34.3; mean (SD) sSE, 70.5 (20.2), 71.1 (18.0); mean (SD) sWASO, 105.1 (80.6), 100.1 (84.6). Median sSOL decreased from the 6mo baseline after 1mo (PBO-LEM5, -3.2; PBO-LEM10, -2.9) and 6mo (PBO-LEM5, -2.7; PBO-LEM10, -5.0). Mean (SD) sSE increased from the 6mo baseline after 1mo (PBO-LEM5, 3.9 [12.1]; PBO-LEM10, 3.5 [8.1]) and 6mo (PBO-LEM5, 3.9 [13.6]; PBO-LEM10, 4.5 [13.0]). Mean (SD) sWASO decreased after 1mo (PBO-LEM5, -8.5 [49.4]; PBO-LEM10, -5.7 [36.1]) and 6mo (PBO-LEM5, -8.2 [49.0]; PBO-LEM10, -10.0 [58.8]). Treatment-emergent adverse events incidence was similar during PBO (62.7%) and LEM treatment (PBO-LEM5, 54.9%; PBO-LEM10, 57.7%). Adverse

events were consistent with those seen in the initial 6mo of treatment for patients originally randomized to LEM.

Conclusion: Rerandomization to LEM was associated with additional improvement in sleep outcomes following the PBO-related response. LEM benefit was evident after 1mo and was sustained throughout treatment. LEM was well tolerated. **Support:** Eisai Inc.

0474

LONG-TERM EFFICACY AND SAFETY OF LEMBOREXANT IN ELDERLY ADULTS WITH INSOMNIA DISORDER: RESULTS FROM SUNRISE-2

Moline, M.¹ Inoue, Y.² Pinner, K.³ Perdomo, C.¹ Filippov, G.¹ Kubota, N.⁴ Yardley, J.³

¹Eisai Inc., Woodcliff Lake, NJ, ²Tokyo Medical University, Tokyo, JAPAN, ³Eisai Ltd., Hatfield, UNITED KINGDOM, ⁴Eisai Co., Ltd., Tokyo, JAPAN.

Introduction: In SUNRISE-2 (NCT02952820; E2006-G000-303), the dual orexin receptor antagonist lemborexant (LEM) demonstrated significant benefit versus placebo (PBO) on subjective sleep endpoints over 6mo in subjects age ≥ 18 y; benefits were sustained over 12mo. Here we present 12mo efficacy and safety data for LEM from the elderly (≥ 65 y) subgroup.

Methods: SUNRISE-2 was a 12mo, randomized, double-blind, PBO-controlled (first 6mo [Period 1]), global phase 3 study. During Period 1, subjects were randomized to PBO or LEM (5mg, [LEM5]; 10mg, [LEM10]). During Period 2 (second 6mo), LEM subjects continued their assigned dose while PBO subjects were rerandomized to LEM5 or LEM10 (not reported here). Patientreported (subjective) sleep endpoints were assessed from sleep diary data (sleep onset latency [sSOL]; sleep efficiency [sSE]; wake after sleep onset [sWASO]).

Results: Of the 949 subjects in the Full Analysis Set, 262 were age ≥65y. At 6mo, in subjects ≥65y, median sSOL significantly decreased from baseline for LEM5 (-21.7) and LEM10 (-26.0) versus PBO (-10.8; P<0.0001, P<0.01, respectively). At 12mo, LEM5 and LEM10 subjects maintained decreases in median sSOL (-29.3, -34.3, respectively). At 6mo, the mean (SD) increase from baseline in sSE was significantly larger versus PBO (8.5[13.3]) for LEM5 (16.9[13.6]; P<0.001) and LEM10 (14.9[15.9]; P<0.01). At 12mo, mean (SD) increase in sSE was maintained for LEM5 (18.1[12.5]) and LEM10 (18.0[16.8]). At 6mo, mean (SD) change from baseline in sWASO was significantly decreased versus PBO (-26.5 [52.9]) for LEM5 and LEM10 (-54.8[64.4], P<0.01; -51.4[69.3], P<0.05, respectively). At 12mo, mean (SD) decrease in sWASO was maintained for LEM5 (-58.6[46.0]) and LEM10 (-60.9[80.4]). Over 12mo, the most common (>10% either group) treatment emergent adverse events with LEM5 and LEM10, respectively, were somnolence (9.3%, 19.0%), nasopharyngitis (9.3%, 10.7%), and headache (10.5%, 6.0%). Conclusion: In elderly subjects, LEM demonstrated efficacy at 6mo, which persisted at 12mo; LEM was well tolerated. Support: Eisai Inc.

0475

REDUCING DYSFUNCTIONAL SLEEP-RELATED COGNITIONS IMPROVES NIGHTTIME SLEEP AND DAYTIME CONSEQUENCES IN OLDER ADULTS WITH INSOMNIA

Song, Y.^{1,2} Kelly, M. R.² Fung, C. H.^{1,2} Dzierzewski, J. M.³ Grinberg, A.² Mitchell, M. N.² Josephson, K.² Fiorentino, L.⁴ Martin, J. L.^{1,2} Alessi, C. A.^{1,2} ¹University of California, Los Angeles, Los Angeles, CA, ²VA Greater Los Angeles Healthcare System, Los Angeles, CA, ³Virginia Commonwealth University, Richmond, VA, ⁴University

of California, San Diego, San Diego, CA.

Introduction: The long-term impact of addressing sleep-related cognitions, which is an important component of cognitive behavioral therapy for insomnia (CBTI), has not been established, particularly in older adults. We examined whether specific changes in sleep-related cognitions predicted long-term changes in sleep and other outcomes following CBTI in older adults.

Methods: We analyzed data from a randomized controlled trial testing CBTI in older veterans with insomnia (N=159, mean age 72 years). Sleep-related cognitions were assessed with the Dysfunctional Beliefs and Attitudes about Sleep scale (DBAS, subscales: Consequences, Worry/Helplessness, Sleep Expectations, Medication). Outcome measures included the Pittsburgh Sleep Quality Index (PSQI), Insomnia Severity Index (ISI), Epworth Sleepiness Scale (ESS), sleep diary variables, Flinders Fatigue Scale (FFS), and Short Form-12 health-related quality of life (QOL). Analyses completed slope of change in DBAS subscales (baseline to post-treatment: T1) between CBTI and control with respect to slope of change in sleep and other outcomes from post-treatment to 6-months (T2).

Results: Compared to controls, the CBTI group had significantly stronger associations between improvement (T1) in DBAS-Consequences and subsequent (T2) improvement in PSQI (difference in slopes [DIS]=0.9, 95%CI=[.29, 1.43], p=0.004), ISI (DIS=1.1, 95%CI=[.18, 2.0], p=0.019), ESS (DIS=0.6, 95%CI=[.10, 1.18], p=0.020), and FFS (DIS=1.9, 95%CI=[.76, 3.09], p=0.001). The CBTI group also had significantly stronger associations between improvement in DBAS-Worry/Helplessness and subsequent improvements in PSQI, ISI, and FFS; improvement in DBAS-Medication and PSQI and ISI; and improvement in DBAS-Sleep Expectations and improved FFS. Slopes were not different between groups for sleep diary variables or QOL.

Conclusion: Significant improvements in sleep-related cognitions with CBTI across DBAS subscales in older adults predicted improvement in several outcomes of nighttime sleep and daytime consequences. These findings suggest the importance of addressing dysfunctional sleep-related cognitions for sustained improvement with CBTI in older adults

Support: The study was supported by VA Health Services, Research and Development (Alessi, IIR 08-295), National Institute on Aging (K23AG055668, Song), National Heart, Lung, and Blood Institute (K24HL 143055, Martin) of the National Institutes of Health and VA Greater Los Angeles Healthcare System, Geriatric Research, Education and Clinical Center.

0476

READINESS AND STAGES OF CHANGE IN A BEHAVIORAL SLEEP MEDICINE CLINICAL SAMPLE: FROM PRE-CONTEMPLATION TO STRUGGLING TO MAINTAIN CHANGE

Amatrudo, G. Puzino, K. Bourchtein, E. Calhoun, S. L. Fernandez-Mendoza, J. Penn State College of Medicine, Hershey, PA.

Introduction: Stages of change in the transtheoretical model are used to assess a patient's readiness to change, which may help

providers tailor behavioral treatment (BT). As research has focused on substance abuse, there is a significant lack of data in individuals presenting for behavioral sleep medicine (BSM) treatment.

Methods: 146 consecutive patients (46.1 ± 16.0 years, 61.6% female, 19.9% minority) who were evaluated at the BSM program of Penn State Hershey Sleep Research & Treatment Center completed the University of Rhode Island Change Assessment Scale (URICA) assessing readiness to change (RtC) and pre-contemplation (P), contemplation (C), preparation/action (A) and struggling to maintain (M) stages of change. Subjects also completed the Insomnia Treatment Acceptability Scale (ITAS) and Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS).

Results: The average RtC score was 9.4, with 21.9% of the sample in stage P, 56.2% in C and 21.9% in A. The average P (53.2 ± 7.6), C (49.1 ± 10.1), A (48.8 ± 12.3) and M (37.7 ± 9.1) scores suggested an overall "contemplation cluster profile". Higher RtC (r=0.37), C (r=0.31) and A (r=0.24) scores were associated with greater acceptability of BT, while higher P scores were associated with lower acceptability of BT (r=-0.22). In contrast, higher M scores were associated with greater acceptability of pharmacological treatment (r=0.21) as well as higher DBAS scores, including sleep medication expectations (r=0.23).

Conclusion: Patients attending a BSM program typically present at a contemplation stage, which indicates intention to start changing sleep behaviors within the next few months with some ambivalence in regards to pros/cons of such change. A significant proportion of patients struggle to maintain change and present with higher acceptability for pharmacological interventions, which may hinder the effectiveness of BT and may require specific therapeutic approaches.

Support: Department of Psychiatry, Penn State College of Medicine

0477

CHARACTERISTICS OF INSOMNIA SUBJECTS SCREENED FOR TRANSITIONING FROM ZOLPIDEM TARTRATE TO LEMBOREXANT IN A MULTICENTER PILOT STUDY

*Ahmad, M.*¹ *Malhotra, M.*² *Amchin, J.*² *Kumar, D.*² *Perdomo, C.*² *Moline, M.*²

¹Clinilabs Drug Development Corporation, New York, NY, ²Eisai Inc., Woodcliff Lake, NJ.

Introduction: Patients who take insomnia medication may change medications for reasons including lack of efficacy, adverse events, and dependence concerns. A pilot study (NCT04009577, E2006-A001-312) assessed a dosing approach for transitioning patients from zolpidem tartrate (ZOL; immediate or extended-release) to lemborexant, a dual orexin receptor antagonist. Here we describe characteristics of subjects who entered the study Screening Period and their reasons for wanting to change medications.

Methods: This multicenter pilot study was conducted in the U.S. and enrolled subjects age $\geq 18y$ with insomnia diagnosed per DSM-5 criteria, and who used ZOL (self-reported intermittently [3-4 nights/ week] or frequently [≥ 5 nights/week]) as their only insomnia treatment. Subjects entered a 3-week Screening Period, during which frequency/ dose of ZOL taken was recorded; subjects also wore an actigraph continuously. Eligible subjects thereafter entered the Treatment Phase to determine lemborexant dosing (5 or 10mg during a 2-week Titration Period with assignment to 1 of 3 treatment schedules based on ZOL usage frequency during Screening), followed by a 12-week Extension (Maintenance) Phase and a 4-week Follow-up Period. **Results:** Forty-nine subjects entered the Screening period and completed the Chief Complaint Form through November 2019; mean(SD) age was 57.1(13.8)y, 67.3% were female, 69.4% were white, and 28.6% were black. 31 subjects reported using ZOL frequently and 15 reported using ZOL intermittently (3 missing). The most common sleep complaint was waking up too early (n=33), followed by difficulty staying asleep (n=13), and difficulty falling asleep (n=3). Reasons for wanting to switch from ZOL included: ZOL not working (n=19), concerns about taking ZOL (n=14), wanting to try something new/potentially better (n=6), side effects (n=5), and residual daytime sleepiness (n=4). 43/49 subjects completed screening through this period.

Conclusion: This study offers the opportunity to understand patients' current use of insomnia medication and their motivation for wanting to change insomnia medications.

Support: Eisai Inc.

0478

A MULTICENTER PILOT STUDY TO EVALUATE NEXT-DOSE TRANSITION FROM ZOLPIDEM TO LEMBOREXANT FOR THE TREATMENT OF INSOMNIA

Rosenberg, R.¹ Amchin, J.² Kumar, D.² Perdomo, C.² Moline, M.² Malhotra, M.²

¹NeuroTrials Research Inc., Atlanta, GA, ²Eisai Inc., Woodcliff Lake, NJ.

Introduction: Switching of medications for insomnia occurs often in clinical practice based on a variety of reasons. However, few clinical studies have examined methods for transitioning patients between different insomnia medications. This is especially important to consider when the classes of drugs are different (e.g., GABA-ergic agonism vs orexin receptor antagonism); thus, clinical guidance would be valuable. The safety and efficacy of the dual orexin receptor antagonist lemborexant (LEM) for the treatment of insomnia was confirmed in two Phase 3 studies, SUNRISE-1 (NCT02783729, E2006-G000-304) and SUNRISE-2 (NCT02952820, E2006-G000-303). This pilot study (NCT04009577, E2006-A001-312) was designed to assess pre-specified dosing approaches for directly transitioning from the sedative-hypnotic zolpidem (ZOL), a commonly prescribed sleep aid, to LEM.

Methods: This multicenter pilot study has enrolled subjects age \geq 18 years with an insomnia diagnosis (DSM-5 criteria), who used ZOL (intermittently or frequently) as their only insomnia treatment. Following a 3-week Screening period, eligible subjects enter the Treatment Phase (2-week titration period: assigned to 1 of 3 treatment arms based on ZOL use during Screening), and then the Extension Phase (maintenance period up to 12 weeks). During both the Treatment and Extension Phases, the dose of LEM is flexible between 5 and 10 mg, depending on efficacy and tolerability. The primary endpoint is to evaluate the proportion of subjects taking ZOL who successfully transition to LEM (lemborexant 5 mg [LEM5] or lemborexant 10 mg [LEM10]) after 2 weeks of LEM treatment.

Results: Enrollment began July 15, 2019. It was initially projected that approximately 110 subjects would be screened to provide about 60 subjects for randomization across 3 treatment arms. Interim data will be presented (planned cutoff date Jan 08, 2020).

Conclusion: This pilot study will help inform on dosing guidance when transitioning a patient from a GABA-ergic drug to an orexin receptor antagonist.

Support: Eisai Inc.

0479

SLEEP ONSET AND SLEEP MAINTENANCE RESPONDER PROFILES OVER 12 MONTHS OF TREATMENT WITH LEMBOREXANT: RESULTS FROM SUNRISE-2

Zammit, G.¹ Yardley, J.² Pinner, K.² Moline, M.³

¹Clinilabs Drug Development Corporation, New York, NY, ²Eisai Ltd., Hatfield, UNITED KINGDOM, ³Eisai Inc., Woodcliff Lake, NJ.

Introduction: In addition to analyzing treatment response by comparing change from baseline (CFB) between active treatment and placebo (PBO), it is also informative to evaluate the percent of patients with a given magnitude of response. This can help establish the expected degree of improvement for patients, thereby helping to determine treatment success.

Methods: SUNRISE-2 (NCT02952820; E2006-G000-303) was a randomized, double-blind, global phase 3 study of lemborexant (LEM) in adults (\geq 18y) with insomnia disorder. Subjects received PBO or LEM (5mg [LEM5]; 10mg [LEM10]) for 6mo. LEM subjects continued their original dose, while PBO subjects were rerandomized to LEM for another 6mo (not reported here). Responder profiles were constructed separately for patient-reported, sleep diary-based subjective sleep onset and sleep maintenance based on the cumulative proportion of subjects with CFBs in 10-minute increments for subjective sleep onset latency (sSOL) or subjective wake after sleep onset (sWASO), respectively. The proportion was based on number of subjects with baseline data (denominator) and data available at time of visit; study dropouts were considered nonresponders.

Results: Baseline values were similar (median sSOL [min]: PBO, 55.9; LEM5, 53.6; LEM10, 55.7; mean[SD] sWASO [min]: PBO, 132.5[80.2]; LEM5, 132.8[82.5]; LEM10, 136.8[87.4]). At 6mo, a higher percentage of subjects with CFB of \geq 20min in sSOL was observed with LEM versus PBO (PBO, 30.4%; LEM5, 45.5%; LEM10, 44.9%). At 12mo, a similar percentage of responders with a \geq 20min CFB in sSOL was observed for LEM (LEM5, 40.4%; LEM10, 43.3%). A higher percentage of subjects with a CFB in sWASO of \geq 60min was observed for LEM versus PBO at 6mo (PBO, 24.2%; LEM5, 27.8%: LEM10, 30.2%); similar percentages were observed at 12mo with LEM (LEM5, 27.8%; LEM10, 27.7%). The majority of treatment-emergent adverse events were mild/moderate.

Conclusion: LEM treatment provided important levels of sustained efficacy over the long term. LEM was well tolerated. **Support:** Eisai Inc.

0480

EFFICACY AND SAFETY OF LEMBOREXANT IN FEMALE SUBJECTS OF PERIMENOPAUSAL AGE WITH INSOMNIA DISORDER

Cheng, J.¹ Yardley, J.² Pinner, K.² Moline, M.¹ ¹Eisai Inc., Woodcliff Lake, NJ, ²Eisai Ltd., Hatfield, UNITED KINGDOM.

Introduction: Insomnia is common in women in the perimenopausal age range. We present 12-month efficacy and safety data for the subgroup of female subjects in the age range associated with perimenopause (age 40-58y) from SUNRISE-2 (NCT02952820; E2006-G000-303).

Methods: SUNRISE-2 was a 12-month, randomized, double-blind, placebo (PBO)-controlled (first 6mo [Period 1]), global phase 3 study. During Period 1, subjects received PBO or lemborexant

(LEM: 5mg, [LEM5]; 10mg, [LEM10]). During Period 2 (second 6mo), LEM subjects continued their assigned dose. PBO subjects were rerandomized to LEM5 or LEM10 (not reported here). Changes from baseline in subjective sleep parameters: subjective sleep onset latency (sSOL), sleep efficiency (sSE), wake after sleep onset (sWASO) were assessed from sleep diary data.

Results: The perimenopausal subgroup comprised 280 subjects (Period 1: PBO, n=90; LEM5, n=82; LEM10, n=108). Baseline median sSOL (min) was 57.5, 51.1, and 54.0 for PBO, LEM5, and LEM10. Greater median decreases from baseline in sSOL (min) occurred at 6mo with LEM vs PBO (PBO, -17.9; LEM5, -20.7; LEM10, -30.4); improvements persisted at 12mo (LEM 5, -27.7; LEM10, -33.9). Baseline mean[SD] sSE was 59.9%[16.6%], 60.7%[20.0%], and 61.2%[17.5%] for PBO, LEM5, and LEM10. Greater mean[SD] increases from baseline in sSE (%) occurred at 6mo with LEM vs PBO (PBO, 12.5%[15.0%]; LEM5, 15.9%[17.0%]; LEM10, 17.2%[14.7%]); improvement persisted at 12mo (LEM5,17.6%[18.2%]; LEM10, 19.1%[14.8%]). Baseline mean[SD] sWASO (min) was 134.9[70.8], 142.4[86.5], and 136.5[84.4]. Greater mean[SD] decreases from baseline in sWASO (min) occurred at 6mo with LEM vs PBO (PBO, -37.0[59.6]; LEM5, -50.1[74.5]; LEM10 -54.5[65.4]); improvement persisted at 12mo (LEM5, -59.1[76.7]; LEM10, -66.2[64.9]). Most treatment emergent adverse events (TEAEs) were mild/moderate.

Conclusion: Consistent with previously reported data from the total population, subjective sleep parameters improved, and improvement was sustained over time in perimenopausal women. LEM was well tolerated, supporting LEM as a potential treatment option for perimenopausal women with insomnia. **Support:** Eisai Inc.

0481

IMPACT OF LEMBOREXANT ON FATIGUE SEVERITY IN SUBJECTS WITH CLINICALLY SIGNIFICANT LEVELS OF FATIGUE AT BASELINE

Rosenberg, R.¹ Kumar, D.² Pinner, K.³ Perdomo, C.² Moline, M.² ¹NeuroTrials Research Inc., Atlanta, GA, ²Eisai Inc., Woodcliff Lake, NJ, ³Eisai Ltd., Hatfield, UNITED KINGDOM.

Introduction: In SUNRISE-1 (NCT02783729; Phase 3 E2006-G000-304) and **SUNRISE-2** (NCT02952820; E2006-G000-303), lemborexant (LEM) provided significant benefit versus placebo on sleep diary-based sleep onset/maintenance outcomes over 1mo and 6mo, respectively, in subjects with insomnia disorder. The impact of LEM on patient-reported fatigue, assessed using the Fatigue Severity Scale (FSS), in subjects with clinically significant fatigue (CSF) at baseline was examined for each study. Methods: SUNRISE-1 was a 1mo, randomized, double-blind, placebo- and active-controlled, parallel-group study in female (\geq 55y) and male (≥65y) subjects (n=1006); subjects received placebo, LEM 5mg (LEM5), LEM 10mg (LEM10) or zolpidem tartrate extended-release (not reported here). SUNRISE-2 was a 12mo, randomized, double-blind study in subjects age $\geq 18y$ (n=949). Subjects received placebo, LEM5, or LEM10 for 6mo. Placebo subjects were rerandomized to LEM5 or LEM10 for another 6mo; LEM subjects continued assigned treatment. CSF was defined as FSS total score (FSSts) \geq 36.

Results: In subjects with baseline CSF, in SUNRISE-1, baseline FSSts was 46.8, 46.5, and 46.6 in placebo (n=117), LEM5 (n=157), and LEM10 (n=153) groups, respectively, and, in SUNRISE-2, was 45.7, 46.4, and 45.8 in placebo (n=169), LEM5 (n=181), and LEM10 (n=173) groups, respectively. At 1mo, mean changes

from baseline in FSSts were not significantly different vs placebo for LEM5 in both studies, and for LEM10 in SUNRISE-1. In SUNRISE-2, LEM10 significantly decreased mean [SD] FSSts from baseline vs placebo at 1mo (LEM10, -11.2[13.9] vs placebo, -8.7[10.5]; *P*=0.03). By 6mo in SUNRISE-2, both LEM5 and LEM10 significantly decreased mean [SD] FSSts from baseline versus placebo (LEM5, -15.4[13.8]; LEM10, -15.0[14.2] vs placebo, -11.2[11.6]; both *P*<0.05). At 12mo, mean [SD] FSSts improvements were sustained for LEM5 (-20.4[12.8]) and LEM10 (-18.1[14.7]).

Conclusion: In subjects with CSF, longer treatments (>1mo) may be needed to observe significant FSSts improvements, which were evident at 6mo and sustained at 12mo with continuous LEM treatment.

Support: Eisai Inc.

0482

A PRELIMINARY STUDY ON THE EFFICACY OF FOREHEAD-COOLING FOR RELIEVING MENOPAUSAL SLEEP DIFFICULTIES AND HOT FLASHES

Baker, F. C.¹ de Zambotti, M.¹ Chiappetta, L.² Nofzinger, E.³ ¹SRI International, Menlo Park, CA, ²DataDIVA, Pittsburgh, PA, ³Ebb Therapeutics, Pittsburgh, PA.

Introduction: Many women experience sleep difficulties in the approach to menopause and post-menopause, with about 25% experiencing severe symptoms that impact daytime functioning and quality of life. Hot flashes contribute to these sleep difficulties, being associated with nocturnal awakenings, poorer sleep quality, and chronic insomnia. New non-pharmacological sleep solutions have become available, including a forehead cooling device designed to target elevated brain metabolism in insomnia sufferers. Here, we explored whether this device was effective in improving subjective sleep and hot flashes in menopausal-age women with insomnia symptoms.

Methods: This study was an open-label, in-home investigation of the efficacy of nightly treatment with a forehead cooling device in 20 women (55.1 \pm 4.2 years) with insomnia symptoms and daily hot flashes. Participants completed daily diaries assessing sleep quality and hot flashes across a baseline week (no treatment) followed by 4 weeks of treatment. They also completed questionnaires before and after treatment including the insomnia severity index and the hot flash related daily interference scale.

Results: Women reported better sleep quality with a shorter sleep onset latency and fewer awakenings (between 14-30% improvement) during the first week of device use, with further improvements over time, relative to baseline (p < 0.001). Women also reported fewer nocturnal hot flashes that were less severe during treatment (p<0.001). They had lower insomnia severity scores post-treatment (9.3 ± 5.8) compared to pre-treatment (20.0 ± 5.7) (p<0.001), with 17 participants showing a reduction of 6 points or greater on the insomnia severity index. There was also a significant reduction in hot flash related daily interference post-treatment (p<0.001).

Conclusion: Use of a forehead cooling device during the night improved subjective sleep quality and reduced insomnia symptoms and hot flash frequency and severity in this preliminary study of menopausal-age women. Further large scale randomized controlled trials are required to determine efficacy.

Support: Ebb Therapeutics

0483

PREDICTORS OF COGNITIVE BEHAVIORAL THERAPY FOR INSOMNIA (CBT) OUTCOMES IN ACTIVE DUTY U.S. ARMY PERSONNEL

Pruiksma, K. E.¹ Hale, W.¹ Mintz, J.¹ Peterson, A.¹ Young-McCaughan, S.¹ Wilkerson, A.³ Nicholson, K.⁴ Dondanville, K.¹ Fina, B.¹ Borah, E.⁵ Roache, J.¹ Litz, B. T.⁶ Bryan, C.⁷ Taylor, D. J.⁸ ¹University of Texas Health Science Center at San Antonio, San Antonio, TX, ²University of Texas Health Science Center at San Antonio, San Antonio, TX, ³Medical University of South Carolina, Charleston, SC, ⁴Carl R Darnall Army Medical Center, Fort Hood, TX, ⁵University of Texas at Austin, Austin, TX, ⁶Massachusetts Veterans Epidemiology Research and Information Center, Boston, MA, ⁷University of Utah, Salt Lake City, UT, ⁸University of Arizona, Tucson, AZ.

Introduction: Cognitive behavioral therapy for insomnia (CBTi) is well established as the first-line treatment for the management of chronic insomnia. Identifying predictors of response to CBTi should enable the field to efficiently utilize resources to treat those who are likely to respond and to personalize treatment approaches to optimize outcomes for those who are less likely to respond to traditional CBTi. Although a range of studies have been conducted, no clear pattern of predictors of response to CBTi has emerged.

Methods: The purpose of this study was to examine the impact and relative importance of a comprehensive group of pretreatment predictors of insomnia outcomes in 99 active duty service members who received in-person CBTi in a randomized clinical trial.

Results: Results indicated that higher levels of baseline insomnia severity and total sleep time predicted greater improvements on the Insomnia Severity Index (ISI) following treatment. Higher depression symptoms and a history of head injury predicted a worse response to treatment (i.e., smaller improvements on the ISI).

Conclusion: Clinically meaningful improvements, as measured by the reliable change index (RCI), were found in 59% of the sample. Over and above baseline insomnia severity, only depressive symptoms predicted this outcome. Future studies should examine if modifications to CBTi based on these predictors of response can improve outcomes.

Support: This study was conducted with support from the U.S. Department of Defense through the U.S. Army Medical Research and Materiel Command, Congressionally Directed Medical Research Programs, Psychological Health and Traumatic Brain Injury Research Program award W81XWH-10-1-0828 (PI: Dr Taylor).

0484

HOW MUCH IMPROVEMENT IN SUBJECT-REPORTED SLEEP ONSET LATENCY IS NEEDED FOR PATIENTS TO REPORT A POSITIVE IMPACT OF THEIR INSOMNIA MEDICATION?

Drake, C.¹ Yardley, J.² Pinner, K.² Perdomo, C.³ Kumar, D.³ Moline, M.³

¹Sleep Disorders and Research Center, Henry Ford Health System, Detroit, MI, ²Eisai Ltd., Hatfield, UNITED KINGDOM, ³Eisai Inc., Woodcliff Lake, NJ.

Introduction: How much improvement would be considered meaningful from the patient perspective is not well defined. In

SUNRISE-2 (NCT02952820; E2006-G000-303), using the Patient Global Impression-Insomnia version (PGI-I), subjects rated how treatment impacted subjective (sleep diary-based) sleep onset latency (sSOL; Question2; positive, neutral, and negative) relative to before starting treatment. Meaningful change can thus be determined based on the change from baseline (CFB) in subjects with a positive score.

Methods: SUNRISE-2 (n=949, full analysis set) was a Phase 3, 12-month, double-blind, global study in subjects age \geq 18y with insomnia disorder. Subjects received PBO (N=318), LEM 5mg (LEM5, N=316) or LEM 10mg (LEM10, N=315) for 6 months. At the end of Month 6, PBO subjects were rerandomized to LEM5 or LEM10; LEM5 and LEM10 subjects continued at the same dose for 6 more months. The ranges of median CFB in sSOL (minutes) at 6 months were examined in response to PGI-I Item 2.

Results: At 6 months, subjects reporting positive medication effect (PBO, n=110; LEM5, n=178; LEM10, n=159) showed median CFB in sSOL from -17.5 to -32.1 minutes across treatment groups. In subjects reporting neutral effect (PBO, n=49; LEM5, n=28; LEM10, n=27), median CFB in sSOL ranged from -10.4 to -25.6 minutes across treatment groups. In subjects reporting negative medication effect (PBO, n=82; LEM5, n=34; LEM10, n=32), median CFB in sSOL ranged from -8.6 to -10.4 minutes across treatment groups. The PBO group provided the smallest numbers for each response category range.

Conclusion: Subjects reporting positive medication effect on PGI-I Item 2, i.e. decreased time to fall asleep, had corresponding changes from baseline in sSOL ranging from -17.5 to -32.1 minutes. Thus, this range may represent a clinically meaningful improvement as perceived by patient-subjects, and may be useful to clinicians in determining whether a treatment regimen is working for their patients.

Support: Eisai Inc.

0485

EXPERIENCE AND ATTITUDES ABOUT PRESCRIPTION INSOMNIA MEDICATIONS: RESULTS FROM AN ONLINE SURVEY OF INDIVIDUALS WITH SLEEPING DIFFICULTIES AND INSOMNIA

Sheehan, D.¹ Zee, P. C.² Steinberg, K.³ Ginovker, A.³ Atkins, Jr., N.⁴ Moline, M.⁴

¹University of South Florida Morsani College of Medicine, Tampa, FL, ²Northwestern University Feinberg School of Medicine, Chicago, IL, ³The Harris Poll, Rochester, NY, ⁴Eisai Inc., Woodcliff Lake, NJ.

Introduction: This survey explored several topics related to living with insomnia/sleeping difficulties. Reported here are patient experience, attitudes, and perspectives about and understanding of prescription medications for insomnia.

Methods: The online survey was conducted by The Harris Poll in the U.S. between February 14 and March 8, 2019. Survey respondents ("patients") were adults age $\geq 18y$ who had been diagnosed with insomnia (11% of respondents), or had experienced sleeping difficulties (defined as difficulty falling asleep or staying asleep for ≥ 3 nights/week for ≥ 3 months; 89% of respondents). Raw survey data were weighted by relevant factors to be representative of the total U.S. adult population with insomnia/sleeping difficulties.

Results: Among 525 patients (mean age 46y; 55% female) who completed the survey, 83 were currently using prescription medication, 45 used prescription medication previously, and 397 had

no prescription medication history. The majority of all patients "somewhat" or "strongly" agreed they were "concerned about the safety risks of sleep medications currently available by prescription" (79%); felt "there have got to be better medications that help people sleep" (74%); and that they "wish there were more medications to choose from" (67%). Within the group of respondents with current/past prescription history (n=128), 63%, 23%, and 14% had tried 1-2, 3-4, or \geq 5 different prescription medications, respectively. Among reasons for missing/skipping a dose, ~20% of respondents with current/past prescription history selected for each response that they "do not feel my medication is effective"; "do not like the way my medication makes me feel when I wake up the next morning"; and "prefer not to take my medication every night unless absolutely necessary."

Conclusion: Results from this online survey provide insights into patient attitudes toward pharmacotherapy and indicate that a significant number of insomnia patients feel dissatisfied with medication treatment options, including concerns regarding safety and side effects.

Support: Eisai Inc.

0486

IMPACT OF INTRINSIC FACTORS ON EFFICACY OF LEMBOREXANT: SUBGROUP ANALYSES OF SUNRISE-2

*Moline, M.*¹ *Inoue, Y.*² *Kubota, N.*³ *Pinner, K.*⁴ *Perdomo, C.*¹ *Yardley, J.*⁴

¹Eisai Inc., Woodcliff Lake, NJ, ²Tokyo Medical University, Tokyo, JAPAN, ³Eisai Co., Ltd., Tokyo, JAPAN, ⁴Eisai Ltd., Hatfield, UNITED KINGDOM.

Introduction: Lemborexant (LEM), a dual orexin receptor antagonist, demonstrated significant benefits vs placebo on patient-reported sleep outcomes in adults age $\geq 18y$ in SUNRISE-2 (NCT02952820; E2006-G000-303). The impact of intrinsic factors (sex, race, and region) on LEM efficacy outcomes was assessed.

Methods: SUNRISE-2 was a randomized, double-blind, global phase 3 study in adults age \geq 18y with insomnia disorder (Full Analysis Set, n=949). Subjects received placebo (n=318) or LEM (5mg [LEM5], n=316; 10mg [LEM10]; n=315) for 6 months. At 6 months, placebo subjects were rerandomized to LEM for another 6 months (not reported here); LEM subjects remained on their assigned dose. Sleep diary-based (subjective) sleep onset latency (sSOL) and wake after sleep onset (sWASO) were assessed for prespecified patient subgroups including: sex (male [n=302], female [n=647]), race (white [n=679], black [n=76], Asian [n=178]), and region (North America [n=302], Europe/New Zealand [n=483], Asia [n=164]). Analyses were not controlled for multiplicity.

Results: LEM5 and LEM10 provided numerically greater median reductions (improvement) from baseline in sSOL vs placebo at 6 months in across all subgroups examined. Also, LEM5 and LEM10 led to mean reductions (improvement) from baseline at 6 months in sWASO for all subgroups. While several subgroups had small numbers of subjects, changes from baseline in sSOL and sWASO were in the direction of improvement in the majority of subgroups. Pharmacokinetic analyses showed no important differences in exposure by these factors.

Conclusion: LEM treatment demonstrated efficacy in improving sSOL and sWASO across patient subgroups, supporting common dosing instructions for both sexes and all races. **Support:** Eisai Inc.

0487

EFFECTS OF SUVOREXANT ON SLEEP ARCHITECTURE IN PATIENTS WITH ALZHEIMER'S DISEASE AND INSOMNIA

Svetnik, V.¹ Wang, T.¹ Ceesay, P.¹ Ceren, O.¹ Snyder, E.¹ Bliwise, D.² Budd, K.¹ Hutzelmann, J.¹ Stevens, J.¹ Lines, C.¹ Michelson, D.¹ Herring, W.¹

¹Merck & Co., Inc., Kenilworth, NJ, ²Emory University School of Medicine, Atlanta, GA.

Introduction: Suvorexant, an orexin receptor antagonist that enables sleep to occur via competitive antagonism of wake-promoting orexins, improved total sleep time (TST) in a sleep laboratory polysomnography (PSG) study of patients with AD and insomnia. Here we report on the effects of suvorexant on sleep architecture in the study.

Methods: This was a randomized, double-blind, 4-week trial (ClinicalTrials.gov NCT02750306). Participants who met diagnostic criteria for both probable AD dementia (of mild to moderate severity) and insomnia were randomized to suvorexant 10mg (could be increased to 20mg based on clinical response) or matching placebo. Overnight sleep laboratory PSG was performed on 3 nights: screening, baseline, and Night-29 (last night of dosing). Suvorexant differences from placebo in changes-frombaseline at Night-29 for sleep architecture were analyzed as exploratory endpoints.

Results: A total of 274 participants were included in the analysis (suvorexant N=135, placebo N=139). At Night-29, suvorexant improved TST by 28 minutes versus placebo (p=0.001). There were no significant differences between suvorexant and placebo in the % of TST spent in REM (1.3%, 95% CI: -0.5, 3.0), N1 (0.6%, 95% CI: -1.2, 2.5), N2 (-1.0%, 95% CI: -3.2, 1.2), or N3 (-0.6%, 95% CI: -1.8, 0.6). There was no significant difference between suvorexant and placebo in latency to REM (-5.4 minutes, 95% CI: -2.3.4, 12.7). **Conclusion:** Suvorexant improves TST without altering the underlying sleep architecture in AD patients with insomnia. **Support:** Merck Sharp & Dohme Corp., a subsidiary of Merck &

Support: Merck Sharp & Dohme Corp., a subsidiary of Merck & Co., Inc., Kenilworth, NJ, USA

0488

PILOT EVALUATION OF AN ACTIGRAPHY WATCH COMPARED TO POLYSOMNOGRAPHY IN A CLINICAL TRIAL OF SUVOREXANT FOR TREATING INSOMNIA IN PATIENTS WITH ALZHEIMER'S DISEASE

Svetnik, V.¹ Wang, T.¹ Ceesay, P.¹ Snyder, E.¹ Ceren, O.¹ Bliwise, D.² Budd, K.¹ Hutzelmann, J.¹ Stevens, J.¹ Lines, C.¹ Michelson, D.¹ Herring, W.¹

¹Merck & Co., Inc., Kenilworth, NJ, ²Emory University School of Medicine, Atlanta, GA.

Introduction: Suvorexant, an orexin receptor antagonist, improved total sleep time (TST) in a sleep laboratory polysomnography (PSG) study of patients with Alzheimer's disease (AD) and insomnia. The study included a pilot evaluation of an actigraphy watch for continuously recording patient's sleep and daytime activity. We report on the utility of the watch for assessing sleep in relation to gold-standard PSG.

Methods: This was a randomized, double-blind, 4-week trial (ClinicalTrials.gov NCT02750306). Participants who met diagnostic criteria for both probable AD dementia and insomnia were randomized to suvorexant 10-20mg or placebo. Overnight sleep laboratory PSG was performed on 3 nights: screening, baseline, and

Night-29 (last dose). An actigraphy watch (Garmin vivosmart® HR) was worn continuously by the patient. Separate analyses were performed for PSG and watch. We compared treatment effects on change-from-baseline in PSG-TST at Night-29 and WATCH-TST at Week-4 (average TST per night over Week-4). We also analyzed Night-29 data only with watch data restricted to the PSG recording time.

Results: A total of 274 participants were included in the Night-29 PSG analysis (suvorexant=135, placebo=139) and 223 in the Week-4 watch analysis (suvorexant=113, placebo=110). Suvorexant improved Night-29 PSG-TST by 28 minutes versus placebo (p=0.001) and Week-4 WATCH-TST by 17 minutes versus placebo (p=0.144). In the subgroup who had usable data for both assessments at Night-29 (suvorexant=57, placebo=50), the watch overestimated TST compared to PSG (e.g. placebo baseline scores = 412 minutes for WATCH-TST and 265 minutes for PSG-TST) and underestimated change-from-baseline treatment effects: the suvorexant versus placebo difference was 35 minutes for PSG-TST (p=0.057) and 20 minutes for WATCH-TST (p=0.405).

Conclusion: The watch was less sensitive than PSG for evaluating treatment effects on TST. However, results obtained with the watch were directionally similar to PSG in indicating a benefit of suvorexant versus placebo for improving TST in AD patients with insomnia.

Support: Merck Sharp & Dohme Corp., a subsidiary of Merck & Co., Inc., Kenilworth, NJ, USA

0489

INITIAL RESULTS FROM THE HYPERTENSION WITH UNSATISFACTORY SLEEP HEALTH (HUSH) CLINICAL TRIAL FOR PRIMARY CARE PATIENTS WITH INSOMNIA AND COMORBID HYPERTENSION (INS-HTN)

Buysse, D. J.¹ Ritterband, L. M.² Yabes, J. G.¹ Rollman, B. L.¹ Strollo, P. J.¹ Smith, K. J.¹ Patterson, C. M.¹

¹University of Pittsburgh, Pittsburgh, PA, ²University of Virginia, Charlottesville, VA.

Introduction: Insomnia is commonly comorbid with, and may contribute to, hypertension. Cognitive-behavioral treatments improve insomnia, but their effects on hypertension are uncertain, and they are often unavailable in primary care practices, where most INS-HTN patients are treated. We evaluated the efficacy of Brief Behavioral Treatment for Insomnia (BBTI) and Sleep Healthy Using the Internet (SHUTi) compared to enhanced usual care (EUC) on insomnia and home blood pressure (HBP) in primary care patients with INS-HTN.

Methods: Patients were recruited via electronic health records from 67 primary care practices and randomized 2:2:1 to BBTI delivered via telephone/videoconferencing; SHUTi, an automated, web-based CBT-I program; or EUC including a patient education video. Assessments included self-report questionnaires, home sleep apnea testing, and one week of sleep diary and HBP, measured at Baseline and 9 weeks/ 6 months post-treatment. The primary outcome was the Patient-Reported Outcomes Measurement Information System (PROMIS) Sleep Disturbance scale. Linear mixed models were fitted for continuous variables on the intent-totreat sample (n=548), adjusting for age and sex. Chi-square tests were used for proportions.

Results: Patients were 61.8 ± 11.3 years old, 67.2% female, and 55.9% were taking hypnotics. Insomnia Severity Index (ISI) was 15.4 ± 4.4 , Apnea-Hypopnea Index 9.8 ± 11.4 , and HBP $130\pm14/81\pm9$. BBTI and SHUTi were significantly better than EUC (p<.002) at 9 weeks

and 6 months on PROMIS Sleep Disturbance and Sleep-Related Impairment scales, ISI, and diary sleep efficiency, but had inconsistent effects on PROMIS depression and anxiety scales (p=0.001-0.9). Greater proportions of BBTI and SHUTi vs. EUC-treated patients had 9-week and 6-month ISI scores <8 (p=.01, p=.04) and ISI changes scores \geq 7 (p=.002, p=.003). HBP did not significantly differ by intervention group.

Conclusion: BBTI and SHUTi improved insomnia, but did not reduce HBP in patients with INS-HTN. These interventions appear suitable for dissemination and implementation in primary care, but may have limited effects on comorbid symptoms and conditions. **Support:** NHLBI UH2/UH3 HL125103

0490

DEVELOPMENT AND PRELIMINARY EVALUATION OF A BRIEF INTERVENTION FOR POST-DEPLOYMENT INSOMNIA

Mellman, T. Howell, M.

Howard University, Washington, DC.

Introduction: Sleep disturbances are common among previously deployed Veterans. Cognitive behavioral therapy for insomnia (CBT-I) has shown promise for Veterans but there are limitations and CBT-I may not optimally target the nocturnal vigilance conditioned by threatening environments. We developed and preliminarily evaluated a brief educational/behavioral intervention that combined established behavioral sleep principles with cognitive exercises intended to reduce the impact of vigilant thoughts and behaviors on sleep.

Methods: Participants were 40 Veterans recruited from the community and local VAMC who had been deployed to combat zones or hazardous duty areas. The mean age was 40, and the majority were male, African American, and screened positively for PTSD. They were assigned to the study intervention or an educational control that utilized a pamphlet promoting healthy sleep habits. Assignment at a 2:1 ratio was intended to allow for refinement of the study intervention during the early phase of the trial. Initial assessment was followed by a week of sleep diary collection, then two intervention sessions a week apart, followed by a repeat assessment. Participants of the study intervention were assessed again 3-months later.

Results: Improvements in sleep efficiency, ratings for feeling rested in the morning, and reduced Insomnia Severity Index scores were seen in the post-treatment week but did not differ between groups. At 3-months, these improvements were sustained in the 14 retained participants of the study intervention, engagement in vigilance reducing exercises remained high, and ratings of how rested one felt in the morning were significantly improved over ratings from the post-treatment week. **Conclusion:** Brief behavioral intervention for post-deployment insomnia can provide benefits and cognitive exercises to reduce the impact of vigilant thoughts and behaviors warrant further evaluation.

Support: Supported by W81XWH-14-1-0066 from the Congressionally Directed Peer-Reviewed Medical Research Program of the Department of Defense

0491

CHANGES IN INSOMNIA SYMPTOMS ARE ASSOCIATED WITH IMPROVEMENTS IN CHRONIC PAIN

Ito, K.¹ Kadotani, H.²

¹Department of Anesthesiology, Shiga university of medical science, Otsu, JAPAN, ²Department of Sleep and Behavioal Sciences, Shiga University of Medical Science, Otsu, JAPAN. **Introduction:** Depression and anxiety is known to associated with insomnia. Chronic pain is also well known to be associated with insomnia. However, relationship among insomnia and anxiety and depression is not well understood. Here We conducted a retrospective cohort study whether improvement insomnia could affect chronic pain release.

Methods: Patients with chronic pain suffering for more than three months, who first visited in our pain management clinic in Shiga University of Medical Science, Otsu, Japan during 09/25/2013-01/26/2017, were included in this study. Patients were asked to complete the same questionnaire at their first visit and six-month later. We designate pain release as 30% and over improvement in numerical rating scale (NRS) for pain release. We define improvement insomnia as subjects with Athene insomnia scale (AIS) ≥ 6 at the first visit became AIS <6 six-month later. We also evaluated patients' psychotic conditions by Questionnaires for anxiety and depression: HADS (Anxiety and depression) and PCS (Pain Catastrophizing score) for negative cognition, respectively. We conducted logistic regression analysis as for dependent variable was pain release.

Results: Characteristics data: n=47, mean age: 53.53 ± 15.45 , male rate: 45%, BMI: 22.81 ± 3.49 , NRS: 5.45 ± 1.85 , AIS: 8.94 ± 2.73 , HADSA: 6.04 ± 2.57 , HADSD: 6.06 ± 2.64 , PCS: 30.70 ± 8.70 , housemate rate: 81%, more than junior college graduated level: 53%, employment rate: 62%, annual income over 400 million yen /year: 62%, antidepressant use; 26%, benzodiazepine use:11%. Logistic Regression odds ratio (OR) with 95% confidence intervals (95%CI) for relationship of improvement insomnia and not with chronic pain were unadjusted OR: 3.600 (95%CI: 1.007-12.865) and adjusted OR: 3.078 (95%CI 0.779-12.161) (adjust for PCS.) **Conclusion:** We showed improvement in insomnia can affect pain release in the pain therapy, and PCS improvement may be also association with chronic pain release. **Support:**

0492

SLEEP AND HYPERAROUSAL: INABILITY TO DISCONTINUE CHRONIC HYPNOTIC USE

Roehrs, T.^{1,2} Koshorek, G.¹ Verkler, J.¹ Roth, T.^{1,2} ¹Henry Ford Health System, Detroit, MI, ²Wayne State University, SOM, Detroit, MI.

Introduction: Inability to discontinue chronic hypnotic use by people with insomnia remains a clinical concern. Sleep and hyperarousal was examined in an on-going "blinded" clinical trial in which people with insomnia are instructed to discontinue their study medication after 6 months of nightly use.

Methods: DSM-V diagnosed people with insomnia (n=31, 26 females), aged 23-61 yrs, with a polysomnographic sleep efficiency (SE) of \leq 85% on a 8-hr polysomnogram, no other sleep disorders, unstable medical or psychiatric diseases or drug dependency have completed the clinical trial. Participants were randomized to zolpidem XR (12.5 mg), eszopiclone (3 mg) or placebo nightly for 6 months (blinded groups A: n=11, B: n=9, C: n=11). After 6 months, over a 2-week choice period, they were given the instruction to discontinue their nightly hypnotic use with an opportunity, *if necessary*, to self-administer either 1, 2, or 3 capsules of their assigned medication (zolpidem XR 6.25 mg as capsule 2, placebo as capsule 3; eszopiclone 2 mg, 1 mg, and placebo as capsules 1, 2 and 3 respectively; or 3 placebos.

Results: Fifteen subjects stopped taking study medication when told to discontinue. The other 16 subjects who took study medication (users) had longer MSLT (a measure of hyperarousal) sleep

Conclusion: Hyperarousal, defined by MSLT and high diurnal urinary cortisol levels, has been found in some people with insomnia. High MSLTs were previously associated with dose escalation in a chronic zolpidem use study. These emerging data would suggest high MSLT may also be predictive of difficulty discontinuing hypnotic use.

Support: NIDA, grant#: R01DA038177 awarded to Dr. Roehrs

0493

USE OF SLEEP AID MEDICATION IS ASSOCIATED WITH MEMORY DEFICITS: A POPULATION-BASED STUDY

Dokkedal-Silva, V. Oliveira, M. M. Galduroz, J. F. Tufik, S. Andersen, M. L.

Universidade Federal de Sao Paulo, Sao Paulo, BRAZIL.

Introduction: Use of medications to treat sleep complaints is a common practice that may incur in cognitive deficits. Evidence, beyond the well-described effects of benzodiazepines on cognition, still needs expansion. This study assessed the use of sleep aid medications of different classes and associated factors on prospective and retrospective memory in a representative populational sample from São Paulo.

Methods: Volunteers in the EPISONO study underwent evaluation through questionnaires and a complete polysomnography exam. A subsample of 500 volunteers (Mean Age=42.9 years; 307 women) was included. Users (N=250) and non-users (N=250; randomly selected among the non-using volunteers) of sleep medication were evaluated regarding scores in 8 subscales and the overall score of the Prospective and Retrospective Memory Questionnaire (PRMQ). Afterwards, users of classes of medications were compared. Covariates, including scores in psychiatric evaluation scales and polysomnographic findings, were added in both segments to identify the factors with highest interference in the results obtained. Results: Medication users consistently performed worse in prospective memory and short-term internal-cued retrospective memory even when covariates were added. Scores in Beck Anxiety and Depression Inventories, Insomnia Severity Index and variables related to wakefulness and sleep architecture were the covariates with the highest interference in the results. When comparing types of medication, few differences were seen, suggesting that for such analysis, a sample with higher power would be appropriate.

Conclusion: Users of sleep medication presented impairment in prospective memory. Factors such as sleep architecture and continuity, as well as insomnia, anxiety and depression symptoms must be considered when evaluating cognitive deficits and pharmacological treatment in patients with sleep complaints, as they may participate in this relationship. Future studies are necessary to characterize the impact of different medication classes on prospective and retrospective memory.

Support: Our studies are supported by Associação Fundo de Incentivo à Pesquisa (AFIP) and Conselho Nacional de Desenvolvimento Científico Tecnológico (CNPq - Grant #133397/2017-3); S.T. and M.L.A. received CNPq fellowships.

0494

ABILITY TO DISCONTINUE CHRONIC HYPNOTIC USE: AN UPDATE

Roth, T.^{1,2} Koshorek, G.¹ Verkler, J.¹ Roehrs, T.^{1,2} ¹Henry Ford Health System, Detroit, MI, ²Wayne State University, SOM, Detroit, MI. **Introduction:** Physicians prescribing hypnotics remain concerned regarding patient's inability to discontinue hypnotics after chronic use. That concern has never been directly tested in a controlled prospective study using self-administration choice procedures. This is an update on results from an on-going "blinded" clinical trial in which insomnia subjects are instructed to stop taking their study medication after 6 months of nightly use.

Methods: DSM-V diagnosed insomnia subjects, aged 23-61 yrs, (n=31, 26 females), with disturbed sleep (i.e., polysomnographic sleep efficiency of \leq 85%), no other sleep disorder, unstable medical or psychiatric diseases or drug dependency completed the trial. Participants were randomized to zolpidem XR (12.5 mg), eszopiclone (3 mg), or placebo nightly for 6 months (blinded groups A: n=11, B: n=9, C: n=11). After 6 months, nightly use, over a 2-week choice period, they were instructed to discontinue hypnotic use, but <u>if necessary</u>, to self-administer either 1, 2, or 3 capsules of their assigned medication (zolpidem XR 6.25 mg, 6.25 mg, placebo; eszopiclone 2 mg, 1 mg, placebo as capsules 1, 2 and 3 respectively; or 3 placebos).

Results: The number of capsules taken declined from week 1 to 2 (p< .001). Over the 2 weeks 15 participants took 0 (48%), $12 \le 6$ (39%) and 4 ≥10 total capsules (1 each took 42, 19, 13, and 10). Among those taking capsules, most took one capsule per night and 6 took > 1 capsule. Those 4 taking ≥ 10 were younger (p<.05), but did not differ in screening sleep efficiency or blinded treatment group. Importantly 1 subject took every capsule available.

Conclusion: The majority (87%) of the participants discontinued 6-month nightly hypnotic use (i.e. took \leq 6 total capsules) and among those taking capsules the rate declined from week 1 to 2. Age may help identify the few with difficulty discontinuing. **Support:** NIDA, grant#: R01DA038177 awarded to Dr. Roehrs

0495

AURICULAR ACUPUNCTURE TREATMENT ASSOCIATED WITH IMPROVED SELF-REPORTED SLEEP AND EMOTIONAL DISTRESS IN HISPANIC AND LATINO IMMIGRANTS: A PRELIMINARY REPORT Hockmeyer, T. R. Rupp, H. R.

EbbTide Wellness, Easton, MD.

Introduction: Hispanic and Latino immigrants face sociocultural stressors that may contribute to sleep disturbance. We investigated: 1. Whether Hispanic/ Latino immigrants with self-reported sleep disturbance showed improvements in sleep with auricular acupuncture; 2. If sleep effects were associated with emotional distress. Methods: Emotional distress and sleep responses to auriculotherapy in Hispanic/ Latino adult immigrants were measured using Emotional Distress (ED), Athens Insomnia (AI), and Pittsburgh Sleep Quality Index (PSQI) scales completed Baseline, Mid (4 treatments), and Follow-up (8 treatments). Randomly assigned intervention was bi-weekly for 8 treatment sessions of 5 needles inserted bilaterally: Active Intervention [NADA protocol (Shen men, sympathetic autonomic, lung, liver and kidney points)] OR Sham Control (outer ear helix with no active points). Scores were compared between Groups (NADA or SHAM) and within (Pre-, Mid- and Post-treatment) sessions using Mixed-Model ANOVA; multiple linear regression assessed ED scores association with sleep.

Results: Ten Hispanic/ Latino female participants [NADA, N=5, mean (SD) age = 41 (14); SHAM, N =5; mean (SD) age = 42 (17)]. Anovas for the ASI, PSQI, and ED showed significant within-subjects effects (p's < .05). Baseline; Mid; Follow-up Mean (SD) = **AIS**: 12.3 (8.9); 8.5 (6.9); 7.1 (12.8); **PSQI**: 10.9 (5.4); 8.9

(2.8); 7.6 (3.4); **ED**: 25.6 (50.4); 17.6 (26.7); 16.9 (29.8). Post-hoc t-tests were significant between Baseline/ Mid and Baseline/ Final for all measures (p's < .05). Linear Regression showed significant association between ED and PSQI (R square = 0.25; p < .05): lower Emotional Distress associated with lower PSQI.

Conclusion: Auriculotherapy may improve sleep in Hispanic/ Latino immigrants after 4 treatments suggesting a novel, low-cost, easily implemented group treatment option for improving sleep in this community. Better sleep was associated with decreases in emotional distress supporting our hypothesis. Data collection continues and larger sample size will allow for increased power to detect between group differences.

Support: This research was made possible with thanks to the American Academy of Sleep Medicine (AASM) Humanitarian Award#:197-FP-18.

0496

SELF-REPORTED SLEEP DURING DISCONTINUATION OF CHRONIC HYPNOTIC USE

Verkler, J.¹ Koshorek, G.¹ Roth, T.^{1,2} Roehrs, T.^{1,2} ¹Henry Ford Health System, Detroit, MI, ²Wayne State University, SOM, Detroit, MI.

Introduction: Inability to discontinue chronic hypnotic use by people with insomnia remains a clinical concern. Self-reported sleep in an on-going "blinded" clinical trial in which people with insomnia are instructed to discontinue their study medication after 6 months of nightly use was examined and compared to objective actigraphic recordings of their sleep.

Methods: DSM-V diagnosed people with insomnia (n=31, 26 females), aged 26-61 yrs, with a polysomnographic sleep efficiency of \leq 85%, no other sleep disorders, unstable medical or psychiatric diseases or drug dependency completed the clinical trial. Participants were randomized to zolpidem XR (12.5 mg), eszopiclone (3 mg) or placebo nightly for 6 months (blinded groups A: n=11, B: n=9, C: n=11). After 6 months, over a 2-week choice period, they were given the instruction to discontinue their nightly hypnotic use with an opportunity, <u>if necessary</u>, to self-administer either 1, 2, or 3 capsules of their assigned medication (zolpidem XR 6.25 mg, 6.25 mg, placebo; eszopiclone 2 mg, 1 mg, placebo as capsules 1, 2 and 3 respectively; or 3 placebos). On post-sleep questionnaires they reported sleep latency (LAT), wake after sleep onset (WASO), and sleep quality (Q:1-5; best - worst).

Results: 15 subjects stopped taking study medication when told to discontinue. The 16 subjects who took study medication had shorter LAT on nights they took capsules then on nights they did not (31 vs 38 min, p<.03), less WASO (22 vs 44 min, p<.02) and better Q (2.3 vs 3.2, p<.002). In contrast, actigraphic recordings of sleep showed no differences in LAT, WASO, or SE.

Conclusion: For subjects who took study medication during the nights they were instructed to discontinue, they reported better sleep than on the nights they used no medication, although object-ively their sleep did not differ.

Support: NIDA, grant#: R01DA038177 awarded to Dr. Roehrs

0497

REBOUND INSOMNIA DURING DISCONTINUATION OF CHRONIC HYPNOTIC USE

Koshorek, G.¹ Verkler, J.¹ Roth, T.^{1,2} Roehrs, T.^{1,2} ¹Henry Ford Health System, Detroit, MI, ²Wayne State University, SOM, Detroit, MI. **Introduction:** Rebound insomnia refers to worsened sleep relative to baseline on 1-2 nights after discontinuation of active hypnotic medication. Rebound is typically assessed using a placebo substitution. We assessed rebound in an on-going "blinded" clinical trial in which people with insomnia are instructed to discontinue their study medication (i.e., no-pill) after 6 months of nightly use.

Methods: DSM-V diagnosed people with insomnia (n=31, 26 females), aged 26-61 yrs, with a polysomnographic sleep efficiency of \leq 85%, no other sleep disorders, unstable medical or psychiatric diseases or drug dependency completed the clinical trial. Participants were randomized to zolpidem XR (12.5 mg), eszopiclone (3 mg) or placebo nightly for 6 months (blinded groups A: n=11, B: n=9, C: n=11). After 6 months, over a 2-week choice period, they were given the instruction to discontinue their nightly hypnotic use with an opportunity, <u>if necessary</u>, to self-administer either 1, 2, or 3 capsules of their assigned medication (zolpidem XR 6.25 mg, 6.25 mg, placebo; eszopiclone 2 mg, 1 mg, placebo as capsules 1, 2 and 3 respectively; or 3 placebos). On baseline and the14 discontinuation nights, sleep was recorded and scored by actigraphy for sleep efficiency (SE), sleep latency (LAT) and wake after sleep onset (WASO).

Results: Relative to the baseline night, on the first discontinuation night there was no difference in SE, LAT, and WASO. Fifteen subjects stopped taking study medication when told to discontinue and 16 subjects took study medication on one night or more. While not differing on baseline or night 1, on night 14 the last study night the medication users had a lower SE (75.9 vs 87.7 %, p<.0.004) and a longer LAT (61.5 vs 14.5 min, p<0.05).

Conclusion: Difficulty discontinuing hypnotic use is not specifically related to rebound insomnia. We reported in a companion abstract those with insomnia and hyperarousal, defined by MSLT, are those with difficulty discontinuing hypnotic use and as shown here slept poorly on the last study night.

Support: NIDA, grant#: R01DA038177 awarded to Dr. Roehrs

0498

EFFECT OF LAVENDER ESSENTIAL OIL ON SLEEP IN POSTMENOPAUSAL WOMEN WITH INSOMNIA: DOUBLE-BLIND RANDOMIZED CONTROLLED TRIAL

Lucena, L. R.¹ Santos-Junior, J. G.² Tufik, S.¹ Hachul, H.¹ ¹Universidade Federal de São Paulo - Department of Psychobiology, Sao Paulo, BRAZIL, ²Faculdade de Ciências Médicas da Santa Casa de São Paulo - Department of Physiological Sciences, São Paulo, BRAZIL.

Introduction: Sleep is essential for women's health and its deprivation leads to serious physiological consequences. In addition, insomnia is a common complaint in postmenopausal women. Thus, the present study aimed to evaluate the effect of Lavandula angustifolia essential oil inhalation on sleep and menopausal symptoms in postmenopausal women with insomnia.

Methods: Experimental, double-blind, randomized controlled trial composed by 33 women (48 - 65 years) with clinical diagnosis of insomnia divided in two groups that inhaled different oils before sleep during 29 days: Placebo Group - PG (sunflower oil) and Aroma Group - AG (Lavandula angustifolia essential oil). Both groups received sleep hygiene guidelines and were followed weekly. To assess the effect of the intervention on menopausal symptoms the Menopause Rating Scale (MRS) and the Hospital Anxiety and Depression Scale (HADS) were used and for sleep evaluation the Insomnia Severity Index (ISI) and the Pittsburgh Sleep Quality Index (PSQI) were used. Additionally, polysomnography were used

to assess sleep pattern. All outcomes were evaluated before and after intervention. Statistical analysis was performed using SPSS 22 through Generalized Estimed Equations test with significance set at $p \le 0.05$.

Results: Comparing the pre and post-intervention periods, AG participants had a significant decrease in sleep onset latency, depression level, hot flashes, menopausal symptoms and a significant increase in sleep quality. Polysomnography data showed increased sleep efficiency and decreased wakefulness after sleep onset on AG participants. After the intervention, all volunteers presented improvement in all outcomes of sleep and menopausal symptoms measured by questionnaires.

Conclusion: The intervention was effective in improving the sleep pattern of AG participants, but was not significant when compared to PG participants. In addition, sleep hygiene instructions were essential to improve the sleep pattern of all volunteers. Therefore, inhalation of lavender essential oil is a safe, low-cost practice that should be considered as a complementary option to conventional treatments, whether medical, psychological or other integrative and complementary practices.

Support: This research was supported by fellowships from Associação Fundo de Incentivo à Pesquisa (AFIP), Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES) -Finance Code 001 and Conselho Nacional de Pesquisa (CNPq).

0499

PREDICTORS OF SESSION ATTENDANCE IN A RCT FOR **CBT-I FOR PERINATAL INSOMNIA**

Rangel, E. Asarnow, L. Simpson, N. Manber, R. Stanford University, Stanford, CA.

Introduction: Cognitive behavioral therapy for insomnia (CBT-I) is recommended as the first-line treatment for chronic insomnia disorder. However, early treatment dropout can negatively impact the success of treatment. In order to design effective strategies to reduce attrition we need to understand predictors of dropout. In this work, we focus on predictors of treatment session attendance among pregnant women in a randomized controlled trial of CBT-I. Methods: Participants were 87 pregnant women with insomnia disorder (mean age=32.5, SD=5.1 years) who were enrolled in a randomized controlled trial of CBT-I and were randomized to the CBT-I arm (5 sessions). We did not include women who did not complete treatment due to early labor. The Insomnia Severity Index (ISI) and Edinburgh Postnatal Depression Scale (EPDS) and demographic questionnaires (age, income, and educational background) were administered at screening.

Results: Logistic regression analyses were conducted to identify predictors of number of CBT-I sessions attended during pregnancy. A logistic regression model that included clinical predictors found that the ISI and EPDS were not significant predictors of session attendance. A logistic regression model that included demographic predictors (income, education, and age) was significant (F(3,76)=6.49, p<0.001) with an R² of .204. Independently, income was not a significant predictor ($\beta = .15$, p=.32), but education (β =-.21, p<.05) and age (β =-.48, p<.01) were significant predictors of fewer sessions attended. Dropping income from the model, there was a significant age by education effect ($\beta = 1.39$, p<.05). Among participants with less than a college degree, those who were younger had attended fewer sessions during pregnancy. Among those who completed a college education and above, number of sessions attended did not differ by age.

Conclusion: Pregnant women with insomnia who were randomized to receive CBT-I were more likely to withdraw early from treatment if they were younger and had less than a college education. Further research that focuses on increasing attendance in CBT-I treatment among pregnant women needs to develop strategies to increase retention in this vulnerable population. Support: NR013662

0500

EVALUATION OF THE HUMAN ABUSE POTENTIAL OF SINGLE ORAL DOSES OF V117957, A NOVEL, HIGHLY POTENT AND SELECTIVE PARTIAL AGONIST FOR NOCICEPTIN/ORPHANIN-FQ PEPTIDE (NOP) RECEPTORS

Harris, S.¹ Zhou, M.¹ Cipriano, A.¹ Kapil, R.¹ Shet, M.¹ He, E.¹ Apseloff, G^2

¹Imbrium Therapeutics, Stamford, CT, ²Ohio Clinical Trials, Inc., Columbus, OH.

Introduction: The satisfactory safety/tolerability profile of V117957, an investigational NOP receptor partial agonist, has been previously established in ~200 healthy subjects with maximum doses at 30mg following a single oral administration and 10mg once daily for 2 weeks. V117957 exhibited linear plasma exposures at doses up to 10mg. In patients with insomnia disorder, V117957 demonstrated dose-dependent improvement in sleep efficiency and sleep maintenance between 0.5mg and 10mg as measured by polysomnography and patient diaries. The current study evaluated the abuse potential and safety of V117957 in healthy, nondependent recreational polydrug users with a history of central nervous system (CNS) depressant use.

Methods: The abuse potential of V117957 (1mg, 6mg, 10mg), placebo, and triazolam (0.5mg, 1mg) were assessed in a randomized, double-blind, double-dummy, placebo- and positive-controlled crossover study. Triazolam was utilized as a positive control based on its comparable pharmacokinetic and pharmacodynamic characteristics. V117957 doses (1mg, 6mg, 10mg) were selected to represent therapeutic, mid-range supratherapeutic, and maximumtolerated supratherapeutic doses, respectively. Subjects were qualified based on pharmacodynamic responses following a single oral 0.75mg triazolam dose. Drug liking was measured through 24 hours, including the primary endpoint of maximum "at the moment" Drug-Liking Visual Analog Scale, as recommended by FDA. Secondary endpoints included Divided Attention Test (DAT) and Choice Reaction Time (CRT).

Results: The positive control (triazolam 0.5mg, 1mg) produced statistically significant greater abuse potential and cognitive/motor impairment versus placebo, which demonstrated study validity. In contrast, V117957 at 1mg was not statistically significantly different from placebo. At the supra-therapeutic doses of 6 and 10mg, V117957 was associated with abuse potential and cognitive/motor impairment greater than placebo, yet similar to those of triazolam 0.5 and 1mg.

Conclusion: Overall, in this valid study, V117957 1mg and placebo were associated with statistically significant lower potential for abuse and reduced cognitive/motor impairment compared with the two supratherapeutic doses of V117957 (6mg, 10mg), and triazolam (0.5mg, 1mg). V117957 1mg met FDA's statistical criterion for similarity to placebo.

Support: Funded by Imbrium Therapeutics, a subsidiary of Purdue Pharma L.P.

0501

DEVELOPMENT AND INITIAL EVALUATION OF WEB-BASED COGNITIVE BEHAVIORAL THERAPY FOR INSOMNIA "NITECAPP" IN RURAL DEMENTIA CAREGIVERS: A MIXED-METHODS STUDY

McCrae, C.¹ Curtis, A. F.¹ Nair, N.¹ Deroche, C. B.¹ Shenker, J.¹ Rowe, M.²

¹University of Missouri, Columbia, MO, ²University of South Florida, Tampa, FL.

Introduction: Informal caregivers (CGs) of persons with dementia frequently experience insomnia. The time consuming and unpredictable schedule of CGs, and associated emotional/physical exhaustion emphasize the need for brief, easily accessible interventions to treat insomnia. Internet-based behavioral insomnia interventions hold promise, particularly for rural CGs who have limited access to traditional in-person treatments. This study aimed to 1) translate an efficacious 4 session cognitive behavioral therapy for insomnia (CBT-I) to web-based "NiteCAPP" for dementia caregivers, and 2) conduct NiteCAPP usability testing/evaluate acceptability of content and features.

Methods: NiteCAPP is an online CBT-I that incorporates guided delivery through weekly therapist moderator feedback. A stepwise approach was implemented in order to explore user needs and validate NiteCAPP content in a focus group of rural dementia caregivers (n=5) and primary care providers (PCPs; n=5). Participants conducted usability testing and provided ratings of program content (1-least favorable to 5-most favorable) regarding ease of use, amount of information, website maintaining interest, adequate font size, videos maintaining interest/easy to understand/helpful. Participants also indicated whether they had at home internet access, method of internet access, and provided open ended feedback on NiteCAPP. Feedback transcripts were compiled and analyzed independently (C.S.M., A.F.C.) through deductive content analysis. Topics mentioned frequently were categorized and merged into common themes during consensus meeting, and NiteCAPP was subsequently adapted.

Results: Average ratings for NiteCAPP features were high, ranging from 4.1/5 to 4.7/5 across all items. All participants had access to internet through both phone and computer. No barriers to use identified. Feedback themes were largely positive (e.g., comprehensive written material, promotes independence, excellent visual tools for therapy moderator feedback, good pacing, use of visual contrast). Negative themes for improvement/adaptation included adding font size options, a light/dark mode, tab with all videos, reducing amount of scrolling, adding a glossary of terms.

Conclusion: Rural dementia CGs and PCPs evaluated NiteCAPP as easy to use with acceptable features and program content and no barriers to access. Improvement themes were used to adapt NiteCAPP. Next steps are to evaluate feasibility and preliminary efficacy of NiteCAPP in rural dementia CGs with insomnia. **Support:** none

0502

SAFETY, TOLERABILITY, AND EFFICACY OF A NOVEL, HIGHLY POTENT AND SELECTIVE PARTIAL AGONIST FOR NOCICEPTIN/ORPHANIN-FQ PEPTIDE (NOP) RECEPTORS IN PATIENTS WITH INSOMNIA DISORDER

Zhou, M.¹ Harris, S.¹ Kapil, R.¹ Cipriano, A.¹ He, E.¹ Shet, M.¹ Fukumura, K.² Matsuo, Y.² Uehira, M.² Zammit, G.³

¹Imbrium Therapeutics, Stamford, CT, ²Shionogi & Co., Ltd., Osaka, JAPAN, ³Clinilabs Drug Development Corporation, New York, NY.

Introduction: V117957 is a recently described investigational oral, potent, and selective nociceptin/orphanin-FQ peptide (NOP) receptor partial agonist which was previously evaluated in ~200 healthy subjects. Its satisfactory safety/tolerability profile has been established with the top doses at 30mg following a single oral administration and 10mg once daily for 2 weeks. V117957 demonstrated favorable drug-like properties for insomnia treatment, including oral bioavailability, fast absorption, and rapid elimination.

Methods: A total of 52 patients with insomnia disorder have been evaluated in two separate randomized, double-blind, crossover, placebo-controlled sleep studies. Insomnia disorder was confirmed by screening polysomnography (PSG). All subjects received orally, for two consecutive nights, either V117957 10mg or placebo in Study #1 or 0.5, 1, 3, 6mg or placebo in Study #2. Efficacy was measured via PSG for the primary endpoint of sleep efficiency (SE) and secondary endpoints of sleep onset (latency to persistent sleep [LPS]) and maintenance (wakefulness after sleep onset [WASO]). Efficacy also was measured by patient diary (subjective sleep latency [sSL], subjective total sleep time [sTST], sWASO). Pharmacodynamics (PD) on next-day residual effects were also measured, including cognitive, psychomotor and mood effects.

Results: V117957 showed statistically significant greater sleep efficiency and less WASO in a dose-dependent manner (0.5-10 mg) and a statistically significant reduction in LPS at 10mg, as compared to placebo. V117957 at 0.5mg and 1mg exhibited next-day residual effects similar to placebo. At doses of 3mg or higher, V117957 showed dose-dependent next-day residual effects. V117957 was safe and well-tolerated across all doses tested with no serious adverse events, with somnolence being the most frequent treatment-emergent adverse event. No concerning laboratory findings and no clinically significant findings on vital signs and electrocardiograms have been attributed to V117957 in these subjects.

Conclusion: V117957 was safe and well-tolerated in patients with insomnia disorder. These results demonstrated that NOP receptors represent a novel mechanistic treatment for insomnia disorder and support continued evaluation of V117957.

Support: Funded by Shionogi and Imbrium Therapeutics, a subsidiary of Purdue Pharma L.P.

0503

REDUCTIONS IN SLEEP AND DAILY RHYTHM VARIABILITY FOLLOWING BRIEF BEHAVIORAL TREATMENT FOR INSOMNIA

Kanady, J. C. Straus, L. D. Gloria, R. Neylan, T. C. Maguen, S. San Francisco VA Medical Center, San Francisco, CA.

Introduction: Brief behavioral treatment for insomnia (BBTI) is efficacious for insomnia symptoms. Here we examine whether BBTI reduces sleep and daily rhythm variability and whether reductions in variability result in improved functioning and quality of life.

Methods: Ninety-one Veterans with insomnia (49.3 ± 18.7 yrs; 18.7% female) were randomized to one of two treatment conditions: BBTI or progressive muscle relaxation training (PMRT; control condition). Variability was assessed using sleep diaries and actigraphy. The sleep diary outcome variables included bedtime variability, wake time variability, and total sleep time variability; actigraphy variables included interdaily stability, intradaily variability, and total sleep time variability. Functioning was assessed

using the Work and Social Adjustment Scale. Quality of life was assessed using the Hotel Dieu-16 Scale.

Results: Compared to PMRT, BBTI resulted in a greater reduction in subjective total sleep time variability ($F_{1,90}$ =6.61, p<0.01, partial η^2 =0.13) and a greater increase in interdaily stability ($F_{1,78}$ =12.41, p<0.01, partial η^2 =0.25). There was a greater decrease in intradaily variability following PMRT ($F_{1,78}$ =27.96, p<0.01, partial η^2 =0.42). Across the entire sample, reductions in subjective wake time variability were associated with improved functioning ($F_{1,88}$ =4.43, p=0.04, η^2 =0.05) and reductions in subjective total sleep time variability were associated with improved quality of life ($F_{1,89}$ =4.91, p=0.03, partial η^2 =0.05).

Conclusion: There was significant improvement in the stability of sleep-wake rhythms following BBTI, suggesting that BBTI not only treats insomnia, but also may stabilize circadian rhythms. Interestingly, PMRT resulted in greater intradaily variability reductions than BBTI. One explanation is that due to BBTI stimulus control guidelines, individuals were getting out of bed in the middle of the night more frequently and thus, these awakenings were better captured by actigraphy. Reductions in wake time and total sleep time variability were associated with improved functioning and quality of life, further demonstrating the importance of stable sleep-wake rhythms.

Support: VA Rehabilitation Research and Development Grant # RX001539-01A2

0504

EFFICACY AND SAFETY OF SELTOREXANT IN INSOMNIA DISORDER

Savitz, A.¹ Saoud, J. B.²

¹Janssen Research & Development, LLC, Titusville, NJ, ²Minerva Neurosciences, Inc., Waltham, MA.

Introduction: Insomnia disorder affects 3-6% of the population and includes functional impairing symptoms for \geq 3 months. Current hypnotics have limitations (eg fall risk, dependency, parasomnia, cognitive impairment). In a previous proof-of-concept study, seltorexant, a selective orexin-2 receptor antagonist, dosed at 40mg nightly improved sleep efficiency, latency to persistent sleep (LPS), and wake after sleep onset (WASO) versus placebo.

Methods: A multicenter, double-blind, randomized, parallelgroup, active- and placebo-controlled, 14-day, dose-finding study (NCT03375203) assessed the efficacy and safety of seltorexant in adult and elderly subjects meeting DSM-5 insomnia disorder criteria. Subjects were randomized (1:1:1:1:1 ratio) to: placebo, seltorexant 5mg, 10mg, 20mg, or zolpidem. Primary endpoint: Based on PSG, dose response in LPS change on Night 1 relative to baseline. Secondary endpoints included change from baseline in: WASO over the first 6 hours (WASO-6) on Night 1, and LPS and WASO-6 on Night 13. Multiple Comparison Procedure-Modeling, ANCOVA and MMRM were used for data analyses.

Results: 365 patients were randomized and 347 (95%) completed the double-blind phase with 68% women and median age of 59yr (22 to 84yr). Statistically significant dose-response relationship (p-value <0.001) was observed for LPS at Night 1 (5mg=12%, 10mg=36%, 20mg=49% improvement compared with placebo). Similar findings were observed for WASO-6 and benefits sustained from Night 1 to 13. The 20 mg dose showed greater improvement than zolpidem on LPS at Nights 1 and 13 and on WASO-6 at Night 13. Overall treatment-emergent adverse events rates were comparable in the seltorexant, placebo, and zolpidem treatment groups.

Conclusion: Statistically significant and clinically meaningful improvements on LPS & WASO-6 were observed for seltorexant 10 and 20mg dose groups versus placebo. No safety concerns were observed. Seltorexant with its novel mechanism holds potential as

Support:

0505

INSOMNIA: ENTRAPMENT OF BINAURAL AUDITORY BEATS ON SUBJECTS WITH INSOMNIA SYMPTOMS

Choi, H.¹ Bang, Y.² Yoon, I.³

a new treatment for insomnia disorder.

¹Veteran Health Service Medical Center, Seoul, KOREA, REPUBLIC OF, ²Dong-A University Hospital, Busan, KOREA, REPUBLIC OF, ³Seoul National University Bundang Hospital, Seongnam, Gyeonggi-do, KOREA, REPUBLIC OF.

Introduction: It has been reported that binaural beat stimulation, which has two different frequencies on both ears, is effective in increasing alertness, memory, reducing anxiety, and controlling mood. This study aims to clarify the brain wave entrainment effect of binaural beat and to identify the mechanism of action of the binaural beat.

Methods: Subjects with subclinical insomnia symptoms between 20 and 59 years of age were recruited from the community. Quantitative electroencephalography (EEG) was measured two times before and after the 2 weeks of binaural beat intervention period. An audio apparatus without the distortion of a sound source is set with theta (6 Hz) binaural beat. Participants used the apparatus for 30 minutes before going to bed for 2 weeks.

Results: When the music with binaural beat was played, the relative power of theta frequency increased (occipital, P=0.009). When the music only was played in the laboratory, the relative power of delta (temporal, P=0.004; parietal, P=0.005; occipital, P=0.006) and theta frequency (temporal, P=0.004; central, P=0.001; parietal, P=0.001; occipital, P=0.003) increased and the relative power of alpha decreased (frontal, P=0.008; temporal, P=0.012; central, P=0.008; parietal, P=0,004; occipital, P=0.005). After listening to music with binaural beat for two weeks, the difference of beta power before and after listening to music first in the laboratory was lower than the difference after using music-only devices (P=0.008). Conclusion: When the binaural beat was played, the entrapment of theta wave appeared. And the music was presumed to have a nonspecific relaxation effect. After exposure to music with binaural beat for 2 weeks, beta power decreased compared to exposure to music alone. Continuous music with binaural beat exposure for 2 weeks is likely to reduce hyper-arousal state and contribute to sleep induction.

Support: None

0506

SHORT AND LONG-TERM EFFECTS OF TRAZODONE VS. COGNITIVE-BEHAVIORAL TREATMENT ON EEG POWER DURING NREM SLEEP IN CHRONIC INSOMNIA

Li, Y.¹ Vgontzas, A.² Fernandez-Mendoza, J.³ Fang, J.² Puzino, K.² Basta, M.² Bixle, E.²

 ¹Sleep Medicine Center, Mental Health Center, Shantou University Medical College, China, Shantou, CHINA, ²Sleep Research & Treatment Center, Department of Psychiatry, Pennsylvania State University College of Medicine, Hershey, PA,
 ³Sleep Research & Treatment Center, Department of Psychiatry, Pennsylvania State University College of Medicine, Hershey, PA.

Introduction: Both trazodone and cognitive-behavioral treatment of insomnia (CBT-I) are widely used to treat patients with chronic insomnia. Animal studies have shown that trazodone increases slow wave sleep (i.e., increased EEG delta power). However, no study to date has compared the long term effects of trazodone vs. CBT-I on spectral EEG activity during sleep in humans.

Methods: We addressed this question in a sample of 19 middleaged men and women who received either trazodone (n=8) or CBT-I (n=11) treatment for 9 months. We examined delta (0.39-3.91 Hz), theta (4.30-7.81 Hz), alpha (8.20-11.72 Hz), sigma (12.11-14.84 Hz), beta (15.23-35.16 Hz) and gamma (35.55-49.61 Hz) relative power during NREM sleep after 3-month and 9-month of treatment.

Results: Compared to CBT-I, trazodone significantly increased relative delta power (p=0.05) and decreased relative sigma (p=0.004) and beta (p=0.05) power during NREM sleep across 9-month treatment. Furthermore, compared to CBT-I, trazodone significantly increased relative delta power (3-month: $\Delta 2.00 \pm 3.27$ vs. Δ -2.63 ± 5.88, p=0.006, Cohen's d=0.93; 9-month: Δ 2.63 ± 4.11 vs. Δ -1.10 ± 3.93, p=0.006, Cohen's d=0.93), while decreased relative sigma power (3-month: Δ -1.55 ± 1.75 vs. Δ 0.90 ± 1.82, p=0.009, Cohen's d=1.37; 9-month: Δ -1.33 ± 1.95 vs. Δ 1.05 ± 1.79, p=0.014, Cohen's d=1.28) during NREM sleep in 3-month and 9-month, respectively. Relative beta power (3-month: Δ -0.85 ± 0.60 vs. $\Delta 0.35 \pm 1.14$, p=0.016, Cohen's d=1.03;) was significantly decreased in 3-month treatment in trazodone group compared to CBT-I. Moreover, across 9-month treatment, relative sigma $(p=0.040, \omega_p^2 = 0.29)$ and beta $(p=0.021, \omega_p^2 = 0.12)$ power during NREM sleep were significantly decreased within trazodone group, while relative sigma power (p=0.096, $\omega_p^2 = 0.230$) increased within CBT-I group.

Conclusion: Our findings suggest that trazodone, but not CBT-I, even after 9-month of use increases slow wave sleep and decreases high-frequency EEG power during NREM sleep. This effect may explain the long-term usefulness of trazodone in chronic insomnia patients with physiologic hyperarousal i.e., activation of the stress system. Further studies should examine this effect in large samples of insomnia.

Support: NIH C06 RR016499, UL1 TR 000127

0507

MOBILE COGNITIVE BEHAVIORAL THERAPY IS EFFICIENT IN IMPROVING SLEEP IN STUDENTS

Eyal, S.¹ Altman, Y.¹ Baharav, A.^{1,2}

¹HypnoCore, Petach Tikva, ISRAEL, ²Wingate Institute, Netanya, ISRAEL.

Introduction: Academic achievements and social life on campus represent the main focus for students, while sleep is neglected. The emergence of social media, gaming reduces sleep opportunity, quality and ability. Students are sleep challenged and prone to develop chronic sleep difficulties in later life. Cognitive behavioral interventions are recognized as effective for insomnia and circadian misalignment. We aimed at detecting sleep difficulties, related habits, and at testing the efficacy of a mobile app in improving sleep in students with insomnia symptoms.

Methods: Observational study of US students who approached wellness staff and were offered the Refresh by Sleeprate mobile app that provides a sleep assessment followed by weekly cycles of personalized digital cognitive and behavioral reframing. The app collects perceived, and optional objective sleep data acquired using wearable devices. 892 students aged 18-30 years registered

an account between Jan 1 and Oct 30, 2019. The study reports engagement data and outcomes of the assessment and the digital intervention.

Results: 507 completed their assessment (6.2 avg nights). 69% presented insomnia symptoms with or without circadian misalignment, 8% circadian misalignment, 12% sleep deprivation, 11% poor sleep hygiene. 192 (55.3% of students with insomnia symptoms) completed at least one week of intervention (5.6 weekly avg nights, 28 avg total nights). Sleep Latency (SL) in minutes decreased from 28.8 (21.5) (Mean/SD) to 22.1 (19.3), p<0.001. When the initial mean SL was longer than 30 minutes, the improvement was larger, from 53.9 (20.8) to 32.7 (25.4) (p<0.001). Mean perceived Wake After Sleep Onset (WASO) longer than 30 minutes decreased from 46.3 (19.0) to 35.8 (21.4), p<0.05. Sleep Efficiency (SE) increased by 1.6% (p<0.002) for all, and by 7.1% (p<0.001) for SE<85%.

Conclusion: The mobile app used reveals sleep problems and is efficient in improving insomnia symptoms in those who remain engaged. 55% of those who started the program also completed it. Engagement remains the main barrier to sleep improvement at scale.

Support: N/A

0508

TRANSCRANIAL MAGNETIC STIMULATION SHOWS FAVORABLE RESPONSE FOR INSOMNIA IN DEPRESSION WITH GREATER RESPONSE IN MALES AND IN THOSE LESS THAN 65 YEARS OF AGE

Stultz, D. J. Osburn, S. Burns, T. Stanley, N. Walton, R. Cope, A. Pawlowska-Wajswol, S.

Stultz Sleep & Behavioral Health, Barboursville, WV.

Introduction: Transcranial Magnetic Stimulation (TMS) is FDA approved for the treatment of resistant depression and multiple studies have demonstrated improvement of insomnia in both those with and without depression.

Methods: 50 patients were studied while undergoing TMS treatment for resistant depression and utilizing the Patient Health Questionnaire-9 (PHQ-9), the Beck Depression Inventory (BDI), the Insomnia Severity Index (ISI), and the Pittsburgh Sleep Quality Index (PSQI) for evaluation of benefit. Using the Brainsway dTMS system over the LDPFC at 120% MT for an average of 31 treatments, our study demonstrated benefit for both mood and insomnia. We observed an improvement on the PHQ-9 from 17.3 to 7.53, on the BDI from 30.44 to 11.75, on the ISI from 13.47 to 9.31, and on the PSQI from 11.78 to 9.08. Focusing specifically on the insomnia response, we compared an equal number of both male versus female patients, and those > and < than 65 years of age.

Results: Using paired t-test comparisons, men and those less than 65 demonstrated statistically significant improvement. The male population demonstrated statistically significant decreases of t=2.39, 13df, P=.03 on the ISI, and t=2.59, 13df, P=.02 on the PSQI. For women the result was t=1.35, 13df, P=.20 on the ISI, and t=2.05, 13df, P=.06 on the PSQI. In the elderly (>65) decreases were not statistically significant at t=.62, 14df, P=.54 on the ISI, and t=1.26, 14df, P=.23 on the PSQI. For those < 65 years old statistically significant decreases observed were t=3.37, 14df, P=.005 on the ISI, and t=3.5, 14df, P=.004 on the PSQI.

Conclusion: TMS treatment of depression resulted in statistically significant benefits on co-existing insomnia in males and those less than 65 years of age. As insomnia may be a precipitating or perpetuating factor in depression and may result in depression relapse, attention to this symptom is of clinical benefit.

Support: **No support was given for this study. Dr. Stultz is a speaker for Harmony Biosciences and has served on their advisory committee. She is also a speaker for Jazz Pharmaceuticals. She is the co-editor for the Clinical TMS Society Newsletter and on the education committee.

0509

USE OF BLINDED HYPNOTIC TAPERING FOR HYPNOTIC DISCONTINUATION

Edinger, J. D.^{1,2} Walmboldt, F.¹ Holm, K.¹ Johnson, R. L.¹ Simmons, B.¹ Tsai, S.¹ Morin, C.³

¹National Jewish Health, Denver, CO, ²Duke University Medical Center, Durham, NC, ³Laval University, Quebec City, QC, CANADA.

Introduction: Many patients have difficulties achieving hypnotic discontinuation due to anxiety that arises when they knowingly reduce their hypnotic dose or withhold it entirely. This study tested a blinded tapering approach to reduce patients' anxiety and help them discontinue their hypnotics.

Methods: The study sample included 78 (M age = 55.2 ± 12.8 yrs.; 65.4% women) users of benzodiazepine and benzodiazepine receptor agonists. Following baseline assessments, enrollees first completed 4 sessions of cognitive behavioral insomnia therapy (CBTI). Subsequently they were randomized to one of three 20-week, double-blinded tapering protocols wherein their medication dosage either remained unchanged (CTRL) or was reduced by 25% or 10% every two weeks. At the end of the 20-week period the study blind was eliminated and those who completed one of the two blinded tapering protocols entered a 3-month follow-up period, whereas CTRL participants were offered an open label taper before completing the follow-up.

Results: Among those who completed one of the blinded tapering protocols, 92.9% totally discontinued their medication use by the end of the 20-week tapering phase, whereas 77.3% in the CTRL group discontinued hypnotic use by the end of their open label tapering. At follow-up 72.1% of those who completed blinded tapering remained medication free whereas only 52% of those who underwent open-label tapering remained medication free. Comparisons at follow-up showed those who received the open-label taper continued to use hypnotics on average 2-3 nights/week compared to about 1 time every other week for the blinded taper group (p = .05). The average weekly diazepam equivalent dose of medication used by the open label tapering group was about 5 times higher than the average weekly dose used by the blind tapering group (p = .025).

Conclusion: CBTI combined with blinded hypnotic tapering is a promising treatment approach for helping hypnotic users overcome their medication dependence.

Support: National Institute of Drug Abuse, Grant # R34 DA042329-01

0510

THE APNEA AND INSOMNIA RESEARCH (AIR) TRIAL: A PRELIMINARY REPORT

Edinger, J. D.¹ Manber, R.²

¹National Jewish Health, Denver, CO, ²Stanford University, Palo Alto, CA.

Introduction: Many sleep apnea patients suffer from comorbid insomnia disorder. Although cognitive behavioral insomnia therapy (CBTI) is recommended as the first line insomnia treatment for such patients, access to trained providers of this treatment remains limited. The current study is testing he efficacy of an online CBTI among CPAP treated sleep apnea patient with comorbid insomnia. **Methods:** Patients enrolled in this trial complete baseline measures and then are randomized to either an online version of Cognitive Behavioral Insomnia Therapy (CBTI) or no additional treatment beyond their CPAP therapy (CTRL). After 8 weeks of treatment all patients are reassessed. The current report considers changes in scores on the ISI and Epworth Sleepiness Scale (ESS) as well as average minutes of nightly CPAP use from pre-treatment to the end of the initial 8 weeks of online treatment relative to the no treatment CTRL. The sample for this report included the first 170 participants enrolled in this trial (mean age = 56.5 ± 12.5 yrs; 60.1%females).

Results: Those receiving online CBTI showed greater reductions in their ISI scores from baseline to the end of the initial 8-week treatment phase than did those in the CTRL group (p = .0009). Average ISI score improvements among those receiving online CBTI moved patients from moderately severe insomnia to mild insomnia symptoms. In contrast, no differences were noted between the online CBTI and CTRL groups in regard to pre- to post-treatments changes on the ESS (p= .3771) scores or amount of CPAP use (p = .8053).

Conclusion: Whereas online CBTI does not seem to reduce daytime sleepiness or improve CPAP adherence among patients with comorbid sleep apnea and insomnia, it appears to be an effective intervention for reducing insomnia severity for this patient group. **Support:** National Heart. Lung and Blood Institute Grant # 1R01HL130559-01A1

0511

PATTERNS OF CONCOMITANT OVER-THE-COUNTER, NATURAL PRODUCT AND PRESCRIPTION SLEEP AID USE: A POPULATION-BASED STUDY

Cheung, J. M.^{1,2,3} Jarrin, D. C.² Beaulieu-Bonneau, S.^{2,4} Ivers, H.^{2,3} Morin, G.² Morin, C. M.^{2,3}

¹School of Pharmacy, Faculty of Medicine and Health, The University of Sydney, Sydney, New South Wales, AUSTRALIA, ²École de psychologie, Université Laval, Quebec CIty, QC, CANADA, ³Centre d'étude des troubles du sommeil, Institut universitaire en santé mentale de Québec, Quebec City, QC, CANADA, ⁴Centre interdisciplinaire de recherche en réadaptation et intégration sociale, Quebec CIty, QC, CANADA.

Introduction: Despite limited evidence, over-the-counter medications (OTC) and natural products (NP) are increasingly combined with prescribed medications (Rx) to manage insomnia symptoms. Self-medication patterns are expected to be heterogenous and may predispose individuals to inappropriate medication-taking behaviors, but little is known about the usage trajectories of sleep aids. This study investigates patterns of concomitant NP, OTC and Rx use in a Canadian population-based sample.

Methods: Data were derived from a longitudinal study on the natural history of insomnia. Participants were 3416 adults (62% female, $M_{age} = 49.7$, $M_{Insomnia\ Severity\ Index} = 8.4$). Self-reported data for OTC, NP and Rx use in the last year (yes/no) was extracted at 0-, 6- and 12-month follow-up. A Latent Class Growth Curve Analysis was conducted to identify patterns of concomitant sleep aid use. Participants also completed a battery of clinical measures including the Ford Insomnia Response to Stress Test, Dysfunctional Beliefs and Attitudes about Sleep scale (16-item), Beck Depression Inventory, Insomnia Severity Index and the

Pittsburgh Sleep Quality Index. Preliminary associations between class membership and baseline covariates were evaluated using the χ^2 test or a one-way ANOVA. Sampling weights were applied to all analyses, adjusting for partial non-response.

Results: Analyses revealed a 6-class solution; each class reflected a preferential agent(s) choice, which remained stable over 12-months: Minimal Use (74.5%), Rx-Dominant (11.3%), NP-Dominant (6.3%), OTC-Dominant (4.3%), Rx-NP-Dominant (2.4%), and Rx-OTC-Dominant (1.1%). Classes with prominent prescribed agent use were older [F(5, 207.6) =27.2, p<0.001], more likely to seek help [$\chi^2(5, n=2977) = 653.1, p < 0.001$] and consume alcohol [$\chi^2(5, n=2968) = 49.2, p < 0.001$]. Clinically, these individuals reported greater stress reactivity [F(5, 2966) =48.4, p < 0.001], depressive symptoms [F(5,197.4) =32.0, p < 0.001], dysfunctional sleep beliefs [F(5, 2987) =54.3, p < 0.001], insomnia severity [F(5, 2983) =88.4, p < 0.001] and poorer sleep quality [F(5, 203.8) =124.2, p < 0.001].

Conclusion: A majority of adults used agents minimally. Stability of medication-taking patterns suggests that individuals adopt less sporadic approaches when combining sleep aids than previously assumed. Clinical profiles and sleep aid choice could pre-empt vulnerabilities to inappropriate self-medication.

Support: Research supported by a grant from the Canadian Institutes of Health Research (MOP#115103)

0512

IMPACT OF A PATIENT DECISION-AID WHEN SELECTING INSOMNIA TREATMENTS AND FACTORS ASSOCIATED WITH DECISIONAL CONFLICT: PRELIMINARY FINDINGS FROM AN ONGOING PRAGMATIC CLINICAL TRIAL

Cheung, J. M.^{1,2,3} *Ji, X.*^{2,3} *Ivers, H.*^{2,3} *Morin, C. M.*^{2,3} ¹School of Pharmacy, Faculty of Medicine and Health, The University of Sydney, Sydney, New South Wales, AUSTRALIA, ²École de psychologie, Université Laval, Quebec City, QC, CANADA, ³Centre d'étude des troubles du sommeil, Institut universitaire en santé mentale de Québec, Quebec City, QC, CANADA.

Introduction: Preferences play an important role in determining insomnia treatment outcomes, but the validity of patient choice is rarely assessed. Uninformed preferences can lead to decisional conflict, which can negatively impact on treatment initiation, adherence, and subsequent outcomes. The current study aims to evaluate the impact of integrating a patient decision-aid as part of a pragmatic clinical trial and to identify baseline covariates associated with clinically significant decisional conflict (CSDC).

Methods: Secondary analysis of an ongoing pragmatic clinical trial for a two-stage cognitive behavioral therapy for insomnia (CBT-I) intervention was undertaken. Participants were referred from primary care clinics in Quebec City, Canada. Upon enrolment, participants were guided by a decision-aid, outlining the risks and benefits of prospective treatment options, when selecting their preferred arm of treatment in Phase 1. Options included SHUTi, SHUTi combined with an existing medication or continuing usual treatment with medication alone. Participants also completed a battery of sleep and mental health measures at baseline. Prior to treatment initiation, the 4-item SURE (Sure of myself; Understand information; Risk-Benefit ratio; Encouragement) scale was administered to screen for CSDC. Relationships between CSDC and baseline covariates were explored using Pearson correlations.

Results: Of the 55 participants initially enrolled, 94.5% (n=52) of participants preferentially selected SHUTi, either as sole treatment (n=24) or in combination with an existing medication (n=28), over usual treatment with medication alone (n=3). Overall, CSDC was only reported by 5.5% (n=3) of the sample population, with no group differences observed, suggesting effective clarification of treatment options through the decision-aid. Interestingly, higher SURE scores (i.e. less decisional conflict) were negatively correlated with depressive symptoms (r= -0.295, n= 55, p= 0.029) and anxiety symptoms (r= -0.301, n= 55, p= 0.026). Correlations with age, insomnia symptoms, duration of insomnia and fatigue were not statistically significant.

Conclusion: The patient decision-aid appeared to resolve decisional conflict for 94.5% (n=52) of participants. Findings allude to the potential influence of emotional status on information processing pathways in an insomnia context, warranting further research. **Support:** Research supported by a grant from the Canadian Institutes of Health Research (CIHR-IRSC:0441002152).

0513

COMPARISON OF PATIENT SATISFACTION AND THERAPEUTIC ALLIANCE FOR TELEMEDICINE VS. FACE-TO-FACE DELIVERED COGNITIVE BEHAVIORAL THERAPY FOR INSOMNIA

Conroy, D. A. Mooney, A. Pace, D. Balstad, S. Dubuc, K. Yang, A. Furgal, A. Sen, A. Arnedt, J. University of Michigan, Ann Arbor, MI.

Introduction: CBT for insomnia (CBTI) is effective but a barrier to its widespread use is the lack of evidence-based delivery modalities other than face-to-face. The perception and acceptability of telemedicine for the delivery of CBTI is unknown. We conducted a randomized controlled non-inferiority trial comparing face-to-face (F2F) and telemedicine (via AASM SleepTM) delivery of CBTI. We compared measures of patient satisfaction with treatment and the perception of the therapist's warmth and skills between F2F and SleepTM.

Methods: Adults with insomnia were recruited from insomnia clinics and the community and screened for sleep, medical, and mental health disorders. Eligible participants were randomized to receive CBTI either via AASM SleepTM or F2F in 6 weekly sessions of 45-60 minutes each. Participants completed the Client Satisfaction Questionnaire (CSQ-8) *and* The Therapy Evaluation Questionnaire (TEQ) after completing treatment. The CSQ-8 score ranges from 8-32 with high scores indicating greater satisfaction. We also analyzed the two items on the TEQ that assess participants' perception of therapist's warmth and skills. Item scores ranged from 1-7, with higher scores indicating greater warmth and skills.

Results: Sixty-five adults with chronic insomnia were recruited primarily from insomnia clinics. Sixty-two participants (41 women, mean age 48.9 \pm 15.4 years) completed all 6 sessions of CBTI via F2F (n=32) or via AASM SleepTM (n=30). Independent samples t-tests revealed no significant differences between conditions on patient satisfaction (SleepTM, 28.5 +/-4.2 vs F2F 29.9 +/-2.4, t(-1.5), p=.14), therapist warmth (SleepTM, 6.0 \pm 1.1 vs F2F, 6.4 \pm 0.95, t(-1.4), p=.16), or therapist skills (Sleep TM 6.4 \pm 1.0 vs F2F, 6.7 \pm 0.59, t(-1.5), p=.15).

Conclusion: Our findings suggest no differences in patient satisfaction, perception of therapist's warmth, or confidence in therapist's skills between telemedicine (via the AASM SleepTM) and F2F delivery of CBTI. Telemedicine-delivered CBTI should be implemented more widely.

Support: Research supported by American Sleep Medicine Foundation Grant # 168-SR-17 (JT Arnedt)

0514

EFFICACY OF A FOREHEAD-COOLING DEVICE FOR TREATING INSOMNIA IN VETERANS

Nofzinger, E.^{1,2}

¹Ebb Therapeutics, Pittsburgh, PA, ²University of Pittsburgh, Pittsburgh, PA.

Introduction: In 2 independent studies, we explored whether a forehead-cooling device was effective in improving insomnia in veterans.

Methods: Both studies were uncontrolled and exploratory in nature. The first study involved 20 veterans who expressed interest in using the forehead-cooling device and received 4 weeks treatment. The second study involved 19 veterans who were recruited via media to participate in a 4-week study and were compensated for their participation. All participants completed questionnaires before and after treatment.

Results: In the retrospective analysis, veterans had improvements over baseline in insomnia severity index (M ± SD =17.6 ± 4.7 pre- vs 6.9 ± 3.5 post-treatment, t(19) = -9.4, p<0.00001), in sleep latency (M ± SD = 61.7 ± 49.1 minutes pre- vs 25.0 ± 20.8 minutes post-treatment, t(19) = -4.6, p<0.001) and in minutes awake after sleep onset (M ± SD =78.7 ± 57.8 minutes pre- vs 29.9 ± 18.3 minutes post-treatment, t(19) = -4.0, p<0.001). In the prospective study, veterans had improvements in insomnia severity index over baseline (M ± SD = 20.7 +3.8 pre- vs 9.5 ± 7.5 post-treatment, t(18) = 5.8, p<0.00001), depression severity on the PHQ-9 (M ± SD = 21.5 ± 6.1 pre- vs 14.2 ± 5.1 post-treatment, t(18) = 4.1, p<0.001 and anxiety severity on the GAD 7 (M ± SD = 9.8 ± 7.1 pre- vs. 6.2 ± 5.4 post-treatment, t(18) = -3.1, p<0.01).

Conclusion: Use of a forehead-cooling device improved insomnia in veterans. These findings were replicated in an independent prospective trial. Reductions in depressive and anxiety symptoms from baseline were also noted in the prospective study. These promising preliminary data suggest the need for further large scale randomized controlled trials to establish the efficacy of forehead-cooling on insomnia in veterans.

Support: Ebb Pharmaceuticals, Pittsburgh, PA 15222

0515

DURABILITY OF EFFECTS OF FOREHEAD COOLING ON EEG SLEEP MEASURES IN INSOMNIA PATIENTS FROM 2- TO 30-NIGHTS USE AND 6-MONTH SAFETY RESULTS

Rippole, D.¹ Schirm, J.¹ Nofzinger, E.^{1,2}

¹Ebb Therapeutics, Pittsburgh, PA, ²University of Pittsburgh, Pittsburgh, PA.

Introduction: Forehead cooling has previously been shown to improve EEG sleep measures in insomnia patients when applied for 2 nights. The current study assessed the durability of these effects after 30 days in home use as well as safety over 6 months in home use.

Methods: This was a prospective, open label trial involving 32 adults meeting diagnostic criteria for primary insomnia who previously had participated in a 2 night in lab EEG sleep study. In the current study, participants received an additional 30 nights in home treatment, then had repeat EEG sleep studies performed

to determine if effects noted at 2 nights remained durable after 30 days. Subjects also participated in an open label 6-month in-home use safety study.

Results: Baseline, 2-night and 30-night EEG sleep measures for sleep latency were 80.7 ± 73.8 , 25.3 ± 22.6 , 26.2 ± 25.8 minutes (2- to 30-night difference p=0.81, NS) and for sleep efficiency were 67.4 ± 15.7 , 81.4 ± 11.2 , 83.2 ± 13.6 (2- to 30-night difference p=0.18, NS). Subjective sleep quality (0-100 scale with 100=best) at baseline, 2-night and 30-nights were 29.8 ± 15.2 , 48.3 ± 20.2 , 57.2 ± 21.1 (linear improvements significant over time p<0.001). No adverse effects were seen across 6-months use.

Conclusion: Forehead cooling demonstrated durability of effects on EEG sleep measures from 2- to 30-nights use. Continuing improvements in subjective sleep quality when measured over time from baseline assessments to the end of the 30-night in-home use period were noted. Forehead cooling was safe over 6 months use in the home as evidenced by no serious device related adverse events. **Support:** Ebb Therapeutics, Pittsburgh, PA 15222

0516

CHINESE PATIENTS WITH INSOMNIA RECRUITED FROM TRADITIONAL CHINESE MEDICINE (TCM) HOSPITAL SUCCESSFULLY TREATED WITH TELEHEALTH GROUP CBT-I IN MANDARIN

Xu, Y.¹ Barwick, F.² Li, C.³

¹Mind & Body Garden Psychology, Los Altos, CA, ²Stanford University School of Medicine, Dept of Psychiatry & Behavioral Sciences - Division of Sleep Medicine, Redwood City, CA, ³Chongqing Traditional Chinese Medicine Hospital, Chongqing, CHINA.

Introduction: Cognitive Behavioral Therapy for Insomnia (CBTI) is the recommended treatment for insomnia in the United States, Europe and China. Individual, group and online formats are effective, with moderate to large effect sizes in treatment outcomes. Although the prevalence of insomnia is 15% in China, most individuals do not seek CBTI and often prefer TCM. This study investigated whether CBTI delivered to Chinese in Mandarin via group telehealth showed similar outcomes compared to other countries. Methods: Chinese patients > 18 years with insomnia > 7 on the ISI were recruited from a sleep clinic in collaboration with Chongqing TCM Hospital in China. CBTI was delivered via 4-session telehealth group. Components included sleep education, sleep restriction, stimulus control, cognitive reframing, and relaxation. TCM use was extracted from medical records. Scores on self-report questionnaires assessing insomnia symptoms, sleep effort, sleep beliefs, and depression were compared pre- and post-treatment using paired sample t-tests with correction for multiple comparisons.

Results: All patients (N=34) completed pre-post measures. Mean age was 44 years, sample was 82% female, and 91% had a bachelor or advanced degree. Average insomnia duration was 4.1 years, and 80% of sample used TCM during CBTI. Post-treatment outcomes showed significant decreases in insomnia symptoms (ISI M_{diff} =4.0, SD_{diff}=6.4, p=.001, ES=.63), sleep effort (GSES M_{diff} =2.3, SD_{diff}=3.2, p=.000, ES=.72), and unhelpful sleep beliefs (DBAS M_{diff} =5.4, SD_{diff}=5.4, p=.000, ES=1.0), along with significantly lower depression symptoms (PHQ9 M_{diff} =2.6, SD=3.8, p=.000, ES=.68) and improvement in daytime functioning (PSQI M_{diff} =0.6 SD_{diff}=1.2, p=.008, SE=.50). Medication use also decreased significantly (PSQI M_{diff} =1.5 SD_{diff}=1.6, p=.000).

Conclusion: Chinese patients with insomnia can be recruited successfully and treated effectively using CBTI via telehealth. Outcomes and effect sizes are similar to those seen with CBTI in other countries. Future comparison of CBTI with and without concurrent TCM could provide clinically relevant information.

Support: None

0517

INTEGRATED COGNITIVE BEHAVIORAL THERAPY (CBT) AND MINDFULNESS GROUP TREATMENT PROTOCOL FOR INSOMNIA AND CHRONIC PAIN

Barwick, F.¹ Poupore-King, H.² You, D.²

¹Department of Psychiatry & Behavioral Sciences - Division of Sleep Medicine, Stanford University School of Medicine, Redwood City, CA, ²Department of Anesthesiology, Stanford University School of Medicine, Redwood City, CA.

Introduction: Chronic pain and insomnia are highly comorbid, and CBT is a recommended treatment for both. CBT protocols that treat these conditions together, however, show improvements in sleep but not pain. As mindfulness, an acceptance-based approach, has been used successfully to treat chronic pain, integrating mindfulness into a combined CBT treatment protocol may help improve outcomes for chronic pain as well as insomnia.

Methods: An integrated CBT/Mindfulness weekly 6-session group protocol for chronic pain and insomnia was developed and piloted. Treatment components included education about pain neuroscience as well as sleep and circadian biology, relaxation, time-based pacing, tracking 24-hour time in bed, sleep compression, stimulus control, cognitive reframing, and mindfulness. Pre-post measures evaluating insomnia symptoms, sleep hygiene, pain acceptance, pain catastrophizing, and unhelpful beliefs about sleep and pain were analyzed using frequency analyses and paired sample t-tests.

Results: Two groups were completed for a total of 16 participants, 94% of whom attended at least 5 sessions. Average age was 56 years, 75% of the sample was female, 88% were White, 6% Asian, and 6% Latino. Post-treatment outcomes showed significant improvement in insomnia symptoms (ISI M_{diff} =6.6, SD_{diff} =5.3, p=.01, ES=1.2), sleep hygiene (SHI M_{diff} =3.8, SD_{diff} =4.6, p=.02, ES=.83), pain acceptance (CPAQ M_{diff} =5.2, SD_{diff} =7.8, p=.03, ES=.67), pain catastrophizing (PCS M_{diff} =5.1, SD_{diff} =7.5, p=.03, ES=.68), and unhelpful beliefs about sleep (DBAS M_{diff} =31.4, SD_{diff} =21.2, p=.009, ES=1.5) and pain (PBAS M_{diff} =11.6, SD_{diff} =10.7, p=.02, ES=1.1).

Conclusion: An integrated CBT/Mindfulness group protocol for chronic pain and insomnia showed significant improvements in post-treatment sleep and pain measures. As previous combined CBT-only protocols showed pre-post improvement in sleep but not pain, the current study demonstrates that including mindfulness might improve outcomes for chronic pain. Future studies should compare CBT protocols for chronic pain and insomnia with and without mindfulness to determine the clinical benefits of including an acceptancebased component.

Support: Poster presented as part of collaborative conversation with Skye Margolies, PhD, Department of Anesthesiology, University of North Carolina School of Medicine.

0518

SLEEP FACILITATION BY ARTIFICIAL CARBONATED BATHING IN HEALTHY ELDERLY; EEG, CORE, PROXIMAL, AND DISTAL TEMPERATURE EVALUATIONS Uemura, S. I.¹ Kanbayashi, T.² Imanishi, A.³ Terui, Y.¹ Satake, M.¹ Shioya, T.⁴ Nishino, S.⁵

¹Akita University Graduate School of Health Sciences, Akita, JAPAN, ²International Institute for integrative sleep medicine, University of Tsukuba, Tsukuba, JAPAN, ³Akita University Graduate School of Medicine, Akita, JAPAN, ⁴Geriatric Health Services Facility Nikonikoen, Akita, JAPAN, ⁵Stanford University, Psychiatry and Behavioral Sciences - Sleep & Circadian Neurobiology Laboratory, Palo Alto, CA.

Introduction: Bathing, especially with hot spring with various mineral compositions, is known to facilitate / improve sleep by warming the body. Artificial carbonated bathing (ACB) is known to keep the body warm too. Previous our study examined that ACB before sleep more specifically affected body temperature and sleep on healthy young subjects. In this study, we evaluated the effects of usual (plain hot water; PH) and artificial carbonated bathing, on sleep using clinical thermometers and EEG in healthy elderly subjects.

Methods: Nine healthy elderly women (average age 71.3 years old) were divided into 2 groups ACB (858 ppm, Awacomachi, Danrei Co.) and PH with a week interval. Subjects soaked in the bath (38 C degree) deep enough their chests touched the water for 10 min. From the time they finished bathing to the next morning, we measured their distal skin temperature (top side of the foot), proximal skin temperature (lower part of the clavicle) and EEG using a single channel portable device (Brain wave sensor, Proassist Co.). Subjects were told to sleep from 23:00-6:00. As the same time, subjects were examined with visual analog scale (VAS) and clinical flicker fusion test (CFF).

Results: There was no significant difference in body temperature (proximal, distal, distal-proximal temperature gradient: DPG) before and during sleep between ACB and PH. The condition of the ACB tended to have less light sleep compared to PH (150min vs 201min, p=0.08), but there was no significant difference in the SWS (44.1min vs 39.1min, ns), EEG delta power analysis, VAS and CFF. In the previous study, bathing was performed for 15 min at a water temperature of 40 C degree, but this condition was considered to be a high risk for the elderly. The reason why there was no significant difference in body temperature. The effects of ACB were not fully demonstrated due to changes in hot water temperature and bathing time.

Conclusion: The effect of ACB on the body temperature of healthy elderly people could not be confirmed. However, a tendency to reduce light sleep was observed.

Support: This work was supported by JSPS KAKENHI Grant Number JP19K11294

0519

MINDFULNESS BASED THERAPY FOR INSOMNIA IMPROVES OBJECTIVE MARKERS OF SLEEP IN THE ELDERLY: PRELIMINARY DATA FROM THE MINDFULNESS SLEEP THERAPY (MIST) STUDY

Wong, K. F.¹ Perini, F.¹ Henderson, S. L.² Teng, J.¹ Hassirim, Z.¹ Lin, J.¹ Leow, Z.¹ Fan, Q.² Ong, J.¹ Lo, J.³ Ong, J. C.⁴ Doshi, K.² Lim, J.¹

¹Duke NUS Medical School, Singapore, SINGAPORE,

²Singapore General Hospital, Singapore, SINGAPORE, ³National University of Singapore, Singapore, SINGAPORE, ⁴Feinberg School of Medicine, Northwestern University, Chicago, IL.

Introduction: Mindfulness-based treatment for insomnia (MBTI) is a viable intervention for improving poor sleep. We report preliminary data from an ongoing pre-registered, randomized controlled trial which investigates the effect of MBTI on elderly adults.

Methods: Participants above 50 years old with $PSQI \ge 5$ were recruited and randomised into either MBTI or an active control group (Sleep hygiene education and exercise program, SHEEP) in sequential cohorts with about 20 participants per cohort (10 per group). Before and after the intervention, 1 night of portable polysomnography (PSG) and 1 week of actigraphy (ACT) and sleep diary (DIARY) data were collected. We report the ACT and DIARY results of the first 3 cohorts (n = 46, male = 23, mean age = 62.3, std = 6.3) and PSG data of the first 2 cohorts (n = 29, male = 12, mean age = 62.5, std = 5.7). Time in bed (TIB), total sleep time (TST), sleep onset latency (SOL), wake after sleep onset (WASO), and sleep efficiency (SE) were analysed with mixed-model repeated-measures ANOVA.

Results: We observed increases in TIB_{DIARY} ($F_{1,44} = 5.151$, p < .05) and SE_{DIARY} ($F_{1,44} = 22.633$, p < .0001), and significant reductions in SOL_{DIARY} ($F_{1,44} = 7.031$, p < .05) and WASO_{DIARY} ($F_{1,39} = 7.411$, p < .05). In the actigraphy data, we found a significant interaction in SOL_{ACT} ($F_{1,39} = 4.273$, p < .05) with an increase in SHEEP SOL_{ACT} ($t_{18} = 2.36$, p < .05). Significant reductions were also observed in WASO_{ACT} ($F_{1,44} = 16.459$, p < .0001) Finally, we observed a reduction in SOL_{PSG} ($F_{1,26} = 5.037$, p < .05). All other tests were non-significant.

Conclusion: Preliminary results suggest that both interventions lead to improvements in sleep with more pronounced effects in subjective sleep reports. Objective sleep data suggest that improvements in sleep is a result of improved sleep quality and not simply extending sleep opportunity. These preliminary data shows that MBTI may be a promising intervention for elderly individuals with sleep difficulties.

Support: This study was supported by an award from the 7th grant call of the Singapore Millennium Foundation Research Grant Programme

0520

TREATING SLEEP MAINTENANCE INSOMNIA MEANS TREATING DEPRESSION?

Batta, B.¹ Szakacs, Z.²

¹Medical Centre Hungarian Defence Forces, Budapest, Hungary, Budapest, HUNGARY, ²Medical Centre Hungarian Defence Forces, Budapest, HUNGARY.

Introduction: Rumination whilst awakening nightly might be a common symptom in fragmented sleep and depression as well. The problem is that the test results on BECK or MMPI DEPRESSION SCALE often do not reach the diagnostic criteria of depression on DSM-V, only the symptoms of worry, rumination and sleep maintenance problems appear as mild depressive signs. The aim of our study was to investigate the assumption to which treating everyday worries through CBT methods would affect the amount and severity of nightly rumination and the severity of depressive mood as well.

Methods: 33 adult patients (M=39,55 SD=10,66) diagnosed with insomnia with the criteria of only sleep maintenance proble. Each

participants filled the PSQI, the Epworth Sleepiness Scale, the Beck Depression Inventory and the Trait-Anxiety Inventory before the beginning of the CBT therapy session and 10 sessions later.

Results: Our results show a significant effect of the CBT therapy to sleep maintenance symptoms through treating everyday ruminations opposite to treating only nightly ruminations on sleeping quality (F(1, 31)= 55,358 p<0,01 η 2 = 0,641). The significant improvement in sleep quality only were shown in the subgroup of everyday rumination CBT group (N=17, M=7,88 SD=2,57), but not in the subgroup of nightly ruminations CBT group (N=16, M=9,87, SD=1,92).

Conclusion: Based on our preliminary findings, treating everyday worries with CBT therapy affect sleep maintenance insomnia, while treating only nightly ruminations and worries of sleeping difficulties would not improve sleeping problems and depressive mood. **Support:** No

0521

DARIDOREXANT (ACT-541468), A DUAL OREXIN RECEPTOR ANTAGONIST FOR THE TREATMENT OF INSOMNIA DISORDER: DOUBLE BLIND, RANDOMIZED, PHASE 3 STUDIES FOR EFFICACY AND SAFETY IN ADULT AND ELDERLY PATIENTS

Zammit, G.¹ Seboek Kinter, D.² Bassetti, C.³ Leger, D.⁴ Hermann, V.² Pain, S.² Roth, T.⁵

¹Clinilabs, Inc., New York, NY, ²Idorsia Pharmeceuticals Ltd., Allschwil, SWITZERLAND, ³Inselspital Universitatsklinik für Neurologie, Bern, SWITZERLAND, ⁴Universite Paris Descartes AP-HP, Paris, FRANCE, ⁵Division of Sleep Medicine and Research Center, Henry Ford Health System, Detroit, MI.

Introduction: Daridorexant, a potent and selective orally administered dual orexin receptor antagonist (DORA), has shown dosedependent efficacy and is well tolerated with minimal residual next-morning effects in two phase 2 studies in adult and elderly subjects with insomnia disorder. Following the favorable phase 2 results, clinical development was pursued with two pivotal phase 3 multi-center, double-blind, randomized, placebo-controlled studies to further assess efficacy and safety in adult and elderly subjects with insomnia disorder. Long-term safety and tolerability are being further evaluated in a double-blind placebo-controlled extension study.

Methods: Each of the pivotal studies include ~900 patients (~40% ≥65y), randomized 1:1:1 to one of two daridorexant arms or placebo. The studies differ in dose only (10 or 25 mg [NCT03545191], 25 or 50 mg [NCT03575104]). Both report objective primary outcomes at 1 and 3 months based on PSG (WASO and LSP). Secondary endpoints include self-reported nighttime benefit with Total Sleep Time (sTST), and daytime benefit using the validated Insomnia Daytime Symptoms and Impacts Questionnaire (IDSIQ). The patients undergo screening (7-13 d) and run-in (7-18 d) periods establishing eligibility and baseline, a 3-month double-blind treatment period, followed by a placebo run-out (7 d) to evaluate rebound insomnia and withdrawal effects and a 30-day safety follow-up. Additionally, subjects completing treatment could enroll in the 40-week double-blinded placebo-controlled extension trial [NCT03679884] to assess long-term safety.

Results: Enrollment in NCT03575104 (25/50 mg) was successfully completed and involves 76 sites across 10 countries; expected completion March 2020. Recruitment to study NCT03545191 (10/25 mg) is advanced; completion expected June 2020.

Conclusion: The comprehensive daridorexant phase 3 program includes 3 dose levels and replication of objective and subjective measurements at 1 and 3 months, while assessing self-reported nighttime benefit, and benefit during the day with a validated PRO instrument, as well as safety in insomnia disorder.

Support: Medical writing Randall Watson, (Idorsia). These studies were sponsored by Idorsia Pharmaceuticals Ltd.

0522

SUBTYPES OF INSOMNIACS TREATED BY COGNITIVE-BEHAVIORAL THERAPY

Sforza, M.¹ Castronovo, V.² Galbiati, A.¹ Zucconi, M.² Oldani, A.² Casoni, F.² Ferini-Strambi, L.¹

¹Sleep Disorder Center, Vita-Salute San Raffaele University, Milan, ITALY, ²Sleep Disorder Center, San Raffaele Hospital, Milan, ITALY.

Introduction: Insomnia disorder (ID) is characterized by high degree of heterogeneity. Aim of our study was to identify ID patients subtypes in terms of sleep and non-sleep clinical baseline (BL) features and CBT-I efficacy.

Methods: 294 chronic insomnia patients (61.6% female, mean age 40.7 \pm 12.3 yrs) underwent 7-sessions group CBT-I. By use of latent class analysis (LCA) we identified ID subtypes according to BL score of Glasgow Sleep Effort Scale (GSES); Epworth Sleepiness Scale (ESS); Dysfunctional Beliefs and Attitudes about Sleep (DBAS -16); Morningness-Eveningness Questionnaire Self-Assessment (MEQ-SA); Perceived Stress Scale (PSS); Profile of Mood States (POMS); Beck Depression Inventory (BDI-II); Stay-Trait Anxiety Inventory (STAI-Y); Treatment effectiveness (Delta score of Insomnia Severity Index ISI between BL and end-of-treatment).

Results: We chose 3 latent classes as most parsimonious model. According to questionnaires' cut-off, we labeled three classes: Class 1 (insomnia+anxiety+depression+stress) (n=62), Class 2 (insomnia+anxiety+depression) (n=153), Class 3 (only insomnia) (n=79). The variables that best differentiate the 3 classes were POMS (.772), STAY (.660), PSS (.545), BDI (.406) and ISI (.228) at BL. In particular, for ISI, the best item predicting groups differentiation was item 3 on the impact of insomnia on daytime functioning (.224). Moreover, we found a significant interaction between CBT-I treatment effect and the 3 classes at the ISI score (p=.001), GSES score (p=.002), DBAS score (p<.05), PSS score (p<.001), POMS score (p<.001), BDI score (p<.001) and STAI-Y score (p<.001).

Conclusion: Our data driven analysis results suggest that the heterogeneity of ID patients can be best represented by non-sleep scores, in particular those regarding depression, anxiety, stress and daytime functioning. These information can be useful in predicting the outcome of CBT-I. **Support:** No

0523

A PILOT, PLACEBO-CONTROLLED TRIAL OF CLOSED-LOOP, ARTIFICIAL INTELLIGENCE DRIVEN, ACOUSTIC NEUROMODULATION FOR INSOMNIA

*Tegeler, C. H.*¹ *Tegeler, C. L.*¹ *Howard, L. J.*¹ *Brown, K. L.*¹ *Kellar, D. C.*¹ *Gerdes, L.*³ *Shaltout, H. A.*⁴

¹Department of Neurology, Wake Forest School of Medicine, Winston-Salem, NC, ²Department of Neurology, Wake Forest School of Medicine, Winston-Salem, NC, ³Brain State Technologies, Scottsdale, AZ, ⁴Hypertension and Vascular Research Center, Wake Forest School of Medicine, Winston-Salem, NC.

SLEEP, Volume 43, Abstract Supplement, 2020

Introduction: In prior studies, High-resolution, relational, resonance-based, electroencephalic mirroring (HIRREM®) reduced symptoms of insomnia, and improved heart rate variability (HRV), but is operator dependent, and difficult to scale. Cereset ResearchTM (CR), is a noninvasive, closed-loop, artificial intelligence (AI) driven, acoustic neuromodulation technology. CR uses the same core technology, echoing tones linked to brainwaves, but includes updated components, standardized AI driven protocols, software management of designs, and shorter sessions to improve scalability. This controlled trial explores use of CR for insomnia.

Methods: Adults with insomnia (Insomnia Severity Index, ISI, of ≥ 8 points for ≥ 1 month) receive ten 60 minute sessions of tones linked to brainwaves (CR), versus random tones (RT). Data is collected at baseline (V1), 0-14 days (V2), and 6-8 weeks (V3) after intervention. Primary outcome is change in ISI at V3. Secondary outcomes include HRV (SDNN and rMSSD) based on 10-minute BP and HR recordings using a BIOPAC device. RT subjects can cross-over to CR after V3. Formal analysis of insomnia outcome awaits the target of n = 20 to complete intervention, but we report preliminary changes in ISI, and secondary outcomes, SDNN and rMSSD.

Results: 21 subjects have enrolled (15 women), with one dropout after first session and one after 6th session due to job changes. For n = 17, change in median ISI score from V1 to V3 is -7 for CR, and -4 for RT. Mean SDNN increased 32.2% (SE 12.8) for CR, and 5.6% (14.7) for RT, while rMSSD increased 88.8% (36.7) for CR, and 33.7% (38.7) for RT, with no serious adverse events reported.

Conclusion: Preliminary results suggest similar, clinically meaningful reductions in ISI score, and increased HRV with CR, as seen with HIRREM, suggesting promise as a scalable, non-drug intervention for insomnia with accompanying impact on autonomic function. Final results will be presented.

Support: Research grant from, The Susanne Marcus Collins Foundation, Inc.

0524

WEB-DELIVERED CBT FOR INSOMNIA INTERVENTION IMPROVES SLEEP AMONG ADULTS WITH INSOMNIA AND DEPRESSIVE SYMPTOMS

Batterham, P. J.¹ Christensen, H.² Thorndike, F. P.³ Ritterband, L. M.⁴ Gerwien, R.³ Enman, N.³ Botbyl, J.⁵ Maricich, Y.³

¹Australian National University, Canberra, AUSTRALIA, ²University of New South Wales, Randwick NSW, AUSTRALIA, ³Pear Therapeutics, Boston, MA, ⁴University of Virginia, Charlottesville, VA, ⁵Provonix, Sewell, NJ.

Introduction: Cognitive behavioral therapy for insomnia (CBT-I) is the first line recommended treatment for adults with chronic insomnia. In a prior randomized controlled trial (RCT), data showed web-delivered CBT-I (SHUTi) reduced insomnia severity as well as symptoms of depression, among adults with insomnia and elevated depressive symptoms. The present study aimed to further evaluate the effectiveness of web CBT-I to improve sleep outcomes as measured by prospectively entered sleep diaries in this same sample.

Methods: A large-scale RCT (N=1149) of Australian adults with insomnia and depressive symptoms compared a 9-week, web CBT-I therapeutic with an attention-matched web program at baseline, posttest and 6-, 12-, and 18-month follow-ups. Although depression outcomes have been presented previously, the online sleep-diary derived variables have not yet been presented, including sleep-onset latency (SOL), wake after sleep onset (WASO), sleep

efficiency (SE), number of awakenings, sleep quality, and total sleep time (TST). Sleep diaries were entered online for 10 days at each assessment period.

Results: Data showed web CBT-I participants demonstrated greater reductions from baseline to posttest compared with control for the following sleep variables: SOL (LS mean difference [95% CI]=-22.3 min [-29.2, -15.3]; p<.0001), WASO (-17.8 min [-23.4, -12.3]; p<.0001), and number of awakenings (-0.38 [-0.68, -0.09]; p=.0113). Web CBT-I also showed greater improvements in SE (9.18% [7.25%, 11.10%]; p<.0001) and sleep quality (0.41 [0.30, 0.53]; p<.0001) from baseline to posttest compared with control. TST was not significantly different between groups at posttest or 6-month follow-up, although it improved over baseline at 12 (18.73 min [7.39, 30.07]; p=.0013) and 18 months (23.76 min [9.15, 38.36]; p=.0015) relative to control. All other significant sleep treatment effects were maintained in the treatment arm at 6, 12, and 18-month follow-up.

Conclusion: Data showed web CBT-I produced lasting improvements in sleep outcomes among adults with insomnia and elevated depressive symptoms.

Support: Clinical trial ACTRN12611000121965 was funded by the Australian National Health and Medical Research Council. The statistical analysis described here was funded by Pear Therapeutics, Inc and conducted by Provonix.

0525

SUBTYPES OF EFFICACY OF COGNITIVE-BEHAVIORAL THERAPY FOR INSOMNIA

Castronovo, V.¹ Sforza, M.² Galbiati, A.² Salsone, M.³ Marelli, S.¹ Ferini-Strambi, L.²

¹Sleep Disorder Center, San Raffaele Hospital, Milan, ITALY, ²Sleep Disorder Center, Vita-Salute San Raffaele University, Milan, ITALY, ³Institute of Molecular Bioimaging and Physiology, Catanzaro, ITALY.

Introduction: Cognitive-Behavioral Therapy for Insomnia (CBT-I) is the first-line treatment for Insomnia disorder (ID). We aimed to identify ID patients' subtypes based on clinical features and their response to CBT-I.

Methods: 294 chronic insomnia patients (61.6% female, mean age 40.7 ± 12.3 yrs) underwent 7-sessions group CBT-I. By use of latent class analysis (LCA) we identified insomnia disorder subtypes according to baseline (BL) evaluation of non-sleep indices and the response to CBT-I (Delta score of Insomnia Severity Index ISI between BL and end-of-treatment (ET). Moreover, we assessed ISI in 123 out of 294 insomnia patients (82 females (66.7%), mean age 40.59 ± 11.89 years) who completed a follow-up evaluation (FU) within a range of 4-10 years.

Results: We chose 3 latent classes as most parsimonious model. We identified Class 1 (insomnia+anxiety+depression+stress) (n=62), Class 2 (insomnia+anxiety+depression) (n=153) and Class 3 (only-insomnia) (n=79). The effect of CBT-I was maintained up to 10 years after the ET in the three classes but with significant difference between classes (p < 0.05). At the ET, the largest percentage of responders (ISI decrease ≥ 8) was found in Class 1 (63.5%). Results of overall CBT-I effectiveness: in Class 3, 98.6% had subthreshold insomnia (ISI score=0-14) at the ET, and 97.2% at the FU; in Class 2, 89.0% at the ET, and 78.2% at the FU; in Class 1, 80.7% at ET and 51.8% at the FU.

Conclusion: Our analysis identified three different subtypes of insomniacs on the basis of clinical outcomes. The presence of anxiety and depression did not diminish the effect of CBT-I both short and long term. However, ID patients characterized by the presence of stress (Class 1) were the best responders at the ET but this was not maintained at the FU evaluation. We can speculate that stress could be considered a risk factor that plays an important role in the long-term outcome of CBT-I. Support: No

0526

OBJECTIVE TOTAL SLEEP DURATION DO NOT PREDICTING THE EFFECTIVENESS OF COGNITIVE-**BEHAVIORAL THERAPY FOR INSOMNIA (CBT-I)**

Galbiati, A.¹ Sforza, M.¹ Leitner, C.¹ Filice, A.¹ Manconi, M.² Ferini Strambi, L.¹ Castronovo, V.³

¹Sleep Disorder Center, Vita-Salute San Raffaele University, Milan, ITALY, ²Sleep Center, Neurocenter of Southern Switzerland, Regional Hospital of Lugano, Lugano, SWITZERLAND, ³Sleep Disorder Center, San Raffaele Hospital, Milan, ITALY.

Introduction: Several studies investigated the role of objective sleep markers, in particular of Total Sleep Time (TST) in identifying different subtypes of Insomnia Disorder (ID) and in evaluating the efficacy of treatments. Based on objective TST two phenotypes of ID are usually distinguished in the literature: normal sleepers (objective sleep duration ≥ 6 hours) and short sleepers (objective sleep duration < 6 hours). Aim of our study was to evaluate in normal and short sleepers (objective sleep duration was assessed by both Polysomnography and Actigraphy) possible different response to Cognitive-Behavioral Therapy for Insomnia (CBT-I).

Methods: 53 ID patients (females = 50.9%; mean age = 56.53 ± 11.43) were divided into "Short Sleep duration" and "Normal Sleep duration" groups. All patients underwent 7-sessions group CBT-I. Main clinical outcome was Insomnia Severity Index questionnaire (ISI); secondary outcomes were Sleep Efficacy (SE), Sleep Latency (SL), Wake After Sleep Onset (WASO), Number of Awakenings (N°awk) according to sleep diaries.

Results: All ID patients showed significant improvements after treatment for all clinical outcomes. Non-significant effects of CBT-I between "Short Sleep duration" and "Normal Sleep duration" measured by patients were found in terms of ISI, SE, SL, WASO and N°awk, neither using Polysomnography nor Actigraphy. Furthermore, no accordance between these two objective measurements was found for the identification of the two subgroups.

Conclusion: Our findings suggest that the use of objective TST (both by Actigraphy and Polysomnography) is not a consistent predictor for CBT-I effectiveness. Moreover, only a small percentage of patients were classified as short or normal sleepers according both to Polysomnography and Actigraphy. These findings underline the instability and poor reliability of using objective TST in subtyping insomniacs.

Support: No

0527

THE UPTAKE OF A FREE DIGITAL CBTI PROGRAMME IN A LARGE COMMERCIAL ORGANISATION

Gardiner, A. Stanley, N.

Sleepstation, Newcastle, UNITED KINGDOM.

Introduction: CBTi is effective in the treatment of insomnia and is now recommended as the first-line treatment. However, despite the desirability of CBTi, access to therapy is restricted due to the lack of sufficient appropriately trained and experienced therapists.

Because of the lack of therapists and the financial and time costs associated with face to face therapy a number of programmes that offer CBTi digitally have been developed, which have been shown to have similar success rates to receiving therapy in person.

Methods: The uptake of Sleepstation www.sleepstation.org.uk, a clinically proven CBTi platform with additional human support, was investigated when it was offered free to the members of a large organisation in the UK. The availability of the programme was promoted via the organisation's website for 3 months.

Results: 1173 people registered an interest in the programme of which 880 were assessed for suitability (73% female, median age 45yrs). 411 where offered treatment due to symptoms indicative of insomnia. 188 initiated treatment. 137 complete the programme or reached recovery. 112 showed an improvement in their sleep.

Conclusion: Simply reporting the success rate of CBTi only tells part of the story. Simply improving access to CBTi, whether face to face or digitally, does not necessarily improve the initiation, retention, and completion of CBTi therapy. Further research is needed to fully understand the real and perceived barriers to the use of CBTi.

Support: This study was facilitated by Sleepstation

0528

INSOMNIA AS A MECHANISM FOR IMPROVEMENT IN ALCOHOL PROBLEMS AMONG YOUNG ADULTS

*Miller, M.*¹ *Freeman, L. B.*¹ *Park, C. J.*¹ *Hall, N.*¹ *Sahota, P. K.*¹ *McCrae, C. S.*¹

¹University of Missouri, Columbia, MO, ²University of Missouri, Columbia, MO.

Introduction: More than half of heavy-drinking young adults report symptoms of insomnia, which have been associated with alcohol-related problems. This study examined improvement in insomnia (via Cognitive Behavioral Therapy for Insomnia; CBT-I) as a mechanism for improvement in alcohol-related problems.

Methods: Fifty-six heavy-drinking young adults with insomnia (ages 18-30y) were randomized to CBT-I (n=28) or single-session sleep hygiene control (SH; n=28). Of those, 43 (77%) completed post-treatment (24 SH, 19 CBT-I) and 48 (86%) completed 1-month follow-up (25 SH, 23 CBT-I). Multiple imputation was used to estimate missing data. Treatment outcomes were assessed using multilevel models. Mediation was tested using bootstrapped confidence intervals for indirect effects in the PROCESS macro.

Results: CBT-I participants reported greater decreases in insomnia severity than those in the sleep hygiene group [group X time interaction, F(2,59)=11.29, p<.001], both post-treatment and at 1-month follow-up. Both groups decreased significantly in diary-assessed sleep quality [time, F(2,55)=40.30, p<.001], with a marginally significant interaction in favor of the CBT-I group [F(2,55)=2.69,p=.08]. There were no significant group by time interactions in the prediction of actigraphy-assessed sleep variables, although again, there was a marginally significant interaction in the prediction of actigraphy-assessed sleep efficiency [F(2,66)=2.75, p=.07]. Both groups reported significant decreases in drinking quantity over time [time, F(2,58=13.88, p<.001]. However, CBT-I participants reported greater decreases in alcohol-related consequences than those in the sleep hygiene group [F(2,67)=4.13, p=.02]. In the mediation model, CBT-I did not have a direct effect on change in alcohol-related consequences (B=1.49, SE=1.06, 95%CI=-0.65, 3.62); however, it influenced change in 1-month alcohol-related consequences indirectly through its influence on post-treatment insomnia symptoms (B=-1.09, SE=0.57, 95%CI=-2.30, -0.05).

Conclusion: CBT-I is effective in treating insomnia among heavydrinking young adults and may be associated with reductions in alcohol-related problems due to its impact on insomnia symptoms. **Support:** This work was supported by funding from the University of Missouri System Research Board Office (PI Miller). Mary Beth Miller's contribution to this project was also supported by the National Institute on Alcohol Abuse and Alcoholism [grant number K23AA026895].

0529

BEHAVIORAL THERAPY COMPONENTS FOR INSOMNIA AND FATIGUE IN COPD

Kapella, M.¹ Steffen, A.¹ Laghi, F.³ Prasad, B.¹ Vispute, S.⁴ Teixeira, C.⁵ Kemner, G.⁶ Peters, T.¹ Carley, D.¹ ¹University of Illinois at Chicago, Chicago, IL, ²University of Illinois at Chicago, Chicago, IL, ³Edward Hines, Jr. Department of Veterans Affairs Hospital, Hines, IL, IL, ⁴Rush University Medical Center, Chicago, IL, ⁵Illinois Sleep Counseling PLLC, Highland Park, IL, ⁶Howard Brown Health, Chicago, IL.

Introduction: Insomnia contributes to fatigue, a common symptom in COPD. Our study aims were: (1) to determine the efficacy of a) cognitive behavioral therapy for insomnia (CBT-I) and b) COPD education (COPD-ED) on insomnia and fatigue, and (2) to define potential mechanistic contributors to pre/post intervention change in insomnia and fatigue in patients with COPD and insomnia.

Methods: A randomized 2x2 factorial design was used with factors representing CBT-I (yes/no) and COPD-ED (yes/no). Attention control (health videos) were used in the absence of CBT-I or COPD-ED. All patients received 6, 75-minute weekly sessions. Dependent variables included insomnia severity (Sleep Impairment Index (SII), range 0-28) and fatigue (Chronic Respiratory Disease Questionnaire (CRQ) range 1-7) measured at baseline, just post-intervention, and at 3-months post-intervention.

Results: One hundred nine patients (FEV1% predicted $67 \pm 24\%$ (mean \pm SD), age 65 ± 8 years, SII 15.9 ± 8 , CRQ 3.7 ± 1.1) participated in the study. After 6 sessions, insomnia decreased more in patients who received CBT-I (-5.8) than those who did not (-2.2; p=0.0002). This effect was sustained at the 3-month follow-up (p=0.0003). Fatigue showed no significant differences for CBT-I at 6-weeks (p=.27) but at 3-months patients receiving CBT-I showed marginally better improvement (.75, a clinically important difference) compared to those who did not receive CBT-I (.43; p=.09). COPD-ED showed no effect on insomnia or fatigue. Two main effects suggest mechanisms for the pre-post efficacy of CBT-I: improved sleep beliefs (p=0.0257) and self-efficacy for sleep (p=0.0619) after 6 sessions which were sustained at 3 months (p=0.0184 and p=0.0431 respectively).

Conclusion: CBT-I produced sustained decreases in insomnia in patients with COPD. Results suggest that changes in beliefs about sleep and improved self-efficacy for managing sleep may mediate CBT-I associated decreases in insomnia.

Support: This research was supported by the National Institute of Nursing Research of the National Institutes of Health R01NR013937.

0530

HYBRID COGNITIVE BEHAVIOR THERAPY FOR INSOMNIA (CBT-I) AND ACCEPTANCE AND COMMITMENT THERAPY (ACT) GROUP TREATMENT MODEL FOR INSOMNIA AND CHRONIC PAIN

Ochsner Margolies, S. UNC, Chapel Hill, NC.

Introduction: Insomnia is a common complaint for individuals with chronic pain. CBT-I as an intervention for these patients shows strong improvement in sleep but not consistently in pain outcomes. Current treatment approaches for chronic pain focus increasingly on acceptance-based interventions. Integrating ACT into a CBT-I group protocol has the potential to optimize both sleep and pain outcomes.

Methods: A hybrid CBT-I/ACT 6-session weekly group protocol for chronic pain and insomnia was developed and piloted. CBT-I components included sleep education, stimulus control, and sleep restriction. ACT components included cognitive defusion, self-as-context, present moment awareness, mindfulness, and values-guided behavioral activation. Pre-post measures assessing insomnia symptoms, sleep parameters based on sleep diary, sleep catastrophizing, pain catastrophizing, pain acceptance, beliefs about pain and sleep, depression, and anxiety were analyzed using frequency analyses and paired sample t-tests.

Results: Group participants (4) recruited from an outpatient pain management clinic were on average 57 years old, 100% female and 75% White. Post-treatment, patients reported significantly improved insomnia symptoms (ISI Mdiff=5.8, SDdiff=3.9, p < .05, ES=1.5), sleep efficiency (SE, Mdiff=16%, SDdiff=10%, p = .05, ES=1.5), pain catastrophizing (PCS Mdiff=7.8, SDdiff=4.6, p < .05, ES=1.6), pain acceptance (CPAQ Mdiff=11.5, SDdiff=7.5, p = .05, ES=1.5), beliefs about the relationship between pain and sleep (PBAS Mdiff=2.3, SDdiff=1.3, p < .05, ES=1.8) and anxiety (GAD-7 Mdiff=3.3, SDdiff=2.1, p < .05, ES = 1.6).

Conclusion: Hybrid CBT-I/ACT group protocol for chronic pain and insomnia showed significant improvements in sleep and, more importantly, pain outcomes. This pilot study demonstrates the benefits of incorporating an ACT approach to optimize pain as well as sleep outcomes. Future efforts will continue to refine the CBT-I/ACT protocol in anticipation of conducting a dismantling study to determine the clinical benefits of adding an ACT framework to the CBT-I model.

Support: NA

0531

EFFICACY OF A SINGLE 4 HOUR CBT-I WORKSHOP IN A COMMUNITY SAMPLE WITH SELF-REPORTED INSOMNIA SYMPTOMS

Okun, M.¹ Glidewell, R.²

¹University of Colorado Colorado Springs, Colorado Springs, CO, ²The Insomnia Clinic, Colorado Springs, CO.

Introduction: Cognitive behavioral treatment for insomnia (CBT-I) is the first line of treatment for insomnia. However, experts have noted that the expanded use of CBT-I is limited by the small number of specialty-trained clinicians, as well as the duration and cost of individual treatment sessions (usually 6-8). One solution is a single-session educational group format delivered by a trained health educator rather than a licensed clinician. Our objective was to evaluate the efficacy of group CBT-I delivered by a Ph.D. level health educator to community dwelling individuals with self-reported insomnia symptoms.

Methods: Participants were referred from clinicians, our website, and social media postings. Participants completed the Insomnia Severity Index, provided information on type of sleep aid use and frequency, and the presence of co-morbid conditions prior to and 1-month post attendance of a single 4-hour CBT-I workshop.

Results: Participants (N = 31) were 58 ± 12 years of age (range 29 - 80); 11 Males, 20 Females; 90.6% white; 66% married; 71.8% at least a college graduate; and 34.3% had an average income of > \$100K. Comorbidities included pulmonary disease (6%), GI disease (9.6%), endocrine disease (9.6%), and headaches (25.8%). Insomnia Severity Index scores significantly improved from baseline (19.6 ± 5.06) to 1-month (FU 13.7 ± 6.33) (t = 21.9, P < .001)). Similarly, frequency of sleep aid use significantly dropped (χ^2 = 105.7, p = .017). Subjective improvement in sleep was reported as the following: 12.5% much better, 56.3% better, 25% the same, and .03% worse.

Conclusion: These data indicate that a single 4-hour CBT-I workshop delivered by a health educator can significantly reduce insomnia symptoms, improve subjective sleep quality, and reduce sleep aid use among community dwelling adults with self-reported insomnia symptoms within 1-month. These data extend what has been shown primarily in older adults. That is, brief behavioral treatment for insomnia can be acceptable and efficacious to anyone reporting insomnia symptoms.

Support: The Insomnia Clinic

0532

COGNITIVE BEHAVIORAL THERAPY DELIVERED VIA TELEMEDICINE VS. FACE-TO-FACE: RESULTS FROM A RANDOMIZED CONTROLLED NON-INFERIORITY TRIAL

Arnedt, J.¹ Conroy, D.¹ Mooney, A.¹ DuBuc, K.¹ Balstad, S.¹ Pace, D.¹ Yang, A.¹ Furgal, A.¹ Sen, A.¹ Eisenberg, D.² ¹Michigan Medicine, University of Michigan, Ann Arbor, MI, ²University of Michigan, Ann Arbor, MI.

Introduction: Telemedicine is increasingly an option for delivery of healthcare services, but its efficacy and acceptability for delivering CBT for insomnia has not been adequately tested. In a randomized controlled non-inferiority trial, we compared face-to-face and telemedicine delivery (via the AASM SleepTM platform) of CBT for insomnia for improving sleep and daytime functioning at post-treatment and 12-week follow-up. Methods: Sixty-five adults with chronic insomnia (46 women, mean age 47.2 \pm 16.3 years) were recruited primarily from insomnia clinics and screened for disgualifying sleep, medical, and mental health disorders. Eligible participants were randomized to 6 sessions of CBT for insomnia delivered face-to-face (n=32) or via AASM SleepTM (n=33). Participants completed self-report measures of insomnia (Insomnia Severity Index, ISI) and daytime functioning (fatigue, depression, anxiety, and overall functioning) at pre-treatment, post-treatment, and 12-week follow-up. The ISI was the primary non-inferiority outcome. Results: Telemedicine was non-inferior to face-to-face delivery of CBT for insomnia, based on a non-inferiority margin of 4 points on the ISI (β = -0.07, 95% CI -2.28 to 2.14). Compared to pretreatment, ISI scores improved significantly at post-treatment $(\beta = -9.02, 95\%$ CI -10.56 to -7.47) and at 12-week follow-up $(\beta = -9.34, 95\% \text{ CI} - 10.89 \text{ to} - 7.79)$. Similarly, daytime functioning measures improved from pre- to post-treatment, with sustained improvements at 12-week follow-up. Scores on the fatigue scale were lower in the telemedicine group at both post-treatment (F=4.64, df=1,119, p<.03) and follow-up (F=5.79, df=1,119, p<.02).

Conclusion: Insomnia and daytime functioning improve similarly whether CBT for insomnia is delivered via telemedicine or face-to-face. Telemedicine delivery of CBT for insomnia should be implemented more systematically to improve access to this evidence-based treatment.

Support: American Sleep Medicine Foundation Grant # 168-SR-17 (JT Arnedt, PhD)

0533

AGE AND EDUCATION LEVEL ARE ASSOCIATED WITH DROPOUT FROM COGNITIVE-BEHAVIORAL THERAPY FOR INSOMNIA IN PARTICIPANTS WITH CO-OCCURRING DEPRESSION: A REPORT FROM THE TRIAD STUDY

Dietch, J. R.¹ Manber, R.² Buysse, D. J.³ Edinger, J. D.^{4,5} Krystal, A.⁶

¹VA Palo Alto Health Care System, Palo Alto, CA, ²Stanford University, Palo Alto, CA, ³University of Pittsburgh, Pittsburgh, PA, ⁴National Jewish Health, Denver, CO, ⁵Duke University Medical Center, Durham, NC, ⁶University of California, San Francisco, San Francisco, CA.

Introduction: Early termination (i.e., dropout) from cognitivebehavioral therapy for insomnia (CBT-I) likely attenuates benefits and may reduce motivation for future treatment. The aim of the current study was to identify characteristics of participants who dropped out of CBT-I in an RCT of combined treatment for depression and insomnia.

Methods: Participants were 148 adults with comorbid insomnia and depression diagnoses (73% female; M age = 46.6[SD = 12.6]) who were randomly assigned to receive depression pharmacotherapy plus 7 weekly sessions of CBT-I (n = 73) or a credible control therapy for insomnia (n = 75). Receiver operating characteristic curve (ROC) analyses were performed to determine which participant characteristics (i.e., demographics, baseline depression and sleep variables) predicted dropout at session 4 (i.e., minimum dose) and session 7 (i.e., full course of CBT-I).

Results: Early termination (prior to session 4) rate was 28% and ROC analyses indicated participants aged 36 or less were more likely to drop out than those older than 36 (49% vs. 22%). The model did not identify additional predictors for either of the two age categories. Overall termination (prior to session 7) rate was 45% and ROC analyses indicated participants aged 46 or less were more likely to drop out than those older than 46 (61% vs. 34%). The model further found that among participants aged 46 or less, those with less than 14y education were at greater risk for dropout than those with greater than 14y education (79% vs. 46%). No other demographic, depression, or sleep variables were significant predictors of dropout.

Conclusion: Age was associated with elevated rate of dropout from CBT-I among individuals with co-occurring depression and insomnia. It appears that the combination of younger age and lower education level is particularly detrimental to treatment engagement. Better understanding of factors that contribute to dropout from CBT-I in this vulnerable group can guide development of retention strategies.

Support: MH078924, MH078961, MH079256

0534

COGNITIVE BEHAVIORAL THERAPY FOR PERINATAL INSOMNIA: EFFECTS ON POSTPARTUM DEPRESSIVE SYMPTOMS

Manber, R.¹ Bei, B.² Simpson, N.¹ Rangel, E.¹ ¹Stanford University, Stanford, CA, ²Monash University, Melbourne, AUSTRALIA.

Introduction: Poor sleep during pregnancy is a risk for postpartum depression. Using data from an RCT of CBT-I for insomnia disorder during pregnancy, we examined whether improvement in insomnia reduced postpartum depression symptom severity. We

hypothesized that better response to treatment during pregnancy would result in lower depressive symptom severity during the postpartum.

Methods: Pregnant women (N=179; gestation age 18-30 weeks) with insomnia disorder were randomized to CBT-I or an active control (CTRL) therapy (5 sessions during pregnancy, one at 6 weeks postpartum). Women with depressive disorders and those using prescription medications that impact sleep were excluded. The Insomnia Severity Index (ISI) and the Edinburgh Postpartum Depression Scale (EPDS) were administered at baseline, during pregnancy, and at 8, 18, and 30 weeks postpartum. The Perinatal Risk Questionnaire (PRQ) was administered at baseline. Included in the analyses were women who provided data for at least one of three postpartum assessments (62 in CBT-I; 55 in CTRL).

Results: Mixed effects models revealed that lower ISI following the pregnancy treatment phase (p < .001) and greater reduction in ISI during pregnancy (p = .053) predicted overall lower EPDS scores during postpartum; but these effects did not differ significantly between treatment arms. Average postpartum EPDS scores, which were low overall, were higher in women with ISI score at or above the median of 9 (6.6±3.9), compared to those below the median (3.5 ± 3.3). Compared to CTRL, participants in the CBT-I condition were nearly twice likely to have ISI scores below the median following the pregnancy treatment phase (29.1% versus 56.5%). Although higher PRQ scores were associated with overall higher postpartum EPDS (p=.0026), PRQ did not moderate postpartum EPDS trajectories.

Conclusion: We have previously shown that CBT-I is effective for antenatal insomnia, which is a risk for postpartum depression. Our current findings suggest that improving insomnia in pregnancy may reduce the risk for postpartum depression. Limitations include a small sample and missing data during the postpartum follow-up. A larger study among women specifically at risk for postpartum depression could help identify patient related factors that predict therapeutic benefits of CBT-I on postpartum depression. **Support:** NR013662

0535

EVALUATION OF INSOMNIA SYMPTOMS IN A DOUBLE-BLIND, RANDOMIZED, PLACEBO-CONTROLLED PHASE 3 TRIAL OF SAGE-217 IN POSTPARTUM DEPRESSION

Mittal, A.¹ Deligiannidis, K.² Huang, M.¹ Suthoff, E.¹ Acaster, S.³ Fridman, M.⁴ Li, S.¹ Gunduz-Bruce, H.¹ Lasser, R.¹ Campbell, A. D.¹ Bonthapally, V.¹ Hodgkins, P.¹ Kanes, S. J.¹ Werneburg, B.¹

¹Sage Therapeutics, Inc., Cambridge, MA, ²Department of Psychiatry, Zucker Hillside Hospital, Glen Oaks, NY, ³Acaster Lloyd Consulting Ltd., London, UNITED KINGDOM, ⁴AMF Consulting, Los Angeles, CA.

Introduction: Postpartum depression (PPD) is a specifier of major depressive disorder (MDD) with peripartum onset. SAGE-217, an investigational, oral neuroactive steroid $GABA_A$ receptor positive allosteric modulator, demonstrated improvements in depressive and anxiety symptoms versus placebo in a Phase 3 trial in PPD (NCT02978326; ROBIN) and a pivotal trial in MDD (NCT03000530). In PPD and MDD, insomnia symptoms are key diagnostic features, comorbid sleep disorders are frequent, and insomnia is a common residual symptom. Here we conducted posthoc analyses to assess insomnia symptoms in the ROBIN trial.

Methods: Women (n=151) ages 18-45, ≤ 6 months postpartum, with PPD (major depressive episode beginning in 3rd trimester or

≤4 weeks postpartum) and a Hamilton Rating Scale for Depression (HAM-D) total score ≥26, were randomized 1:1 to receive outpatient SAGE-217 30mg or placebo for two weeks, with 4 weeks follow-up. Change from baseline (CFB) in HAM-D score at Day 15 was the primary endpoint. Secondary endpoints included CFB in HAM-D at other time points and the Montgomery-Åsberg Depression Rating Scale (MADRS). Post-hoc analyses assessed HAM-D insomnia subscale (HAM-D-Ins) and MADRS individual insomnia item (MADRS-Ins) scores. HAM-D and MADRS measures were evaluated using a mixed-effects model for repeated measures. Safety and tolerability were assessed by adverse event (AE) reporting.

Results: SAGE-217 demonstrated statistically significant Day 15 CFB versus placebo in HAM-D (primary endpoint: -17.8 vs. -13.6, p=0.0028), MADRS (-22.1 vs. -17.6, p=0.0180), and associated insomnia sub-scales/items (difference SAGE-217 vs. placebo; HAM-D-Ins: -1.003, p=0.0038; MADRS-Ins: -0.773, p=0.0116). Significant CFB in insomnia sub-scales/items favoring SAGE-217 were observed by Day 3 (HAM-D-Ins: -0.841, p=0.0142; MADRS-Ins: -0.710, p=0.017) and at Day 45 (HAM-D-Ins: -0.730, p=0.0207; MADRS-Ins: -0.636, p=0.0221). Most common (\geq 5%) AEs were somnolence, headache, dizziness, upper respiratory tract infection, diarrhea, and sedation.

Conclusion: SAGE-217 demonstrated improvements in depression symptoms, including insomnia symptoms, and was generally well tolerated.

Support: This study was sponsored by Sage Therapeutics, Inc.

0536

MOTIVATION AT PRETREATMENT AND ITS CORRELATES IN A TRIAL OF DIGITAL CBT FOR INSOMNIA: PRELIMINARY FINDINGS

JI, X.¹ Cheung, J. M.² Ivers, H.³ Morin, C. M.⁴ ¹Laval University, Québec city, QC, CANADA, ²The University of Sydney, Sydney, AUSTRALIA, ³Laval University, Québec City, QC, CANADA, ⁴University Laval, Québec city, QC, CANADA.

Introduction: Pretreatment motivation is a critical variable in any intervention seeking to modify behaviors. Lack of motivation may hamper the effects of cognitive-behavioral therapy for insomnia (CBT-I), especially when delivered online. This study aims to investigate baseline correlates of pretreatment motivation and its influence on treatment outcomes in the context of digitalized CBT-I. Methods: This is a secondary analysis of an ongoing pragmatic trial conducted in primary care clinics of Québec City, Canada. The trial was designed to assess the efficacy of a stepped-care intervention for chronic insomnia in which participants received a digital CBT-I (SHUTi), alone or in addition to sleep medication they were already using. Pre-treatment motivation was measured using two items based on the perceived importance of improving sleep and readiness to change behaviors to improve sleep (Score range: 0 to 20; cronbach's alpha 0.79). Baseline questionnaires included an extended version of Insomnia Severity Index (ISI), Fatigue Severity Scale (FFS), Generalized Anxiety Disorder (GAD-7) and Patient Health Questionnaire (PHQ-9). Treatment outcome was measured by a change in ISI scores (i.e. ISI post - ISI pre).

Results: A total of 28 participants were included in the analysis. All participants preferentially selected ISI either as monotherapy (n=13) or in combination with their usual sleep medication (n=15). Participants' motivation before treatment was high (Mean: 18.04; SD: 1.93). We did not find any associations between motivation

and ISI score change or incidence of dropout. However, baseline fatigue was positively correlated with pretreatment motivation (r = 0.51, p = 0.005) and more severe insomnia symptoms were also associated with higher motivation (r=0.43, p=0.03). Specifically, perceived importance was associated with both nighttime and day-time insomnia symptoms while readiness for behavioral change was only associated with daytime impairments on energy, mood and social activities (all p = 0.01). Baseline anxiety and depressive symptoms were not correlated with motivation.

Conclusion: Insomnia-related daytime impairments and elevated fatigue levels appear to be linked to pretreatment motivation, especially for behavioral changes. Further study with greater statistic power is warranted to understand the relationship between participants' motivation and treatment adherence or outcomes. **Support:** CIHR0083000212

0537

COGNITIVE FUNCTIONING BEFORE AND AFTER INSOMNIA TREATMENT IN WOMEN VETERANS

Dzierzewski, J. M.¹ Zhu, R.² Donovan, E. K.¹ Perez, E.¹ Song, Y.³ Kelly, M. R.² Carlson, G.² Fung, C. H.^{2,3} Alessi, C.^{2,3} Martin, J. L.^{2,3}

¹Department of Psychology, Virginia Commonwealth University, VA, ²VA Greater Los Angeles, Los Angeles, CA, ³University of California, Los Angeles, Los Angeles, CA.

Introduction: Women are at higher risk for cognitive impairment and dementia compared to men. Identifying potentially treatable risk factors such as insomnia is an important clinical goal. In a trial comparing two behavioral treatments for insomnia in women veterans, we hypothesized that 1) worse baseline insomnia severity would be associated with poorer cognitive function, and 2) improvement in insomnia severity with treatment would be associated with improvement in cognitive functioning.

Methods: 347 women veterans with insomnia disorder [mean age 48.3 (12.9) years] completed baseline testing. Of these, 149 women were randomized to receive cognitive behavioral therapy for insomnia (CBT-I) or acceptance and commitment (ACT) based insomnia treatment (both treatments included sleep restriction, stimulus control, and sleep hygiene). Insomnia Severity Index (ISI) was assessed at baseline, post-treatment, and 3-month follow-up. Cognitive functioning was measured with Symbol Digit Coding (SDC) and Trail Making Test A and B (TMTA and TMTB). Pearson correlations were used to examine associations between insomnia severity and cognitive functioning at baseline and changes in both insomnia severity and cognitive functioning from before to after treatment.

Results: At baseline (N=347), mean ISI was 14.1 (5.3). Worse baseline ISI was associated with worse baseline cognitive functioning on TMTA (r=-.15, p<.01) and SDC (r=-.12, p<.05). In the randomized sample (N=149), ISI scores improved at post-treatment (mean ISI change= -9.0; p<.001) and 3-month follow-up (mean change= -8.0; p<.001) relative to baseline. Improvement in ISI from baseline to post-treatment was significantly associated with improvement in SDC from baseline to post-treatment (r=-.18, p<.05), but not improvement in TMTA and TMTB. Change in ISI was not significantly related to change in cognitive tasks from baseline to 3-month follow-up.

Conclusion: More severe insomnia is associated with worse cognitive functioning in women veterans. The magnitude of improvement in insomnia symptoms may be associated with improvement in cognition.

Support: NIH/NIA K23AG049955 (PI: Dzierzewski); VA/HSR&D IIR-HX002300 (PI: Martin), NIH/NHLBI K24HL143055 (PI: Martin).

0538

WHAT HAPPENS AFTER PRESCRIPTION OF INSOMNIA MEDICATION AMONG OLDER ADULTS?

Jobe, S. L.¹ Albrecht, J. S.² Scharf, S. M.¹ Johnson, A. M.³ Wickwire, E. M.^{1,4}

¹Sleep Disorders Center, Division of Pulmonary and Critical Care Medicine, Department of Medicine, University of Maryland School of Medicine, Baltimore, MD, ²Department of Epidemiology and Public Health, University of Maryland School of Medicine, Baltimore, MD, ³Department of Pharmaceutical Health Services Research, University of Maryland School of Pharmacy, Baltimore, MD, ⁴Department of Psychiatry, University of Maryland School of Medicine, Baltimore, MD.

Introduction: Despite consensus recommendations regarding need for caution and careful management, sedative hypnotic insomnia therapies remain commonly prescribed among older adults. Further, sleep medications are often prescribed in the absence of a thorough sleep history or evaluation. However, little is known about delivery of sleep-related care following prescription of insomnia medications. Thus, the purpose of this study was to characterize the course of sleep-related care following a prescription fill for insomnia medication among older adults.

Methods: Our data source was a random 5% sample of Medicare administrative claims data from 2006-2013. Insomnia medications were identified by searching the Part D prescription drug claims and included FDA-approved insomnia-related medication classes and drugs. Sleep disorders were operationalized using International Classification of Diseases, Ninth Revision, Clinical Modification codes. Descriptive analyses were performed to estimate the number of insomnia medication users who received sleep disorder diagnoses.

Results: A total of 33,252 Medicare beneficiaries without prior history of sleep disorders received at least one FDA-approved insomnia medication fill between 2006-2013. Of these, 43.2% (n=14,354) eventually received a sleep disorder diagnosis. Among those receiving a sleep diagnosis after being prescribed insomnia medication, insomnia was the most common disorder (71.0%; n=10,198). Further, 15.0% (n=2,149) of individuals who were prescribed insomnia medication received an obstructive sleep apnea diagnosis, 6.6% (n=947) were diagnosed with sleep disturbances, and 5.9% (n=845) were diagnosed with restless legs syndrome. Of those who received a sleep disorder diagnosis, 95.1% (n=13,649) were diagnosed with one sleep disorder, 4.5% (n=639) were diagnosed with two sleep disorders, and 0.4% (n=66) were diagnosed with three or more sleep disorders.

Conclusion: Fewer than half of Medicare beneficiaries prescribed FDA-approved sedative hypnotic insomnia medications ever received a formal sleep-related diagnosis. These results suggest that clinicians prescribe sedative hypnotics without thoroughly evaluating sleep complaints.

Support: This research was supported by an AASM Strategic Research Award from the AASM Foundation to the University of Maryland, Baltimore (PI: EMW).

0539

ARE SHORT SLEEPERS UNCOUPLED SLEEPERS?

Hartescu, I.¹ Morgan, K.¹ Stensel, D. J.¹ Thackray, A. E.¹ King, J. A.¹

¹Loughborough University, Loughborough, UNITED KINGDOM, ²Loughborough University, Loughborough, UNITED KINGDOM.

Introduction: While short sleep durations (<7h/night) are associated with increased diabetes risk, there is limited evidence that increasing the habitual sleep duration of short sleepers is either feasible, or will reliably improve metabolic health outcomes. Furthermore, in the absence of insomnia disorder, it remains unclear whether habitual short sleep mainly reflects a genetic predisposition or a lifestyle choice. In a randomized controlled study we delivered a sleep extension protocol based on CBTi principles to overweight 'short sleepers' at increased risk of Type II diabetes.

Methods: 18 male short sleepers (M_{age} =41.4; M_{BMI} =29.57; baseline mean TST=5.8 h/ night) with no complaints of insomnia were randomized to the sleep extension intervention or control condition (printed sleep hygiene advice). The 6-week intervention commenced with personalized sleep re-scheduling negotiated in a 60-minute 1-to-1 session, and supported by elements of sleep hygiene, stimulus control, relaxation and cognitive strategies. Outcomes included sleep duration (actigraphy), fasting insulin, Mean Amplitude of Glycemic Excursions (MAGE) from continuous glucose monitoring, and blood pressure. Data were analyzed in linear fixed effects models including time, group and baselines values.

Results: Adherence to the 6-week protocol was high. Relative to controls (n=8), intervention participants (n = 10) showed a significant increase in TST (95%CI 46.91min, 101.64min, p<0.001; $M_{\text{Diff}} = 79.4$ min, p<0.001) and significant reductions in fasting insulin (95%CI -32.08 pmol/L, -.97.0 pmol/L; p=0.04; $M_{\text{Diff}} = -10.2$ pmol/L, p=0.06); MAGE (95%CI -0.77, -0.08, p=0.02; M_{Diff} -0.35, p=0.05) and diastolic (95%CI -22, -5, p=0.004; M_{Diff} =-12, p=0.004); and systolic blood pressure (95%CI -20, -2, p=0.03; M_{Diff} =-10, p=0.006).

Conclusion: CBTi-based sleep extension protocols offer feasible and effective lifestyle interventions in the management of metabolic health in overweight short sleepers who fit published categorization of non-complaining poor sleepers with an undeveloped insomnia identity whose subjective sleep experience and objective sleep characteristics are 'uncoupled'.

Support: School of Sport, Exercise and Health Sciences, Loughborough University

0540

INSOMNIA SYMPTOM TRAJECTORIES DURING AND FOLLOWING COMBINED TREATMENT FOR INSOMNIA AND DEPRESSION: A REPORT FROM THE TRIAD STUDY

Atwood, M. E.¹ Dietch, J. R.² Buysse, D. J.³ Edinger, J. D.⁴ Krystal, A.⁵ Manber, R.¹

¹Stanford University, Palo Alto, CA, ²VA Palo Alto Health Care System, Palo Alto, CA, ³University of Pittsburgh, Pittsburgh, PA, ⁴National Jewish Health, Denver, CO, ⁵University of California, San Francisco, San Francisco, CA, ⁶Stanford University, Palo Alto, CA.

Introduction: Cognitive behavioral therapy for insomnia (CBT-I) reduces insomnia severity among individuals with insomnia and major depressive disorder (MDD). Understanding the long-term trajectories of insomnia symptom severity has the potential to inform optimization of CBT-I in this population. The objectives of this study were to examine trajectories of change in insomnia

severity over a 16-week treatment phase and 2-year naturalistic follow-up, and explore correlates of symptom trajectories.

Methods: 148 adults (age 46.6 ± 12.6 , 73.0% female) with insomnia and MDD were randomly assigned to receive depression pharmacotherapy plus seven sessions of either CBT-I or control insomnia therapy. Depression and insomnia severity were assessed via the Hamilton Depression Rating Scale and Insomnia Severity Index at baseline, bi-weekly during treatment, and every 4 months over follow-up. Sleep effort and beliefs about sleep were assessed at baseline, midtreatment, and posttreatment.

Results: Latent class linear mixed modeling revealed four insomnia response trajectories: 1) Early Sustained-Responders (16%) showed marked improvement early in treatment, sustained over follow-up; 2) Gradual-Responders (36.7%) achieved substantial symptom reduction by posttreatment, sustained over follow-up; 3) Initial-Responders (25.3%) had substantial symptom reduction during treatment but increased in severity over follow-up; and 4) Partial-Responders (20.7%) achieved minimal improvement over treatment, and maintained moderate symptom severity over follow-up. Chi-square analyses revealed that classes did not differ significantly on sex, ethnicity, employment, relationship status, or treatment received (all ps > .05). One-way ANOVAs with Tukey's HSD, showed that Partial-Responders consistently endorsed higher depressive symptom severity, sleep effort, and unhelpful beliefs about sleep at baseline, throughout treatment, and follow-up (ps < .05). Early Sustained-Responders endorsed lower sleep effort by midtreatment (ps < .01).

Conclusion: Results suggest four temporal patterns of treatment response and identified clinical correlates. Future work will be needed to determine if addressing sleep effort early in the course of treatment might enhance sustained insomnia outcome.

Support: MH078924, MH078961, MH079256

0541

EXPLORATORY STUDY OF CLOSED-LOOP, ARTIFICIAL INTELLIGENCE DRIVEN NEUROTECHNOLOGY IMPROVES SELF-REPORTED SYMPTOMS OF INSOMNIA, STRESS, AND ANXIETY IN FIRST RESPONDERS

*Tegeler, C. L.*¹ *Howard, L. J.*¹ *Brown, K. L.*¹ *Kellar, D. C.*² *Shaltout, H. A.*³ *Gerdes, L.*⁴ *Tegeler, C. H.*⁵

¹Department of Neurology, Wake Forest School of Medicine, Winston-Salem, NC, ²Department of Neurology, Wake Forest School of Medicine, Winston Salem, NC, ³Hypertension and Vascular Research Center, Wake Forest School of Medicine, Winston-Salem, NC, ⁴Brain State Technologies, Scottsdale, AZ, ⁵Wake Forest School of Medicine, Winston-Salem, NC.

Introduction: First responders (FR) have decreased life expectancy, attributed to work-related exposure to traumatic stress and circadian disruption. In prior studies, High-resolution, relational, resonance-based, electroencephalic mirroring (HIRREM®) reduced symptoms and improved heart rate variability (HRV) in law enforcement personnel. HIRREM is operator dependent, difficult to scale, and many medications were excluded for prior studies. Cereset ResearchTM (CR) uses HIRREM core technology, echoing tones linked to brainwaves, with updated components, artificial intelligence (AI) driven protocols, and software management of designs to improve scalability. We report symptom changes in a series of first responders enrolled in an exploratory study evaluating CR for self-reported symptoms of insomnia, stress, or anxiety, including subjects taking previously excluded medications.

Methods: 11 adults (4 females) meeting criteria for insomnia (ISI, of \geq 8 points for \geq 1 month), stress (PSS of \geq 14), or anxiety (GAD-7 of \geq 5), who are also FR, enrolled in this ongoing exploratory trial. Subjects receive six to twelve 60 minute sessions of CR, plus continued current care. Data collection is at baseline (V1), 0-21 days post-intervention (V2), 4-7 weeks later (V3), and 4-7 weeks thereafter (V4). Primary outcome is change in autonomic cardiovascular regulation at V3, with change in ISI, PSS, GAD-7, CES-D, and PCL-C as secondary outcomes. We report interim results for symptom outcomes at V3. HRV and baroreflex sensitivity results are pending.

Results: 2 dropped out during follow-up. For n=9, median change from V1 to V3 ISI score: -6; PSS score: -4; GAD-7: -4; CES-D: -3; and PCL-C: -8. The cohort moved to no anxiety and low stress categories from V1 to V3.

Conclusion: Results suggest relevant symptom reductions among FR following use of CR, as seen previously with HIRREM for law enforcement, even with additional medications. This informs future randomized clinical trials using this scalable, non-drug intervention. Updated results will be presented.

Support: Support: Research grant received from, The Susanne Marcus Collins Foundation, Inc.

0542

EXPLORATORY USE OF ARTIFICIAL INTELLIGENCE DRIVEN ACOUSTIC NEUROMODULATION IMPROVED SLEEP, DEPRESSION, ANXIETY, AND STRESS IN ADULTS WITH PERSISTING POST-CONCUSSION SYMPTOMS

*Tegeler, C. L.*¹ *Howard, L. J.*¹ *Brown, K. L.*¹ *Kellar, D. C.*² *Shaltout, H. A.*³ *Gerdes, L.*⁴ *Tegeler, C. H.*⁵

¹Department of Neurology, Wake Forest School of Medicine, Winston-Salem, NC, ²Department of Neurology, Wake Forest School of Medicine, Winston Salem, NC, ³Hypertension and Vascular Research Center, Wake Forest School of Medicine, Winston-Salem, NC, ⁴Brain State Technologies, Scottsdale, AZ, ⁵Wake Forest School of Medicine, Winston-Salem, NC.

Introduction: In prior studies, High-resolution, relational, resonance-based, electroencephalic mirroring (HIRREM®) reduced persisting post-concussion symptoms (PPCS) of insomnia and depression and improved heart rate variability (HRV), but is operator dependent, with difficulty scaling. Cereset Research[™] (CR), a noninvasive, closed-loop, artificial intelligence (AI) driven, acoustic neuromodulation technology uses the same core technology, echoing tones linked to brainwaves, but includes updated components, standardized AI driven protocols, software management of designs, and shorter sessions to improve scalability. This open label trial explores use of CR for PPCS.

Methods: 5 adults (1 female, median age = 48, 31-64) with PPCS received a median of 8 CR sessions (range 7-9, 60 minutes each) over 11 (5-18) days as part of an open label IRB-approved exploratory study of CR for diverse health conditions. Data is collected at baseline (V1), 0-21 days (V2), 4-7 weeks later (V3), and 4-7 weeks thereafter (V4). Pre- and post-CR symptom inventories included concussion (RPQ), insomnia (ISI), depression (CES-D), anxiety (GAD-7), PTSD (PCL-C), and stress (PSS). Primary outcome is change in autonomics at V3 via HRV (SDNN and rMSSD) based on 10-minute BP and HR recordings using a BIOPAC device. Formal analysis of HRV outcome is pending, but we report preliminary changes in symptom outcomes. **Results:** 6 subjects have enrolled for sleep trouble related to PCCS, with 1 lost to follow-up after receiving intervention. For V1-V2 (n=5), median change in RPQ score is -23, ISI -10, CES-D -16, GAD-7 -7, PCL-C -16, and PSS -7. For V1-V3 (n=4), median change in RPQ -19, ISI -10.5, CES-D -6, GAD-7 -4.5, PCL-C -14.5, and PSS -3. No serious adverse events reported.

Conclusion: Preliminary results suggest similar, clinically meaningful reductions in ISI score, and concussion symptoms, as seen with HIRREM, suggesting promise as a scalable, non-drug intervention for insomnia with PPCS. Updated results will be presented. **Support: Support:** Research grant received from, The Susanne Marcus Collins Foundation, Inc.

0543

IMPLEMENTING INSOMNIA CARE PATHS FOR OLDER ADULTS AND PEOPLE WITH DEMENTIA

Benca, R.¹ Ferziger, R.² Wickwire, E. M.³ Bertisch, S.⁴ Biddle, J.⁵ Boustani, M.⁶ Culpepper, L.⁷ Gooneratne, N.⁵ Lett, J.⁸ Manderscheid, R.⁹ Mehra, R.¹⁰ Reynolds, C.¹¹ Grandner, M. A.¹² ¹University of California, Irvine, Irvine, CA, ²Merck Research Laboratories, Upper Gwynedd, PA, ³University of Maryland, Baltimore, MD, ⁴Harvard Medical School, Boston, MA, ⁵University of Pennsylvania, Philadelphia, PA, ⁶Indiana University, Indianapolis, IN, ⁷Boston University, Boston, MA, ⁸Avar Consulting, Rockville, MD, ⁹National Association of County Behavioral Health and Developmental Disability Directors, Washington, DC, ¹⁰Cleveland Clinic, Cleveland, OH, ¹¹University of Pittsburgh, Pittsburgh, PA, ¹²University of Arizona, Tucson, AZ.

Introduction: Despite the high prevalence of insomnia in older adults and those with dementia, screening and treatment remain inconsistent and suboptimal. Implementing a care path in a health system, though, is difficult. To determine what issues are relevant for implementation, a consensus meeting was convened, which included discussion, voting on components, and further consensus-building.

Methods: All N=20 participants, representing a wide range of stakeholders including research, industry, sleep, primary care, implementation science, and others, voted whether they agreed or disagreed with 36 different statements regarding what issues are important for implementing geriatric insomnia care paths. These represented a range of items addressing strategies for identifying and incentivizing stakeholders, identifying patients in most need and who would receive benefit, addressing comorbidities and multiple specialties, understanding how specific organizations make decisions about and changes to care, size and scope of the care path, determining the process for implementation, how it will improve outcomes, addressing specific needs of primary care, and addressing costs, reimbursements, and liabilities. Items were scored as 0=strongly agree, 1=agree, 2=disagree, and 3=strongly disagree. Mean scores were evaluated and responses were dichotomized to agree/disagree).

Results: Despite the diversity among attendees, median rate of agreement for was 95% (IQR=90-95%). All items were endorsed by >=80% of respondents. Mean score was 0.48 (SD=1.85). 95%CIs were computed for each proportion and compared to the mean. The only item that significantly differed from the mean score indicated that understanding benefits of a care path to the general community is less important of an issue than others (M=0.85).

Conclusion: Implementing an insomnia care path for older adults in an institution will likely require addressing a wide range of

issues, including questions about stakeholders, the health system/ context, patients, and practical considerations. **Support:** Merck Research Labs provided support

0544

RECONSIDERING STIMULUS CONTROL: ACTIVITIES IN BED ASSOCIATED WITH SLEEP-RELATED OUTCOMES

*Phan, S.*¹ *Perlis, M. L.*² *Hale, L.*³ *Branas, C.*⁴ *Killgore, W. D.*¹ *Wills, C. C.*¹ *Grandner, M. A.*¹

¹University of Arizona, Tucson, AZ, ²University of Pennsylvania, Philadelphia, PA, ³Stony Brook University, Stony Brook, NY, ⁴Columbia University, New York, NY.

Introduction: The typical advice is that in order to avoid insomnia, people should avoid activities in bed other than sleep. Yet, activities such as reading and watching TV in bed are common.

Methods: Data were obtained from the Sleep and Health Activity, Diet, Environment, and Socialization (SHADES) Study, N=1,007 adults age 22-60. Sleep hygiene was assessed using items from the Sleep Practices and Attitudes Questionnaire (SPAQ), which asked whether respondents agree/disagree that they do the following in bed: Read, Watch TV, Eat, Work, Worry, and/or Argue. These were analyzed in relation to Insomnia Severity Index (ISI) score, Pittsburgh Sleep Quality Index (PSQI) score, Epworth Sleepiness Scale (ESS) score, Fatigue Severity Scale (FSS) score, and selfreported sleep duration (TST), sleep latency (SL), and wake after sleep onset (WASO). Covariates included age, sex, education, and income.

Results: Those that frequently engaged in activities were: reading (75%), watching TV (63%), eating (42%), working (32%), worrying (82%), and arguing (23%). Reading was associated with less WASO (B=-14min, p=0.02). Watching TV was associated with higher ISI (B=1.22, p=0.04), PSQI (B=1.04, p=0.007), and ESS (B=0.87, p=0.049), and less TST (B=-0.29, p=0.04). Eating was associated with higher ISI (B=1.75, p=0.01), PSQI (B=1.23, p=0.008), and FSS (B=4.36, p=0.002). Working was associated with higher ISI (B=1.82, p=0.019), PSQI (B=1.65, p=0.001), and ESS (B=1.78, p=0.002). Worrying was associated with higher ISI (B=7.34, p<0.0005), PSQI (B=4.40, p<0.0005), ESS (B=2.53, p=0.001), FSS (B=9.51, p<0.0005), and SL (B=19.39, p<0.0005), and less TST (B=-0.55, p=0.023). Arguing was associated with higher ISI (B=3.78, p<0.0005), PSQI (B=3.15, p<0.0005), ESS (1.47, p=0.023), and SL (B=10.97, p=0.013), and lower TST (B=-0.71, p=0.001).

Conclusion: Individuals who perform mentally distressing activities such as worrying and arguing experience especially worse sleep, and those who read in bed have fewer awakenings.

Support: The SHADES study was funded by R21ES022931. Dr. Grandner is supported by R01MD011600.

0545

BI-LAYER MELATONIN TABLET PROVIDES IMMEDIATE AND EXTENDED ABSORPTION TO ADDRESS PRIMARY SLEEP ISSUES

Mun, J. G.¹ Wang, D.¹ Doerflein Fulk, D. L.¹ Fakhary, M.² Gualco, S. J.³ Grant, R. W.¹ Mitmesser, S. H.¹ ¹Science & Technology, Pharmavite, LLC, Los Angeles, CA, ²Analytical Research Services, Pharmavite LLC, Los Angeles, CA, ³Quality Standards, Pharmavite LLC, Los Angeles, CA.

Introduction: Melatonin is a naturally-occurring hormone that functions in the regulation of the sleep-wake cycle. Exogenous

melatonin has demonstrated utility for the induction and maintenance of sleep. Modulation of tablet dissolution rates can be achieved by strategically using binding agents in two layers to rapidly raise melatonin levels and sustain elevated levels over time to better emulate normal nocturnal melatonin secretion.

Methods: A randomized, double-blind, controlled cross-over study was conducted in healthy adults (n=18) to investigate the pharmacokinetics of a 4.5 mg melatonin bi-layer tablet with either prolonged-release (PR) or immediate-release (IR) characteristics. Blood samples were collected at 0.25, 0.5, 0.75, 1, 2, 3, 4, 5, 6, 8 and 10 h post-ingestion and melatonin concentrations were quantified. Results: The PR formulation had a lower maximum concentration (C_{max}, p<0.002), but a longer time to reach maximum concentration (T_{max} , p<0.001). Absorption rate constant (K_a) and terminal disposition rate constant (λ) were both lower in PR melatonin than the IR formulation (p<0.001), indicating slower absorption and elimination rates. These measures were consistent with significantly greater absorption ($t_{a1/2}$, p<0.002) and elimination half-lives $(t_{1/2}, p < 0.001)$ in the IR formulation compared to the PR formulation. Additionally, plasma melatonin concentrations reached preingestion values by 8 h post-administration of the PR formulation, indicating that individuals sleeping for a recommended 7-9 hours will wake with normal melatonin levels, when taken an hour before bedtime.

Conclusion: Prolonged-release bi-layer melatonin tablet safely and effectively extends the absorption of exogenous melatonin compared to an immediate-release formulation.

Support: Pharmavite, LLC

0546

SLEEP SCIENCE AT HOME - DELIVERING SLEEP ASSESSMENT AND DIGITAL CBT-I AT SCALE

*Arnal, P. J.*¹ *Thorey, V.*² *Debellemaniere, E.*² *Mordret, E.*² *Llamosi, A.*² *Chouraki, A.*²

¹Dreem Science Team, New York, NY, ²Dreem Algorithms Team, Paris, FRANCE.

Introduction: Cognitive-behavioral therapy for insomnia (CBT-I) is the current first-line treatment for insomnia disorder, recommended by the AASM and SRS. Digital versions of CBT-I have been developed and validated to address the need for implementation at scale but still suffer from poor accessibility and compliance. Therefore, the aim of this open-label, Real-World Study (RWS) was to assess the engagement and efficacy of a next-generation CBT-i 6-weeks program.

Methods: 1304 subjects were included in the analysis between Dec 23rd, 2018 and December, 14th 2019. The main inclusion criteria were having an Insomnia Severity Index ISI \geq 15 and completion of one week of Dreem program. The variables have been measured by the Dreem headband (DH) for objective variables, and on subjects' answers to questionnaires for subjective ones.

Results: The retention during this RWS was 70.4 % (Pre: n = 1304 and Week 4: n = 935). The program led to a clinically significant decrease of 7.42 points on the ISI (p < 0.001). The obj-WASO was reduced by 35% (n = 359, p < 0.001), obj-Awakenings were reduced by 37% (n = 359 p < 0.001), obj-SE was increased by 2.56 points (n = 305, p < 0.001) and obj-SOL was reduced by 22% (n = 359, p < 0.001). The subj-SOL was reduced by 41% (n = 176, p < 0.001), subj-SE was increased by 8.9 points (n = 168, p < 0.001), subj-SD was increased by 16% (baseline: 307.50 ± 88.86 min; post 357.07 ± 91.24 min, subj-SD (n = 174, p < 0.001).

Conclusion: The results of this RWS suggest this insomnia program has a high engagement compared to other digital CBT-I programs and is as effective as traditional in-person CBT-I. This new generation of Insomnia therapy combining hardware, software and therapist serves as an efficient and engaging treatment implementable at scale.

Support: This study has been supported by Dreem sas.

0547

INSOMNIA IN VETERANS WITH COPD: PREVALENCE, CORRELATES, AND HEALTH CARE UTILIZATION

Luyster, F. S.¹ Boudreaux-Kelly, M. Y.² Bon, J. M.^{1,2} ¹University of Pittsburgh, Pittsburgh, PA, ²VA Pittsburgh Healthcare System, Pittsburgh, PA, ³University of Pittsburgh, Pittsburgh, PA.

Introduction: Veterans with COPD are a vulnerable group for developing insomnia. The objectives of this study were to examine the prevalence and correlates of insomnia and its impact on health care utilization in a national sample of Veterans with COPD.

Methods: National data from electronic medical records of Veterans who utilized the Veterans Health Administration between FY2011 and FY2017 was accessed from the Veterans Affair Corporate Data Warehouse. COPD was based on International Classification of Diseases diagnostic codes (ICD-9/10). Insomnia was defined by ICD-9/10 codes or sedative-hypnotic prescription of >30 doses in a given fiscal year. ICD-9/10 codes for other conditions were documented. Outpatient and inpatient health care service utilization included number of physician encounters and emergency room visits and hospitalizations with a primary diagnostic code for COPD or COPD exacerbation. Bivariate comparisons between veterans with COPD and insomnia and COPD only were made for sociodemographic, comorbidities, and health care utilization variables using t-tests and Chi-square tests, as appropriate.

Results: A total of 1,542,642 Veterans with COPD were identified during the 6-year period. Of those with COPD, 575,539 (37.3%) were identified as having insomnia. Veterans with COPD and comorbid insomnia were younger (64 years vs. 69 years) and more likely to be female (6.3% vs. 3.7%), Black (14.0% vs. 11.0%), be a current smoker (46.1% vs. 35.5%), and have comorbidities including obstructive sleep apnea, diabetes, asthma, stroke, depression, or posttraumatic stress disorder compared to those without insomnia (all p's <0.001). Compared to Veterans with COPD only, those with comorbid insomnia had a greater number of COPDrelated outpatient (6.9 vs. 10.5) and emergency room (1.1 vs. 1.2) visits and hospitalizations (1.9 vs. 2.3) (p's <0.001).

Conclusion: Insomnia is highly prevalent in Veterans with COPD and is associated with greater COPD-related health care utilization. Future research is needed to determine if targeted treatment for insomnia can improve COPD outcomes.

Support: This study was funded by the VA Competitive Career Development Fund.

0548

OVERNIGHT MEMORY CONSOLIDATION IN INSOMNIA VERSUS NORMAL AND EXPERIMENTALLY DISRUPTED SLEEP

Omlin, X. Reid, M. J. Sharman, R. Schneider, J. Espie, C. A. Kyle, S. D.

Sleep and Circadian Neuroscience Institute, Nuffield Department of Clinical Neurosciences, University of Oxford, Oxford, UNITED KINGDOM.

Introduction: Healthy sleep is assumed to play an important role in the consolidation of newly acquired memories. Evidence suggest that periods of sleep after learning facilitates memory consolidation relative to wakefulness. Insomnia is associated with cognitive impairment but few studies have assessed overnight memory consolidation. We compared overnight consolidation in people with insomnia to a group of good sleepers who were randomised to either a normal night of sleep (uninterrupted sleep, US) or one night of sleep continuity disruption via forced awakenings (FA).

Methods: 51 good sleepers (37 female: mean age: 24 years, SD: 3.63), randomised to either one night of US (n=24) or one night of FA (n=27), were compared to 27 participants meeting criteria for insomnia disorder (23 female; mean age: 53 years, SD: 8.34) who were assessed at baseline as part of a randomised controlled trial of digital cognitive-behavioural therapy. Overnight memory consolidation (difference in correctly recalled word pairs between evening and morning recall) was assessed using the same word-pair task and protocol in the two lab-based studies.

Results: Overnight memory consolidation was significantly lower in the insomnia (mean: 5.4, SD: 5.8, p=0.001) and the FA (mean: 6.7, SD: 4.1, p<0.001) group compared to the US group (mean: 11.6, SD: 5.3). However, when adjusted for age only the FA group, but not the insomnia group, showed significantly lower memory performance than the US group.

Conclusion: While our findings suggest that overnight memory consolidation is impaired in insomnia relative to normal sleep we cannot rule out an age-related explanation given the difference in ages between the study samples. Future work will assess whether overnight consolidation improves following cognitive behavioural therapy.

Support: The research was supported by the National Institute for Health Research (NIHR) Oxford Biomedical Research Centre (BRC) and the Dr Mortimer and Theresa Sackler Foundation. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health.

0549

MISPERCEPTION OF NEUTRAL FACES IN INSOMNIA

Akram, U.

Sheffield Hallam University, Sheffield, UNITED KINGDOM.

Introduction: Emotional faces have been widely used amongst populations with mental health conditions to examine alterations in attention and perception relative to controls. Insomnia is associated with reduced emotion intensity ratings for facial expressions of fear, sadness and happiness. However, research is yet to examine whether neutral faces are accurately perceived amongst individuals with insomnia. This study compared normal-sleepers and individuals experiencing insomnia symptoms in their expression intensity rating of neutral faces.

Methods: Fifty-six normal-sleepers (NS: 19.69±4.07yrs, 73% female) scoring <5 on the Insomnia Severity Index (ISI; 2.70 \pm 1.69) and 58 individuals experiencing clinically significant insomnia symptoms (INS:20.32±4.08yrs, 85% female) scoring ≥15 on the ISI (19.24±3.53), observed 12 neutral facial photographs. Between 0-100, participants were required to rate the extent to which each face appeared as: attractive; sad; happy; trustworthy; approachable; healthy; and sociable. 0 indicated not at all, 100 indicated very much so. The facial stimuli were taken from Karolinska Directed Emotional Faces database, and were presented in random order.

Results: The results revelated a main effect of group (F(1,117)=4.04, p=.047) and expression (F(7, 819)=39.08, p=.001)

on intensity ratings. Whilst no significant group x expression interaction was confirmed (F(7,819)=1.03,p=.41), simple effects analysis determined that those experiencing insomnia symptoms rated neutral faces as significantly more attractive $(34.30 \pm 14.82; t(117) = -$ 2.73, p=.007; Cohens' d=.50) and happy (34.83±13.87; t(117)=-2.23, p=.028; Cohens' d=.41) when compared to normal-sleepers (Attractive: 26.89±14.76; Happy: 28.90±12.48). No significant differences were observed for all other ratings.

Conclusion: The present outcomes tentatively suggest that individuals experiencing clinically significant insomnia symptoms differentially perceive neutral faces when comparted with normalsleepers. Specifically, neutral faces of other people were rated in a more positively valanced manner (i.e. more attractive and happier). Considering an individual's capacity to correctly gauge facial expressions remains fundamental for effective social interaction, and in influencing social judgments, these outcomes present negative psychosocial implications for those with insomnia. Support: n/a

0550

INSOMNIA IS ASSOCIATED WITH GREATER ARTERIAL STIFFNESS AND CARDIAC DYSFUNCTION

Petrov, M. E.¹ Youngstedt, S. D.¹ Mookadam, F.³ Jiao, N.¹ Lim, L. M.¹ Wong, B.¹ Angadi, S. S.¹ ¹Arizona State University, Phoenix, AZ, ²Arizona State University, Phoenix, AZ, ³Mayo Clinic, Phoenix, AZ, ⁴Arizona State University, Phoenix, AZ.

Introduction: Insomnia is a novel and modifiable risk factor for incident cardiovascular disease (CVD). However, identification of early markers of subclinical CVD in diagnosed insomnia is understudied. Our aim for this ongoing study is to contrast markers of cardiovascular structure and function between people with insomnia and good-sleeping controls.

Methods: Persons with insomnia (met ICSD-III criteria) and good sleeping controls (<8 Insomnia Severity Index, mean 8-night SOL and WASO<31min) were recruited from the community. Twentytwo adults (21-39y; 55% women) with no history of CVD, diabetes, inflammatory conditions, significant hypertension, or current sleep-disordered breathing (WatchPat200, Itamar Medical) were enrolled and underwent fasting cardiovascular testing. Testing included: Central augmented aortic pressure (AP) and carotidfemoral pulse wave velocity (cfPWV) for vascular stiffness; brachial artery flow mediated dilation (FMD) to assess endothelial function; and 2D echocardiography to assess ejection fraction (EF%), left ventricular global longitudinal strain (LVGLS), left atrial volume index (LAVI), mitral valve E/e' ratio (E/e'), and lateral e'. ANCOVA models, adjusting for age, comparing persons with insomnia (n=6) to good sleeping controls (n=16) on each cardiovascular measure were conducted.

Results: AP (range:-5,10mmHg), cfPWV (range: 4.8-7.6m/s), EF% (range:55.0-72.0%), LVGLS (range:-26,-19%) LAVI (range:14.1-26.7mL/m²), E/e' (range:3.2-7.8), and lateral e' (range:0.09-0.22cm/ sec) were all within normal ranges according to age and sex normative standards. Mean FMD was 8.8% (SD=4.3, range:4.3-19.8%). Age adjusted ANCOVA models indicated that the insomnia group had significantly worse cardiovascular function than good sleeping controls on cfPWV (M=6.8±0.3 vs. M=5.7±0.2; p=0.004), EF% $(M=60.0\pm1.7 \text{ vs. } M=65.2\pm1.0; p=0.017)$, LVGLS $(M=-21.6\pm0.6$ vs. $M=-24.3\pm0.4$; p=0.001), and lateral e' ($M=0.12\pm0.01$ vs. $M=0.18\pm0.01$; p=0.003). No group differences were found for AP, FMD, LAVI, and E/e'.

Conclusion: Among relatively healthy young adults, people with insomnia had greater arterial stiffness and worse left ventricular systolic and diastolic functioning.

Support: American Academy of Sleep Medicine Foundation Focused Projects Award for Junior Investigators 179-FP-18

0551

LONGITUDINAL MEASUREMENT INVARIANCE OF THE INSOMNIA SEVERITY INDEX IN VETERANS WITH SLEEP APNEA

Wohlgemuth, W.¹ Fins, A.² Tutek, J.³ Gonzalez, A.² Martinez-Garcia, A.² Satyanarayana, S.⁴ Marchetti, D.⁴ Wallace, D.⁵ ¹MIami VA, Miami, FL, ²Nova Southeastern University, Ft. Lauderdale, FL, ³Miami VA Sleep Center, Miami, FL, ⁴University of Miami, Miami, FL, ⁵University of Miami, MIami, FL.

Introduction: The Insomnia Severity Index is a commonly used instrument to assess the presence of insomnia symptoms as well as an outcome measure following an intervention. Longitudinal measurement invariance is a necessary property of an assessment instrument when it is repeated over time. The validity of conclusions regarding change in the construct 'insomnia severity' depend on scale equivalence at each measurement timepoint. Assessment of measurement invariance of the ISI in sleep apnea patients has never been performed.

Methods: Veterans with sleep apnea (n=654; AHI=36 \pm 28; 93% male; age=52 \pm 12; BMI=33 \pm 6) completed the ISI on the night of their overnight PSG and again when they picked up their PAP device. Invariance was determined by imposing a series of more restrictive equivalence constraints on a 2-factor model of the ISI. The series of constraints tested for configural, weak, strong and strict invariance. Invariance testing was modeled with exploratory structural equation modeling in Mplus (v. 7.0).

Results: The 2-factor model that emerged from the analysis showed items relating to nighttime symptoms loading on factor 1 and daytime symptoms loading on factor 2. The sleep 'satisfaction' item, however, had weak but similar loadings on both factors. The increasingly restrictive constraints imposed on the model revealed no decrement in model fit (RMSEA=.039 to.043; CFI=.987 to .980; TLI=.981-.977; SRMR=.027-.041).

Conclusion: The ISI met strict criteria for longitudinal measurement invariance demonstrating that it is a valid instrument to be used in repeated measures study designs of insomnia in sleep apnea patients. Change over time on the ISI is not due to the changing measurement characteristics of the ISI but to true changes in the 'insomnia severity' construct.

Support: None

0552

INVESTIGATING PRE-SLEEP PROCESSES AND HOW THEY INFLUENCE SLEEP: A DIARY AND ACTIGRAPHY STUDY

Maeder, T.¹ Whitford, J.¹ Feinaigle, P.¹ Karlen, W.² Seifritz, E.³ Pace-Schott, E. F.⁴ Kleim, B.¹

¹University of Zurich, Zurich, SWITZERLAND, ²Swiss Federal Institute of Technology Zurich, Zurich, SWITZERLAND, ³Psychiatric University Hospital Zurich, Zurich,

SWITZERLAND, ⁴Massachusetts General Hospital, Harvard Medical School, Charlestown, MA.

Introduction: The current study examined the relationship between pre-sleep processes and sleep in the context of real-world stress exposure in medical students during an internship. Medical students are often exposed to a variety of stressors and potentially traumatic events and have been shown to be at risk to develop psychopathology. Previous research has shown an association between stress, psychological distress, and sleep disturbances. In this context, studies have investigated possible predictors for sleep disturbances. Recently, the period just prior to sleep onset has received increased interest. At the moment, little is known, however, about the influence of such pre-sleep processes. In this study, we investigated the influence of pre-sleep rumination and mindfulness on sleep disturbances.

Methods: In a prospective study, we examined a sample of 50 medical students from the University of Zurich. All participants completed their first medical internship over 9 months as part of medical school. Pre-sleep processes and sleep were indexed mid-internship using sleep diaries over seven consecutive days. Additionally, a Fitbit tracking device was used to objectively measure sleep. Correlational analyses and multilevel linear models were conducted.

Results: Results show associations between pre-sleep processes and sleep disturbances in this stress-exposed at-risk population. Multilevel mixed-effects models showed that over the period of 7 consecutive nights, pre-sleep rumination was a significant predictor of lower subjective sleep quality (B = -.085, SE = .036, p = .02), lower subjective total sleep time (B = -.124, SE = .043, p = .005), higher subjective sleep onset latency (B = 1.535, SE = .678, p = .025), and higher subjective wake-up frequency (B = .033, SE = .011, p = .003).

Conclusion: Together, our data suggest pre-sleep processes as potential targets for stress-prevention programs that could help reduce the negative influence of stress in at risk populations. **Support:** NA

0553

HALLUCINOGEN USE AMONG COLLEGE AND UNIVERSITY STUDENTS: ASSOCIATIONS WITH INSUFFICIENT SLEEP AND INSOMNIA

Holbert, C.¹ Bastien, C.² c, S.³ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹

¹University of Arizona, Tucson, AZ, ²Universite Laval, Quebec City, QC, CANADA, ³University of Pennsylvania, Philadelphia, PA.

Introduction: Previous studies have shown that poor sleep is associated with alcohol use, smoking, and other substance use, especially among young adults. Yet, very little is known about hallucinogen use.

Methods: Data from the 2011-2014 National College Health Assessment were used (N=113,749), representing a wide range of students across the US. Hallucinogen use was reported as "never," "past," and "present" (reflecting use in the past 30 days). Students also self-reported nights/week they did not get enough sleep to feel rested (insufficient sleep), as well as nights/week they had difficulty falling asleep (initial insomnia). Responses for both were categorized as 0, 1-2, 3-4, 5-6, or 7 nights/week. Multinomial logistic regressions examined hallucinogen use as outcome (past or present vs never) and sleep as predictor, with adjustment for covariates (age, sex, race/ethnicity, and survey year) and mental health (past 30 days depression/anxiety).

Results: Hallucinogen use was infrequently reported, with 4.8% (N=5,493) reporting past use and 0.98% (N=1,119) reporting present use. In adjusted analyses, increase likelihood of past use was associated with insufficient sleep on 1-2 (RRR=1.28, p=0.001), 3-4 (RRR=1.37, p<0.0005), 5-6 (RRR=1.30, p<0.0005), and 7 (RRR=1.34, p<0.0005) nights per week, as well as 1-2 (RRR=1.30, p<0.0005), 3-4 (RRR=1.52, p<0.0005), 5-6 (RRR=1.58, p<0.0005), and 7 (RRR=1.49, p<0.0005) nights per week of initial insomnia. Present use was associated with 1-2 (RRR=1.44, p<0.0005), 3-4 (RRR=1.76, p<0.0005), 5-6 (RRR=2.05, p<0.0005), and 7 (RRR=1.83, p<0.0005) nights per week of initial insomnia. When mental health was entered into the model, results were maintained. Conclusion: Past use of hallucinogens was associated with insufficient sleep as well as insomnia. Present use was also associated with insomnia. When mental health was included in models, all results were maintained. It is not clear whether hallucinogen use leads to. or is predicted by, sleep difficulties.

Support: Dr. Grandner is supported by R01MD011600

0554

INSOMNIA DISORDER PREDICTS STRESSFUL LIFE EVENTS IN INDIVIDUALS WHO HAVE RECENTLY EXPERIENCED INVOLUNTARY JOB LOSS

Skobic, I.¹ Howe, G.² Haynes, P. L.¹

¹Health Promotion Sciences Department, Mel and Enid Zuckerman College of Public Health, University of Arizona, Tucson, AZ, ²Department of Psychological and Brain Sciences, Columbian College of Arts and Sciences, George Washington University, Washington D.C., DC.

Introduction: The stress generation hypothesis posits that depressed (versus non-depressed) individuals generate more stressful life events, especially events for which they are at least partly responsible (i.e., dependent events). Insomnia disorder interferes with emotion regulation, potentially leading to impaired decision-making and increased stress generation. We hypothesized that insomnia disorder would lead to an increased number of stressful life events in our sample of adults who have recently experienced involuntary job loss.

Methods: Assessing Daily Activity Patterns through Occupational Transitions is a longitudinal study examining linkages between job-loss, sleep, obesity, and mental health. We used baseline and 3-month follow-up data from 137 participants who completed the Life Events and Difficulties Schedule, a contextual life event measure. Insomnia disorder was coded yes if participants met ICSD-3 criteria for a current chronic or acute insomnia disorder on the Duke Structured Interview for Sleep Disorders. Covariates included age, gender, and race. Linear and logistic regression were employed to assess changes in number of events over time. Secondary analysis examined the relationship between insomnia and dependent stressful life events specifically.

Results: When controlling for covariates, insomnia disorder at study baseline predicted the number of stressful life events generated between baseline and 3-month follow-up (β =.60, se=.30, t=1.99, p=.05). Conversely, events at baseline did not predict insomnia disorder at follow-up when controlling for baseline insomnia disorder (OR=.98, CI=.82-1.17). Secondary analysis revealed a trend toward increased generation of dependent events among individuals with insomnia disorder (β =.37, se=.23, t = 1.6, p=.11).

Conclusion: Our analyses provide preliminary evidence for a causal relationship between insomnia disorder and stress generation.

SLEEP, Volume 43, Abstract Supplement, 2020

Additional research is needed to replicate and examine the mechanisms behind this relationship. This extension of the stress generation hypothesis may have important implications for harm reduction interventions for insomnia disorder. **Support:** #1R01HL117995-01A1.

he-

A212

THE RELATIONSHIP BETWEEN SLEEP EFFICIENCY AND APNOEA HYPOPNOEA INDEX (AHI) IN ADULT OBESE MALES

Ankita, A.¹ Mehta, B.¹ Dutt, N.¹ Sharma, P.¹ ¹AIIMS Jodhpur, JODHPUR, INDIA, ²AIIMS Jodhpur, JODHPUR, INDIA, ³AIIMS Jodhpur, JODHPUR, INDIA.

Introduction: Prevalence of obesity is increasing worldwide. According to OECD 2017, the prevalence of obesity is 19.5% worldwide. Obesity leads to disturbed sleep due to complete or partial obstruction of upper airways i.e. obstructive sleep apnoea. This disturbed sleep leads to increased sympathetic discharge & further obesity and thus forms a vicious cycle of obesity disturbing sleep and sleep disturbance increasing obesity. The purpose of the present study was to correlate the sleep efficiency with apnoea hypopnea index (AHI) in adult obese males.

Methods: Nineteen adult obese males (26 years- 60 years), nonsmokers were recruited for this cross-sectional study. The obesity criteria was taken as BMI ≥ 25 kg/m². They underwent an overnight polysomnographic examination with total 68 channels and 32 EEG inputs. The episodes of apnea were defined as complete cessation of airflow for ≥ 10 s, and hypopnea consisted of a $\ge 30\%$ reduction in oronasal airflow accompanied by a reduction in oxygen saturation measured by pulse oximetry of at least 4%. AHI was determined by the frequency of these events per hour during sleep time based on the results of the overnight polysomnography. Sleep efficiency index was calculated by dividing total duration of sleep stages (N1+ N2+ N3+ REM) by total time in bed.

Results: We tested for normality through Shapiro Wilk test and our data was found to be non-parametric. Hence Spearman correlation between sleep efficiency and AHI was performed. The correlation was non-significant with p value 0.1245 and r = -0.365. The correlation of BMI with sleep efficiency was significant (p= 0.0195) with r value= -0.5303.

Conclusion: The results conclude that the sleep efficiency worsens with obesity. Although the correlation between AHI and sleep efficiency was not found significant, a negative r value indicates that the sleep efficiency decreases with increased obstructive events during sleep.

Support: All India Institute of Medical Sciences Jodhpur

0556

SEX-SPECIFIC RELATIONSHIP BETWEEN ANXIETY AND AUTONOMIC NERVOUS SYSTEM DYSFUNCTION IN OBSTRUCTIVE SLEEP APNEA

Pal, A.¹ Akey, M. A.¹ Chatterjee, R.¹ Aguila, A. P.¹ Martinez, F.¹ Aysola, R.² Macey, P. M.¹

¹UCLA School of Nursing, University of California, Los Angeles, CA, ²Division of Pulmonary and Critical Care, University of California, Los Angeles, CA.

Introduction: Cardiovascular co-morbidities in obstructive sleep apnea (OSA) are hard to treat, perhaps due to autonomic nervous system (ANS) dysfunction. In OSA, intermittent hypoxia and poor tissue oxygen perfusion damage endothelial and nervous tissue, potentially underlying the dysfunction. Moreover, OSA is strongly associated with anxiety, which is independently associated with ANS dysfunction. We assessed sex-specific relationships between anxiety and cardiovascular markers of ANS dysfunction in OSA.

Methods: We studied people diagnosed with OSA and healthy controls. We collected 5 minutes of wakeful resting ECG, continuous

non-invasive blood pressure, and respiration data. We calculated heart rate (HR), heart rate variability (HRV; sympathetic-vagal balance related to brainstem ANS output), mean arterial blood pressure (MAP), beat-to-beat MAP variability (BPV; related to peripheral autonomic function) and breathing rate (BR). We analyzed these measures with a multivariate regression model of anxiety symptoms (generalized anxiety disorder; GAD-7 scores), sex, and group (OSA vs. control), age/BMI/AHI covariates, and Bonferroni-corrected post-hoc comparisons ($p \le 0.05$).

Results: We analyzed 64 subjects (32 OSA: AHI [mean±SEM] 24±4events/hour, 12 female, age 52±21years, BMI 33±2kg/m2; 32 control: 19 female, age 46±2; BMI 26±1). We observed significant main effects of anxiety, BMI, AHI, sex on HRV, but only group on BPV; post-hoc comparisons revealed high BPV only in OSA females. Secondary analyses included classifying by anxiety symptoms (GAD-7≥5), showing only OSA females with anxiety had higher BPV. Males showed higher HRV. AHI and anxiety were positively correlated with HRV in OSA males. AHI was negatively correlated with BR in OSA females.

Conclusion: We observed higher anxiety associated with higher BPV in OSA, especially in females. Unexpectedly, BR was lower in OSA females; longer breaths may have led to the greater BPV. Higher HRV in males complicated by OSA severity and anxiety could be related to higher sympathetic tone. The slightly older control group may have influenced the findings. Overall, our findings suggest anxiety in OSA is associated with peripheral and centrally-mediated autonomic dysfunction, but in a sex-specific manner. **Support:** National Institutes of Health R56-NR-017435 and RO1-HL-135562.

0557

POSSIBLE CONTRIBUTORY AND PROTECTIVE FACTORS IN MEDICATION ASSOCIATED OBSTRUCTIVE SLEEP APNEA (OSA): RESULTS FROM THE US FOOD AND DRUG ADMINISTRATION (FDA) ADVERSE EVENTS REPORTING SYSTEM (FAERS)

Gupta, M. A. Vujcic, B.

Department of Psychiatry, Schulich School of Medicine and Dentistry, University of Western Ontario, London, ON, CANADA.

Introduction: There are conflicting opinions regarding the role of medications in OSA. A Vigibase (WHO pharmacovigilance database) study (Linselle M, 2017) has suggested several drug groups in OSA pathogenesis. Sodium oxybate, benzodiazepines and opioids are most consistently associated with OSA. We examined the adverse drug reaction (ADR) of OSA with the 'primary suspect' drugs for the ADR, in the US Food and Drug Administration Adverse Events Reporting System (FAERS) database.

Methods: The FAERS database from January 1, 2004-March 31, 2019 (total ISR =12,330,939) was examined for Individual Safety Reports (ISR) with OSA as ADR and associated 'primary suspect (PS)' medications. Reporting odds ratios (ROR) were calculated to assess disproportionality signals with the 'PS' drug and OSA versus the 'PS' drug associated with all other ADRs in the database.

Results: 15,316 ISR were associated with OSA [mean \pm SD age: 49.21 \pm 21.76 years (based on 10,311 ISR); 53.78% female (based on 12,274 ISR]. Increased disproportionality signals for OSA were detected with some of the following 'PS' drugs/drug groups: so-dium oxybate [ROR=31.75, (95% CI 30.36-33.20)]; rofecoxib [ROR=7.85 (95% CI 7.40-8.32)], alendronate [ROR=3.60 (95% CI 3.37-3.84)], zoledronic acid [ROR=24.70 (95% CI 21.88-27.89)],

omalizumab [ROR=2.36 (95% CI 2.09-2.68)], quetiapine [ROR=3.78 (95% CI 3.32-4.30), finasteride [ROR=6.03 (95% CI 5.17-7.04), pregabalin [ROR=1.19 (95% CI 1.01-1.41), isotretinoin [ROR=1.49 (95% CI 1.23-1.80)], ondansetron [ROR=3.35 (95% CI 2.77-4.06), olanzapine [ROR=2.64 (95% CI 2.17-3.20), sitagliptin [ROR=3.46 (95% CI 2.82-4.24, digoxin [ROR=2.83 (95% CI 2.24-3.57), benzodiazepines [ROR=1.80 (95% CI 1.33-2.42), and opioids [ROR=1.34 (95% CI 1.09-1.67)]. A decreased disproportionality signal was detected with several biologics including: adalimumab [ROR=0.87 (95% CI 0.79-0.95)], etanercept [ROR=0.55 (95% CI 0.49-0.61)], and infliximab [ROR=0.51 (9% CI 0.40-0.65)].

Conclusion: The FAERS data supports many of the earlier findings suggesting the heterogeneity of medications associated with OSA. Biologics (mainly TNF-alpha antagonists) were associated with the previously unreported finding of a decreased OSA risk. **Support:** None.

0558

SLEEP DISORDERED BREATHING AND RIGHT VENTRICULAR ELECTROCARDIOGRAPHIC AND FUNCTIONAL CHARACTERISTICS IN GROUP 1 PULMONARY ARTERIAL HYPERTENSION

Bhat, A. S.¹ Wang, L.² Kaur, S.¹ Nawabit, R.¹ Highland, K.³ Park, M.⁴ Jellis, C.⁴ Kwon, D.⁴ Hill, N.⁵ Mehra, R.¹ PVDOMICS. P.⁶

¹Cleveland Clinic Neurologic Institute, Cleveland, OH, ²Cleveland Clinic Lerner Research Institute, Cleveland, OH, ³Cleveland Clinic Respiratory Institute, Cleveland, OH, ⁴Cleveland Clinic Heart and Vascular Institute, Cleveland, OH, ⁵Tufts Medical Center Pulmonary, Critical Care & Sleep Divission, Boston, MA, ⁶National Heart, Lung, Blood Institute (NHLBI), Bethesda, MD.

Introduction: Right ventricular (RV) electrophysiologic and functional alterations related to sleep disordered breathing (SDB) in pulmonary arterial hypertension (PAH) are not well understood. We hypothesize an association between SDB and RV electrophysiological/functional measures in World Symposium of Pulmonary Hypertension (WSPH) Group 1 PAH.

Methods: The NHLBI multicenter **PVDOMICS** study (NCT02980887) enrolls patients with PAH undergoing a battery of assessments including home sleep apnea testing(NOX-T3, Carefusion®) or with historical sleep study data. Logistic(OR,95%CI) and linear(beta coefficients,95%CI) regression models adjusted for age, sex, race, body mass index (BMI, kg/m²), PAH medications, supplemental oxygen(O2), positive airway pressure(PAP) were used to assess associations of SDB(apnea hypopnea index, (AHI), $\geq 3\%$ desaturations(hypopnea), percentage recording time with SaO2<90% (TRT<90%) with electrocardiographic measures: RV hypertrophy(RVH), right bundle branch block(RBBB), and right axis deviation(RAD), echocardiographically-derived RV systolic pressure(RVSP) and RV ejection fraction(RVEF) from cardiac MRI. Analyses were performed based on an overall significance level of 0.05, using SAS software (version 9.4, Cary, NC).

Results: The analysis consisted of 182 PAH participants with age: 52.5 ± 13.9 years, 71.4% female, 88.9% Caucasian, BMI: 30.3 ± 7.8 kg/m², RVEF: 37.3 ± 11.6 , and RVSP: 67.0 ± 23.4 . None of the electrocardiographic measures were associated with AHI and only RVH was significantly associated with TRT<90% (1.25:1.09,1.43),p=0.001. Although AHI was not associated with RVSP, a 10% increase in TRT<90% was associated with a 2.60mmHg increase in RVSP (2.60:1.44,3.76),p<0.001. Each

10-unit increase in AHI was associated with a 2.72% reduction of RVEF (-2.72:-4.89,-0.56),p=0.014, and each 10-unit increase in TRT<90% was associated with a 0.72% reduction of RVEF (-0.72:-1.38,-0.06),p=0.033.

Conclusion: We identify nocturnal hypoxia as a predictor of RV electrophysiological and functional alterations even after consideration of confounding factors. SDB as determined by AHI was also more so associated with reduced RVEF than hypoxia. Future mechanistic studies should focus on further elucidation of SDB and nocturnal hypoxia on pathogenesis of RV dysfunction in PAH. **Support:** U01HL125218/U01HL125205/U01HL125212/U01HL125208/U01HL125175/U01HL125215, U01HL125177/Pulmonary Hypertension Association

0559

ASSOCIATION OF RAPID EYE MOVEMENT SLEEP WITH INSULIN RESISTANCE IN HAN CHINESE PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

ZHANG, C.^{1,2,3} XU, H.^{1,2,3} ZOU, J.^{1,2,3} GUAN, J.^{1,2,3} YI, H.^{1,3,2} YIN, S.^{1,2,3}

¹Department of Otolaryngology Head and Neck Surgery and Center of Sleep Medicine, Shanghai Sixth People's Hospital Affiliated to Shanghai Jiao Tong University, Shanghai, CHINA, ²Shanghai Key Laboratory of Sleep Disordered Breathing, Shanghai, CHINA, ³Otolaryngological Institute of Shanghai Jiao Tong University, Shanghai, CHINA.

Introduction: Obstructive sleep apnea (OSA) is increasingly associated with insulin resistance. The underlying pathophysiology remains unclear but rapid eye movement (REM) sleep has been hypothesized to play a key role. To investigate the associations of insulin resistance with respiratory events and sleep duration during REM sleep, 4,062 Han Chinese individuals with suspected OSA were screened and 2,899 were analyzed.

Methods: We screened 4,062 participants with suspected OSA who underwent polysomnography in our sleep center from 2009 to 2016. Polysomnographic variables, biochemical indicators, and physical measurements were collected. Logistic regression analyses were conducted to determine the odds ratios (ORs) and 95% confidence intervals (95% CIs) for insulin resistance as assessed by hyperinsulinemia, the homeostasis model assessment of insulin resistance (HOMA-IR), fasting insulin resistance index (FIRI), and Bennet's insulin sensitivity index (ISI).

Results: The final analyses included 2,899 participants. After adjusting for age, gender, body mass index, waist circumference, mean arterial pressure, smoking status, alcohol consumption, and the apnea and hypopnea index during non-REM sleep (AHI_{NREM}), the results revealed that AHI during REM sleep (AHI_{REM}) was independently associated with insulin resistance; across higher AHI_{REM} quartiles, the ORs (95% CIs) for hyperinsulinemia were 1.340 (1.022, 1.757), 1.210 (0.882, 1.660), and 1.632 (1.103, 2.416); those for abnormal HOMA-IR were 1.287 (0.998, 1.661), 1.263 (0.933, 1.711), and 1.556 (1.056, 2.293); those for abnormal FIRI were 1.386 (1.048, 1.835), 1.317 (0.954, 1.818), and 1.888 (1.269, 2.807); and those for abnormal Bennet's ISI were 1.297 (1.003, 1.678), 1.287 (0.949, 1.747), and 1.663 (1.127, 2.452) (P < 0.01 for all linear trends). Additionally, the results showed that for every 1-h increase in REM duration, the risk of hyperinsulinemia decreased by 22.3% (P < 0.05).

Conclusion: The present study demonstrated that AHI_{REM} was independently associated with hyperinsulinemia and abnormal HOMA-IR, FIRI, and Bennet's ISI. Additionally, REM sleep duration was independently associated with hyperinsulinemia.

Support: This study was supported by Grants-in-aid from Shanghai Municipal Commission of Science and Technology (No.18DZ2260200).

0560

THE EFFECT OF SMOKING ON OSA ENDOTYPES: A RETROSPECTIVE COHORT STUDY

Al-Azzawi, S. Orr, J. E. De Young, P. Owens, R. L. Malhotra, A. Schmickl, C. N.

University of California, San Diego, La Jolla, CA.

Introduction: Smoking is a purported risk factor for obstructive sleep apnea (OSA), but the mechanisms through which smoking may cause OSA are largely unclear. Our goal is to assess the effect of smoking on the pathophysiological traits ("endotypes") underlying OSA.

Methods: Based on a chart review we are creating a retrospective cohort of consecutive patients who were newly diagnosed with OSA based on an inlab polysomnogram between 1/2016 and 6/2018 and who have a documented smoking status. For each subject we are quantifying the endotypes (e.g. arousal threshold, loop gain, upper airway muscle recruitment) via a validated polysomnography-based algorithm. Additionally, we are estimating the arousal threshold based on a clinical prediction score. We are comparing OSA endotypes (primary outcomes), sleep apnea severity (apnea-hypopnea index, SpO₂ nadir) and sleep parameters (e.g. total sleep time, sleep efficiency, sleep stages) in current vs former vs never smokers using Kruskal-Wallis tests (+Dunn's test for *post hoc* comparisons).

Results: To date we have screened 334 of 2,138 subjects and identified 99 eligible subjects (5 current smokers at the time of polysomnography, 37 former smokers, and 57 never smokers). The clinical arousal threshold was similar across groups (P=.69); polysomnography-based endotype measures are pending. Further, there was no significant difference in sleep apnea severity or sleep parameters across groups, except stage N2 which was less in current vs former smokers (median-percentage 48.5 vs 66.3%, P<.05) and less in never vs former smokers (61.6 vs 66.3%, P<.05).

Conclusion: Overall, former vs never smokers appear to be similar with regards to sleep and sleep apnea parameters. Prevalence of current smokers appears to be low (5%) in our cohort; larger sample size and polysomnography-based endotypes are needed before firm conclusions about the effects of smoking on OSA mechanisms can be reached (data collection continues).

Support: This study had no specific funding. Christopher Schmickl is supported by NIH T32 grant HL134632.

0561

DIFFERENCES IN NASOPHARYNGOSCOPIC AIRWAY FORM BETWEEN AWAKE AND SLEEP, SITTING AND RECUMBENT POSITION AND TECHNIQUES

Nishimura, Y.¹ Hamed, M.²

¹Division of Otolaryngology, Teikyo University Chiba Medical Center, Chiba, JAPAN, ²Department of Otolaryngolgoy, Sohag University, Sohag, EGYPT.

Introduction: To examine and compare the information derived from flexible fiber-optic nasopharyngoscopy in awake mimic snoring (AMS), Müller's Maneuver (MM) and drug-induced sleep endoscopy (DISE), to determine if AMS and MM can be used in substitution for DISE as a streamlined method. We investigated their relation with the level and pattern of obstruction detected on AMS, MM and DISE.

Methods: This is a retrospective study of 15 obstructive sleep apnea patients with apnea hypopnea index from 8.3 to 105.2, ages 20 to 80 were included. Each patient underwent polysomnography and thorough a physical examination, including flexible nasopharyngoscopy with AMS, MM and DISE. Airway obstruction on these endoscopic procedures were described according to airway level and pattern of obstruction. They were classified 5 different types; Uvula type: anterior-posterior vibration of the uvula, no airway obstruction; L-R velum type: lateral (the left and right directions) airway narrowing at velum level. no airway obstruction: A-P velum type: anterior-posterior total airway obstruction at velum level; Tonsillar type: total airway obstruction at pharyngeal level; Circumferential type: circumferential total airway obstruction at velum level. AMS and MM were performed with patients in sitting and in recumbent position. DISE was performed only in recumbent position.

Results: In review of the three procedures, the results were much different. Airway was obstructed in all cases(100%, 15 of 15)in DISE, but not all cases in AMS and MM. When tonsillar type was seen in AMS, it was also seen in MM and DISE(100%, 5 of 5).

Conclusion: Flexible fiber-optic nasopharyngoscopy appears to be useful for evaluating airway obstruction. It might be not suitable to use AMS and MM in substitution for DISE(except tonsillar type). Muscle tonic relaxation of the upper airway between AMS, MM and DISE might be different (DISE>MM>AMS, recumbent>sitting).

Support:

0562

DIFFERENCES IN UPPER AIRWAY SOFT TISSUE VOLUMES BETWEEN AFRICAN AMERICAN AND CAUCASIAN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA USING MAGNETIC RESONANCE IMAGING

Webster, J. C.¹ Keenan, B.² Schwab, R. J.²

¹University of Pennsylvania, Philadelphia, PA, ²The University of Pennsylvania, Philadelphia, PA.

Introduction: Research suggests greater obstructive sleep apnea (OSA) severity in African-Americans than Caucasians. However, the underlying mechanisms causing this ethnic disparity are unknown. To evaluate possible mechanisms, we compared the size of the tongue between African American and Caucasian OSA patients using magnetic resonance imaging (MRI), controlling for age, body mass index (BMI) and apnea-hypoxia index (AHI). Given prior evidence of more severe OSA in African Americans, we hypothesized these patients would have larger soft tissue volumes compared to Caucasians.

Methods: Upper airway soft tissue volumes, (total tongue, tongue fat, lateral walls, pterygoids, total soft tissue) were quantified using MRI and compared between Caucasian (n=133) and African American (n=175) patients with moderate OSA. Analyses were conducted using regression models controlling for age, sex, BMI and AHI.

Results: Among all OSA patients, African Americans had higher BMI than Caucasians (40.0 ± 8.6 vs. 37.1 ± 8.1 kg/m2, p=0.0024) and a higher proportion of females (66.3% vs. 36.1%; p<0.0001). There were no significant differences in age (p=0.143) or AHI (p=0.314). Controlling for these covariates, there were no differences between African American and Caucasian OSA patients in tongue fat volume (mean [95% confidence interval] difference = 479 [-3156, 4115] mm3; p=0.794). However, African Americans had a 13,286 (6,439, 20,132) mm3 larger total tongue volume compared

to Caucasians (p=0.0002). Larger volumes in African Americans were also observed for the soft palate (p<0.0001), retropalatal lateral walls (p=0.003), pterygoid (p=0.034) and total soft tissue volume (p=0.0003).

Conclusion: African Americans were observed to have larger volumes of the tongue, soft palate, retropalatal lateral walls, pterygoids and total soft tissue volume compared to Caucasians, although there were no differences in AHI and tongue fat volume. The study contributes to the overall understanding of ethnic-specific pathology of OSA and can potentially influence how African Americans and Caucasians are diagnosed and treated specifically for the disease.

Support:

0563

INSPIRATORY FLOW LIMITATION IN NON OSA INDIVIDUALS: RISK FACTORS AND CLINICAL COMPLAINTS AFTER 8 YEARS OF FOLLOW IN A GENERAL POPULATION

Palombini, L.¹ de Godoy, L. B.² Andersen, M. L.¹ Poyares, D.¹ Tufik, S.²

¹Universidade Federal de São Paulo, São Paulo, BRAZIL, ²Universidade Federal de São Paulo - Brazil, São Paulo, BRAZIL.

Introduction:

Inspiratory flow limitation (IFL) indicated by the flattening of inspiratory curve, is a respiratory pattern in polysomnography (PSG) suggested as a parameter used to identify individuals with sleep breathing disorder. The cutoff value is still not well defined. The purpose of this study was to evaluate the frequency and clinical impact of IFL after 8-year follow up ingeneral population.

Methods: MethodsBaseline sample was derived from a prospective population-based study (Epidemiologic Sleep Study, EPISONO). A total of 1,042 subjects completed the study assessments in 2007 and 712 from these participants were reassessed in 2016.Full night PSG at baseline and follow-up was performed and clinical outcomes were analyzed. IFL was manually scored and TST spent in IFL during sleep was calculated.

Results: In 2007 and 2016, IFL in non OSA individuals' mean value was 10.1% and 17.7% of TST and 95th percentile was 37.8% and 49.9% respectively. OSA patients had higher BMI than nonOSA in 2007 (p = 0.01), however, BMI and age were not risk factors to develop OSA or to increase IFL after 8 years. Individuals with IFL that became OSA after 8 years had a higher BMI, regardless of time, than those that did not develop OSA. Individuals who presented <15% of TST with IFL in 2007 and changed to ≥15% in 2016demonstrated significantly worse Beck depression score compared to group that maintained IFL < 5%. Individuals with ≥ 15% of TST with IFL in 2007 and in 2016 also increased depression severity score and Whoqol social relationship and environment domainscompared to the group with <15%.

Conclusion:

Non OSA individuals presented 10.1% in 2007 and 17.7% in 2016 of TST in IFL. After 8 years, individuals with ≥15% of IFL presented worsening of depression severity and sleep quality.

Support: The authors would like to thank for the support by grants from Associação Fundo de Incentivo a Pesquisa (AFIP), Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP), Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) and Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES).

0564

ASSESSMENT OF TONGUE AND SOFT PALATE MUSCLES MECHANICAL PROPERTIES IN PATIENTS WITH OSA

Li, W. Gakwaya, S. Sériès, F.

Unité de recherche en pneumologie, Centre de recherche, Institut Universitaire de Cardiologie et de Pneumologie de Québec, Université Laval., Québec, QC, Canada, QC, CANADA.

Introduction: Soft palate muscles are crucial in the maintenance of UA patency. Different contraction tasks have been used to investigate tongue mechanical properties, but not to soft palate muscles. This study aimed to investigate the mechanical consequences of tongue and soft palate muscles fatigue in moderate-severe OSA patients.

Methods: 12 moderate and 8 severe patients with OSA were enrolled. Measurements include strength, endurance, and fatigue indices. During the soft palate fatiguing protocol, subjects were asked to develop repetitive intra-oral positive pressure during cheek-bulging maneuvers while wearing a mouth piece to keep the jaw opened. Tongue mechanical properties were also assessed using protrusion tasks with similar protocol. Subjects were encouraged to develop sustained maximal bulging pressure or tongue protrusion force for 5 sec every 10 sec until the peak pressure did not reach 85% of baseline maximal pressure for 2 consecutive times. The influence of age and BMI were also investigated.

Results: The sex, age were not significantly different between the 2 OSA groups. BMI was significantly higher in severe OSA patients (p<0.05). Overall, the tongue maximal voluntary contraction force (MVC), endurance time and total muscle work were respectively positively associated with the ones obtained from the soft palate fatiguing task (r_s =0.51, 0.43, 0.66, respectively). The MVC of both tongue and soft palate muscles were positively correlated with BMI in all subjects (r_s =0.43, 0.5 respectively). The recovery time from soft palate fatigue was significantly longer in moderate than severe OSA patients (270s ± 192.3s and 120s ± 0, p =0.02). Interestingly, the recovery time was positively correlated with AHI in tongue fatiguing task, while negatively correlated with supine AHI and age in soft palate fatiguing task (p<0.05). In both tasks, MVC was negatively correlated with the endurance time (p<0.05).

Conclusion: Moderate patients are less likely to recover from soft palate muscle fatigue. A more severe apneic disease is associated with longer recovery time from tongue fatigue, but with shorter recovery time from soft palate fatigue. Our results suggest that alteration in tongue and velopharyngeal muscles function may differ according to the severity of disease.

Support: SBD from IUCPQ Foundation.

0565

OBSTRUCTIVE SLEEP APNEA, BUT NOT SHORT SLEEP DURATION, IS INDEPENDENTLY ASSOCIATED WITH INSULIN RESISTANCE: A LARGE-SCALE COHORT STUDY

YIN, S.^{1,2,3} XU, H.^{1,2,3} ZOU, J.^{1,2,3} ZHANG, C.^{1,2,3} GUAN, J.^{1,2,3} YI, H.^{1,2,3}

¹Department of Otolaryngology Head and Neck Surgery and Center of Sleep Medicine, Shanghai Sixth People's Hospital Affiliated to Shanghai Jiao Tong University, Shanghai, CHINA, ²Otolaryngological Institute of Shanghai Jiao Tong University, Shanghai, CHINA, ³Shanghai Key Laboratory of Sleep Disordered Breathing, Shanghai, CHINA.

Introduction: Both short sleep duration and obstructive sleep apnea (OSA) seem to be associated with insulin resistance. However, the majority of previous studies addressing the relationship between OSA and insulin resistance did not evaluate short sleep duration, and vice versa. In this study, we used a large-scale hospital-based cross-sectional dataset, including 5,447 participants, to examine 1) whether objectively measured short sleep duration and OSA are independently associated with insulin resistance, and 2) whether the presence of OSA modulates the association between sleep duration and insulin resistance.

Methods: Participants were consecutively enrolled from our sleep center during the period from 2007 to 2017. The index of homeostasis model assessment insulin resistance (HOMA-IR) was calculated from insulin and glucose. Sleep duration was determined by standard polysomnography. The associations between sleep duration and insulin resistance were estimated by logistic regression analyses.

Results: A total of 5,447 participants (4507 OSA and 940 primary snorers) were included in the study. In comparison to primary snorers, OSA combined with extremely short sleep duration (< 5 hours) increased the risk of insulin resistance by 34% (OR, 1.34; 95% CI, 1.01-1.77) after adjusting for confounding factors that are frequently associated with insulin resistance and OSA. In subgroup analysis stratified by sleep duration, the risk of insulin resistance in patients with a short sleep duration (5-6 hours or < 5 hours) was increased in those with OSA compared to primary snorers, but not in the other three sleep duration groups (6 - 7, 7 - 8, and > 8 hours). **Conclusion:** OSA, but not short sleep duration, was independently associated with insulin resistance. It is worth noting that OSA combined with extremely short sleep duration showed a greater detrimental effect than OSA itself with regard to insulin resistance.

Support: This study was supported by grants-in-aid from Shanghai Municipal Commission of Science and Technology (Grant No.18DZ2260200).

0566

ASSESSMENT OF SOFT PALATE MUSCLE FATIGUE AND ITS EFFECT ON VELOPHARYNGEAL UPPER AIRWAY (UA) MECHANICAL PROPERTIES

Li, W.¹ Gakwaya, S.¹ Masse, J.² Series, F.¹

¹Unité de recherche en pneumologie, Centre de recherche, Institut Universitaire de Cardiologie et de Pneumologie de Québec, Université Laval., Québec, QC, Canada, QC, CANADA, ²Université Laval., Québec, QC, Canada, QC, CANADA.

Introduction: Soft palate muscles are crucial in the maintenance of UA patency. This study aimed to investigate the fatigability of soft palate muscles and to quantify its effects on velopharyngeal UA dynamic properties in OSA patients and control subjects.

Methods: 8 control (AHI \leq 10 /h), 21 OSA patients (13 with mild/ moderate disease: 10 /h < AHI \leq 20 /h and 8 with moderate/severe: AHI > 20/h) were included in the study. Subjects were asked to develop repetitive intra-oral positive pressure during cheekbulging maneuvers while wearing a mouth piece to keep the jaw opened. Subjects were asked to develop sustained maximal bulging pressure for 5 sec every 10 sec until the peak pressure could not reach 85% of baseline maximal pressure for 2 consecutive times. UA dynamic properties were assessed by measuring instantaneous airflow and velopharyngeal pressure in response to phrenic nerve magnetic stimulation (PNMS) performed before, immediately and every 3 minutes after the fatiguing protocol for a maximum of 30 II. Sleep-Related Breathing Disorders

minutes' recovery time. UA closing pressure (Pcrit) was estimated by modeling the flow/pressure relationship in response to PNMS. Results: The sex, age, BMI and the soft palate mechanical properties (including the baseline strength, endurance time, total muscle work) did not significantly differ between the 3 groups. Maximal peak bulging pressure measured using cheek-bulging maneuver significantly changed following the fatigue task (p < 0.05). Baseline velopharyngeal Pcrit were less negative in moderate/severe OSA group compared to mild/moderate OSA (- 6.5 ± 2.6 vs. - 11.9 ± 3.2 , p < 0.05). In mild/moderate OSA patients, PNMS-induced drop in maximal instantaneous airflow tend to increase 3 mins after the fatiguing trial compared to baseline (22.7±21.11.s⁻¹vs. 9.6±5.81.s⁻¹, p < 0.1), and their Velopharyngeal linear resistance 3 mins after the fatiguing trial tend to be higher than the moderate/severe OSA group $(3.9 \pm 5.0 \text{ cmH}_2\text{O}\cdot\text{l}^{-1}\cdot\text{s}^{-1} \text{ vs. } 1.8 \pm 1.1 \text{ cmH}_2\text{O}\cdot\text{l}^{-1}\cdot\text{s}^{-1}, p < 0.1).$ Conclusion: The cheek-bulging maneuver could induce soft palate muscle fatigue, with no difference observed in soft palate mechanical performances among patients with different OSA severity. The fatiguing maneuver could further alter velopharyngeal UA mechanical properties in patients with mild/moderate OSA. Support: SBD from IUCPQ Foundation

0567

HYPOXIC BURDEN AND APNEA-HYPOPNEA DURATION IN PATIENTS WITH POSITIONAL OBSTRUCTIVE SLEEP APNEA

Ramzy, J. A. Rengan, R. Mandal, M. Rani, S. Vega Sanchez, M. E. Jaffe, F. D'Alonzo, G. Shariff, T. Chatila, W. Weaver, S. Krachman, S.

Lewis Katz School of Medicine at Temple University, Philadelphia, PA.

Introduction: Recently, the measurement of the hypoxic burden and apnea-hypopnea duration has been shown to correlate with mortality in patients with obstructive sleep apnea (OSA). We hypothesized that in patients with mild positional OSA (apnea-hypopnea index [AHI] < 5 events/hr in the non-supine position) the hypoxic burden would be increased and apnea-hypopnea duration shortened and similar to patients with non-positional OSA.

Methods: Fourteen patients with positional OSA and 24 patients non-positional OSA with similar severity of OSA based on the respiratory event index (REI) were included. All patients had a home sleep apnea test for suspected OSA. The hypoxic burden was calculated by the multiplication of REI and the mean area under the desaturation curves.

Results: Thirty-eight patients [12 (35%) males, 50±12 yrs, BMI 35±7 kg/m², Epworth Sleepiness Scale (ESS) 11±8, REI 10±3 events/hr, apnea-hypopnea duration 19±4 sec, mean SaO₂ 94±2%, lowest SaO₂ 79±8%, % total sleep time (TST) SaO₂ < 90% 11±16%, hypoxic burden 30±17 %min/hr] completed the study. Fourteen patients [9 (64%) males, 46±14 yrs, BMI 31±6 kg/m², ESS 7±5, REI 9±3 events/hr, mean SaO₂ 94±2%, lowest SaO₂ 81±6%, %TST SaO₂ < 90% 4±6%] had positional OSA (supine REI 16±7 events/hr, non-supine REI 3±1 events/hr) and 24 patients had non-positional OSA [3 (13%) males, 52±10 yrs, BMI 38±7 kg/m², ESS 12±9, REI 10±3 events/hr, mean SaO₂ 94±2%, lowest SaO₂ 77±9%, %TST SaO₂ < 90% 14±19%]. The hypoxic burden was elevated in both the positional and non-positional OSA patients with no difference between the groups (26±19 %min/hr and 32±15 %min/hr, respectively, p=0.13). The apnea-hypopnea duration was

similar in positional and non-positional OSA patients (20 ± 3 sec and 18 ± 4 sec, respectively, p=0.08 sec).

Conclusion: In patients with mild positional OSA the hypoxic burden, which has been associated with cardiovascular mortality, is elevated and similar to patients with non-positional OSA. **Support:** None

0568

PHYSIOLOGIC OSA TRAITS AND CPAP ADHERENCE AMONG PATIENTS WITH CORONARY ARTERY DISEASE AND OSA

Zinchuk, A.¹ Yaggi, H.¹ Liang, J.¹ Chu, J.¹ Op De Beeck, S.² Stepnowski, C.³ Wellman, A.⁴ Peker, Y.⁵ Sands, S.⁴ ¹Yale University, New Haven, CT, ²University of Antwerp, Antwerp, Belgium, BELGIUM, ³Veterans Medical Research Foundation, San Diego, CA, ⁴Brigham and Women's Hospital, Boston, MA, ⁵Koc University, Istanbul, TURKEY.

Introduction: Obstructive sleep apnea (OSA) is common in patients with coronary artery disease (CAD), but adherence to continuous positive airway pressure (CPAP) in this population is poor. Low arousal threshold (ArTH), a pathophysiologic OSA trait, is associated with low rates of regular CPAP use in sleep clinic populations. We aimed to determine whether ArTH or other physiologic OSA traits (i.e. pharyngeal collapsibility, muscle compensation, loop gain) are associated with CPAP adherence in patients with CAD and OSA.

Methods: A secondary analysis of a randomized controlled trial of OSA treatment in patients with CAD (RICCADSA) was performed. OSA (apnea hypopnea index, AHI≥5/hour) was assessed by polysomnography. Arousal threshold (% eupneic ventilation, $%V_e$), loop gain (LG), pharyngeal collapsibility ($%V_e$) and compensation ($%V_e$) were estimated from polysomnography using a validated method. Adherence to auto-titrated CPAP (hours/night) was obtained from machine downloads at 1, 3, 6, 12 and 24 months. Mixed modelling was used to assess the association between OSA traits and CPAP adherence.

Results: Participants (n=262) were 64.1±7.9 years old, with BMI of 29.2±4.2 and 86% were men. The mean AHI was 40.8±23.6 events/ hour with oxygen nadir of 81.3±7.1%. The median (IQR) CPAP adherence (hrs/night) was 3.0 (0.9, 5.8) at 1-mo and 3.0 (0.0, 5.6) at 24-mo. Compared to reference studies, the CAD patients exhibited an elevated LG 0.63 (0.53, 0.79), similar ArTH (%V_e) of 117.5% (106.5%, 136.4%), higher collapsibility (%V_e) at 90.1% (82.3%, 94.8%) and lower compensation (%V_e) at 3.7% (-0.7%, 8.7%).Only increasing pharyngeal muscle compensation was associated with lower CPAP adherence (β -0.04, p-value 0.048), effect modified by pharyngeal collapsibility (Compensation x Collapsibility, β <0.01, p-value 0.042).

Conclusion: In this group of patients with CAD, increasing muscle compensation was associated with lower CPAP adherence. Physiologic OSA traits may provide insight into prediction of CPAP adherence among patients with OSA and CAD.

Support: Zinchuk: Parker B. Francis Fellowship Program in Clinical Research. Sands: American Heart Association. Peker: Swedish Research Council, Swedish Heart-Lung Foundation.

0569

SOFT PALATE FAT BETWEEN LEAN ADULTS WITH OBSTRUCTIVE SLEEP APNEA AND HEALTHY CONTROL Xu, L. Keenan, B. T. Wiemken, A. S. Pack, A. I. Schwab, R. J.

Division of Sleep Medicine, Department of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania., Philadelphia, PA.

Introduction: Previous studies have shown that obese patients with obstructive sleep apnea (OSA) have a significantly greater percentage of fat tissue in soft palate than normal subjects. However, the influence of soft palate fat is not clear in non-obese adults with OSA. This study compared the volume of fat in the soft palate between lean adults with OSA and lean controls.

Methods: We examined soft palate fat in 21 lean OSA cases and 16 lean controls with body mass index (BMI) <25 kg/m². All subjects underwent a magnetic resonance imaging (MRI) with three-point Dixon scan. We used volumetric reconstruction algorithms to quantify the amount of soft palate fat, which was compared between apnecis and controls. Analysis reproducibility was quantified using intraclass correlation coefficients (ICC) from repeated analyses of 20 randomly-chosen MRIs.

Results: Analysis of soft palate fat was highly reproducible, with an ICC (95% confidence interval) of 0.968 (0.923, 0.987). Lean apneics were younger than lean controls (45.3 ± 13.0 vs. 62.1 ± 10.4 years; p<0.0001). No significant differences between apneics and controls were observed in the average BMI (23.4 ± 2.2 vs. 23.5 ± 2.6 kg/m²; p=0.824), the fat pads volume (4198 ± 1728 vs. 3880 ± 1544 mm³; p=0.646), and the proportion of males (61.9% vs. 68.8%; p=0.666). In unadjusted analyses, the lean OSA group showed significantly higher soft palate fat volume than lean controls (7605 ± 2109 vs. 5327 ± 1783 mm³; p=0.003). When adjusting for age, gender and BMI, no differences was observed between groups in soft palate fat volume (p=0.702).

Conclusion: Analysis of soft palate fat volume from Dixon MRI is highly reproducible. Our results indicate no significant difference in deposition of fat at soft palate between lean patients with OSA and lean controls when accounting for age, gender and BMI.

Support: This study is supported by National Institutes of Health Grant: 2P01HL094307-06A1. LX is supported by Young Elite Scientists Sponsorship Program of China Association for Science and Technology.

0570

APNEA-HYPOPNEA INDEX IS POSITIVELY CORRELATED WITH MOOD DISTURBANCE

Stubbers, K. M. Thosar, S. S. Butler, M. P. Bowles, N. P. McHill, A. W. Berman, A. M. Herzig, M. X. Roberts, S. A. Clemons, N. A. Morimoto, M. Shea, S. A. Emens, J. S. Oregon Health and Science University, Portland, OR.

Introduction: The prevalence of mood disorders such as depression is higher in individuals with obstructive sleep apnea (OSA). Previous studies have found no significant correlation between the apnea-hypopnea index (AHI) and measures of mood and have only included participants who met diagnostic criteria for OSA. The current analysis sought to determine whether mood correlated with AHI in individuals with any AHI values including those that did not meet diagnostic criteria for OSA.

Methods: 31 volunteers were studied (BMI=29.2 \pm 1.0 kg/m², mean \pm SE), free from medication and without psychiatric illness or chronic medical conditions with the exception of untreated OSA, uncomplicated hypertension (BP<160/100), or obesity. Following 1-3 weeks of an 8h habitual at home sleep schedule, participants completed the POMS-Brief questionnaire (POMS-B) to assess mood after undergoing overnight polysomnography to determine AHI. Total mood disturbance (TMD) scores were calculated by

adding the scores on the POMS-B for each mood state subscale and subtracting the score for vigor-activity.

Results: The average AHI was 15.3 ± 3.1 (range of 1.1-74.1) events per hour. The average POMS-B TMD score was 21 ± 1.5 (range of 4-46). There was a significant correlation between the POMS-B TMD score and AHI (p=0.037, r²=0.14). This result was also seen in only those individuals with AHI scores >5 (p=0.002, r²=0.4).

Conclusion: In this sample, individuals with higher AHI values displayed higher TMD scores. These results differ from previous data that showed no significant correlation between AHI and TMD. This is the first analysis to demonstrate a correlation between TMD and AHI while including individuals who didn't meet diagnostic criteria for OSA. However, the relationship between AHI and TMD was also significant in those with AHI>5. More data on these measures with larger sample sizes and a more equal representation of AHI values should be gathered to provide additional evidence for this relationship.

Support: Support: NIH R01-HL125893; CTSA UL1TR000128, R21HL140377

0571

REPOLARIZATION VARIABILITY PREDICTS CARDIOVASCULAR DEATH IN OBSTRUCTIVE SLEEP APNEA

Patel, S. I.¹ Zareba, W.² Couderc, J.² Xia, X.² LaFleur, B.³ Torabzadeh, E.³ Woosley, R.⁴ Parthasarathy, S.¹ ¹UAHS Center for Sleep and Circadian Sciences, University of Arizona, Tucson, AZ, ²Division of Cardiology and Heart Research, University of Rochester Medical Center, Rochester, NY, ³UAHS Center for Biomedical Informatics and Biostatistics, University of Arizona, Tucson, AZ, ⁴Division of Data Analytics and Decision Support, University of Arizona College of Medicine-Phoenix, Phoenix, AZ.

Introduction: Patients with untreated obstructive sleep apnea (OSA) have a 2-3—fold increased risk of cardiovascular mortality (CVD) compared with individuals without OSA. QTc prolongation and increased QT variability among OSA patients may contribute to this association.

Methods: Patients with OSA from the Sleep Heart Health study were identified based on polysomnography criteria and their continuous electrocardiograms (ECG) analyzed for QTc duration and QT variability. Both Fridericia's and Bazett's heart rate corrections were used to calculate QTc. QT variability was measured as standard deviation of QT intervals (SDQT) and normalized QT interval variance (QTVN) at 1- and 5-minute intervals and shortterm interval beat-to-beat QT variability (STVQT) was measured at 5-minute intervals. Lasso with elastic-net regularization was used as the variable/covariate selection method. Cox proportional hazards regression models were used to determine predictors of CVD. Results: Data from 365 patients with OSA were screened. Ninetyseven patients were excluded from analysis due to low quality ECG data (n=50) or extremely high (> ln (10)) variability in QT/QTc and/or QT variability (n=12). Fifty two percent of the sample was male with mean age 65 years (± 10). Fifty-six of these patients died of CVD. The mean (SD) QTc in the group that died was 411 (30) ms and 416 (34) ms compared to 406 (24) ms and 411 (25) ms using Fridericia (Cox LR p-value 0.055) and Bazett (p=0.090), respectively. Gender, age, race, diabetes, SDQT and STVQT were significant predictors for CVD. We fit models with the covariates and SDQT (at both 1 and 5 min) and STVQT as three models and demonstrate that both SDQT and STVQT are significantly associated

with CVD death (p-values of 0.0048, 0.0089, and 0.0113, respectively) and all models had high area under the curve (0.8095, 0.8085, and 0.8125, respectively).

Conclusion: In patients with OSA, QT variability was associated with CVD.

Support: American Academy of Sleep Medicine Foundation

0572

USING ANTHROPOMETRIC MEASURES TO SCREEN FOR OBSTRUCTIVE SLEEP APNEA IN THE SLEEP HEART HEALTH STUDY COHORT

Vana, K. D.¹ Silva, G. E.² Carreon, J. D.³ Quan, S. F.^{4,5} ¹Edson College of Nursing and Health Innovation, Arizona State University, Phoenix, AZ, ²College of Nursing, University of Arizona, Tucson, AZ, ³The Clovers' Leaves Limited Company, Tempe, AZ, ⁴Division of Sleep and Circadian Disorders, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, ⁵College of Medicine, University of Arizona, Tucson, AZ.

Introduction: Individuals at high risk for obstructive sleep apnea (OSA) may not access sleep clinics for reasons including immobility, transportation difficulties, or living in rural areas. An easy-to-administer OSA screening tool for different body types, independent of witnessed apneas or body mass index (BMI), is lacking to identify this group quickly. We compared the sensitivities (SNs), specificities (SPs), and receiving operator curves (ROCs) of the neck circumference/height ratio (NHR) and waist circumference/height ratio (WHR) in predicting moderate and severe OSA (apnea-hypopnea index [AHI] ≥15/hr) with the SN, SP, and ROC of the derived Stop-Bang Questionnaire (dSBQ), which was created from proxy variables from the Sleep Heart Health Study (SHHS).

Methods: Data from the SHHS baseline evaluation were used and included participants (N=5431) who completed polysomnograms and had neck and waist circumferences, height measurements, and the SHHS proxy variables. This data then was divided randomly into 1/3 for derivation and 2/3 for validation analyses.

Results: No statistical differences were seen for gender, age, or ethnicity between the derivation and validation samples. In the validation sample (n=3621), the NHR cut-point of 0.21 resulted in a SN of 91% and a SP of 26% for AHI ≥15/hr. The WHR cut-point of 0.51 resulted in a SN of 91% and a SP of 21% for AHI ≥15/hr. Comparing the validation NHR and the dSBQ ROC curves showed no significant difference (AUCs=0.69 and 0.70, respectively; p=0.22). However, the ROC curve for WHR was significantly lower than for the dSBQ (AUCs=0.63 and 0.70, respectively; p<0.0001). Comparing the derivation and validation ROCs showed no significant differences between NHR ROCs, p=0.81, or between WHR ROCs, p=0.67.

Conclusion: The NHR is a viable screening tool, independent of witnessed apneas and BMI, that can be used for different body types and is statistically comparable to the dSBQ.

Support: This work was supported by U01HL53938 and U01HL53938-07S (University of Arizona).

0573

SCREENING FOR OBSTRUCTIVE SLEEP APNEA AT HOME BASED ON DEEP LEARNING FEATURES DERIVED FROM RESPIRATION SOUNDS

Romero, H. E.¹ Ma, N.¹ Hill, E. A.² Brown, G. J.¹ ¹Department of Computer Science, University of Sheffield, Sheffield, UNITED KINGDOM, ²Sleep Research Unit, University of Edinburgh, Edinburgh, UNITED KINGDOM.

Introduction: Analysis of sleep breathing sounds has been employed to screen obstructive sleep apnea (OSA). However, most current methods rely on specialized equipment (e.g., tracheal microphones), require additional physiological data (e.g., oxygen saturation), are rule-based, or are trained on data collected in-lab, making them less suitable for home use. In this study, deep learning methods were leveraged to explore the hypothesis that sleep audio recordings collected via smartphones can be used alone to screen for OSA by exploiting the temporal pattern of respiration sounds. Methods: Adult participants with suspected sleep-disordered breathing of varying degrees of severity were recruited from the general population and from GP referrals to sleep clinic. Audio recordings were collected via smartphones during home sleep apnea testing (HSAT). HSAT data were scored by a registered polysomnographic technologist in accordance with current international guidelines (AASM V2.5, 2018) and used as reference. To exploit acoustic respiration temporal pattern, time interval histograms were computed for sequences of audio-words that were automatically learned from spectral features with a deep neural network. Means and standard deviations of the time intervals for each audio-word were employed by a Gaussian mixture model to classify 2-minute audio recording segments as either containing OSA events or not.

Results: Preliminary data from 4 valid nights' recordings obtained from 2 consented participants was analysed. 550 segments were used for training, with 180 segments used for evaluation. Audio recording demonstrated a sensitivity of 0.71 and specificity of 0.66 when compared with manually-scored HSAT.

Conclusion: Preliminary results suggest that an approach to OSA screening based on deep learning with inter-audio-word intervals to capture information about respiration temporal pattern may be a useful tool in diagnosis of OSA. Further model development is underway using data collected from up to 200 patients and full study data will be presented.

Support: The project is supported by an Innovate UK grant (project number 157358). HR is supported by a joint scholarship from Passion for Life Healthcare Ltd and University of Sheffield. LH acknowledges the financial support of NHS Research Scotland (NRS), through NHS Lothian.

0574

PREVALENCE AND CHARACTERISTICS OF RAPID EYE MOVEMENT OBSTRUCTIVE SLEEP APNOEA (REM OSA) IN A MULTI-ETHNIC OSA COHORT

Wong, H. Poh, Y. Mok, Y.

Changi General Hospital, Singapore, SINGAPORE.

Introduction: Recent studies have shown that REM OSA is associated with increased incidence of hypertension and insulin resistance. However, there is a lack of Asian data on REM OSA. Our study aimed to examine the prevalence and characteristics of REM OSA in a multi-ethnic OSA cohort.

Methods: This was a retrospective observational study of all patients who underwent an overnight diagnostic polysomnography at a Singapore tertiary hospital from 1st August 2017 to 31st August 2018. All patients with a diagnosis of OSA (Apnoea Hypopnea Index (AHI) \geq 5) were included in the study. REM OSA is defined as an overall AHI \geq 5, REM AHI/Non REM (NREM) AHI>2, NREM AHI<15 and at least 15 minutes of REM sleep.

Results: 457 OSA subjects were included in the analysis. 19% (87/457) had REM OSA. Univariate analysis showed that REM OSA was more prevalent among female OSA than male OSA [34/115 (29.6%) versus 53/342 (15.5%) respectively, p<0.001].

Compared to non REM OSA, REM OSA had milder OSA severity [mean AHI 12.74 \pm 4.71 versus 45.34 \pm 28.38, p<0.001] and lower prevalence of hypertension [21/87 (24.1%) versus 138/370(37.3%), p=0.02]. No differences were found between both groups for age (p=0.273), ethnicity (p=0.615), Body Mass Index (p=0.336), diabetes mellitus (p=0.245) and Epworth Sleepiness Scale (0.06). Gender and OSA severity differences between both groups remained statistically significant in multivariate analysis (higher prevalence of REM OSA in female, p=0.043 and milder disease severity in REM OSA, p=0.006).

Conclusion: REM OSA was common in our OSA cohort and had higher prevalence in female and milder disease severity compared to non REM OSA. However, we did not find an increased prevalence of hypertension or diabetes mellitus in REM OSA. Further population-based study on REM OSA is needed to understand this phenotype better.

Support: NIL

0575

SLEEP MONITORING WITH A SINGLE CHANNEL EEG RECORDER IN PATIENTS WITH PSYCHIATRIC DISORDERS

Miyata, S.¹ Iwamoto, K.¹ Banno, M.¹ Ito, Y.² Noda, A.³ Ozaki, N.¹ ¹Department of Psychiatry, Nagoya University Graduate School of Medicine, Nagoya, JAPAN, ²Department of Clinical Laboratory, Nagoya University Hospital, Nagoya, JAPAN, ³Department of Biomedical Sciences, Chubu University Graduate School of Life and Health Sciences, Kasugai, JAPAN.

Introduction: The gold standard of sleep measurement has been laboratory polysomnography (PSG). However, electrodes and cables can cause discomfort, and exposure to an unfamiliar environment can cause the "first-night effect." Difficulty falling asleep or maintaining sleep, poor sleep quality, and nightmares are some of the key clinical symptoms observed among individuals with psychiatric disorders. Those suffering from sleep disorders often present with symptoms of discontent with regard to sleep quality, timing, and quantity, and these symptoms have an adverse impact on function and quality of life. A minimally invasive technique would be preferable in patients with psychiatric disorders, who tend to be sensitive to environmental change. Accordingly, we evaluated the performance of a single-channel electroencephalography (EEG)-based sleep monitoring system in patients with psychiatric disorders.

Methods: Fifty-nine patients undergoing PSG were enrolled in this study. Single-channel EEG sleep monitoring was performed simultaneously with PSG. PSG and the EEG recordings were used to evaluate sleep parameters, such as total sleep time (TST), sleep efficiency, rapid eye movement (REM) sleep, light sleep (stages N1 and N2), and deep sleep (stage N3). Correlation analysis was used to evaluate the agreement on sleep parameters and attributing factors to the inaccuracies of the single-channel EEG recording.

Results: TST, sleep efficiency, REM sleep duration, and non-REM sleep duration of the single-channel EEG-based sleep monitoring showed a significant correlation with those of PSG. Lower sleep efficiency, a decrease in REM sleep, and increases in waking after sleep onset, arousal index, and apnea/hypopnea index were associated with the difference of sleep parameters between the two methods.

Conclusion: Among patients with psychiatric disorders who are sensitive to environmental change single-channel EEG sleep monitoring would be a useful technique to objectively evaluate sleep quality.

Support: Collaboration study with The KAITEKI Institute, Inc.

0576

VARIATION IN NIGHT TO NIGHT HOME SLEEP TESTING Rosenberg, C.

VHA Cleveland, Louis B Stokes VHA, Cleveland, OH.

Introduction: Home sleep testing (HST) is becoming common in the evaluation of Obstructive Sleep Apnea (OSA). Studies confirmed good HST AHI correlations from different nights in a single patient. The following reviewed AHI and additional measures from HST's. (Alice Night One))

Methods: We collected data from 20 patients from two consecutive nights of HST's. 5 F 15 M, means AGE 49 (sd 14) and BMI 36 (sd 8). Both studies had over 4 hours of good sleep and acceptable data. Measures include abs(Night 1- Night 2) of AHI (Diff.AHI), of mean EKG (Diff.EKG) mean time SaO2 less than 90% (Diff. SaO2).

Results: These results reproduced the strong correlation of AHI, Time SaO2 less than 90 %: and mean EKG between two nights, .96, .72, .87 respectively. There was a strong correlation between Diff.AHI and Diff.SaO2, .63 (p .003). There were weaker correlations between AHI and Time SaO2 less than 90% on Night 1, .67 and Night 2, .75. Linear regression: Diff.AHI on Age (p=.2), BMI (p = .9), and Diff.EKG (p=.4).

Conclusion: These results again validate the small degree of AHI variation in night to night HST. They confirm a small degree of variation in the mean EKG and Time SaO2 less than 90%. There is a high correlation between AHI and time SaO2 less than 90% as these variables are dependent and the fall in SaO2 is used to define an event, especially on the HST. The BMI did not explain variation in AHI, there is a low correlation between AHI and BMI. Age could be a factor in AHI variation; yet, this is highly speculative with an N = 20. The correlations between AHI and Time SaO2 less than 90% are likely to be due to the relative health of the subjects and small number of subjects. One night of good, greater than 4 hours HST may be sufficient. This study did not evaluate success in meeting these parameters with a single night of testing.

Support: Louis B Stokes VHA, Cleveland, OH

0577

CLUSTER ANALYSIS FOR THE ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA PHENOTYPES: A POPULATION-BASED LONGITUDINAL STUDY

Tempaku, P. F. Silva, L. O. Guimaraes, T. M. Vidigal, T. A. D'Almeida, V. Andersen, M. L. Bittencourt, L. Tufik, S. Universidade Federal de Sao Paulo, Sao Paulo, BRAZIL.

Introduction: The identification of subgroups of obstructive sleep apnea (OSA) is critical to understand disease causality and ultimately develop optimal care strategies customized for each subgroup. In this sense, we aimed to perform a cluster analysis to identify subgroups of individuals with OSA based on clinical parameters. Furthermore, we aimed to analyze whether subgroups remain after 8 years.

Methods: We used data derived from the Sao Paulo Epidemiologic Sleep Study (EPISONO) cohort, which was followed over 8 years. All individuals underwent polysomnography, answered questionnaires and had their blood collected for biochemical exams. OSA was defined according to an AHI equal or greater than 15 events per hour. Cluster analysis was performed using latent class analysis (LCA).

Results: Of the 1,042 individuals in the EPISONO baseline cohort, 68.3% accepted to participate in the follow-up study (n=712). We were able to replicate the OSA 3-cluster solution observed in previous studies: disturbed sleep, minimally symptomatic and excessively sleepy in both baseline (35.5%, 45.4% and 19.1%, respectively) and follow-up studies (41.9%, 43.4% and 14.8%, respectively). 44.8% of the participants migrated clusters between the two evaluations and the factor associated with this was a greater delta-AHI (B=-0.033, df=1, p=0.003). The optimal cluster solution for our sample based on Bayesian information criterion (BIC) was 2 clusters for baseline (disturbed sleep and excessively sleepy) and 3 clusters for follow-up (disturbed sleep, minimally symptomatic and excessively sleepy).

Conclusion: The results found replicate and confirm previously identified clinical clusters in OSA even in a longitudinal analysis. Support: This work was supported by grants from AFIP, FAPESP and CAPES.

0578

INCIDENT HYPERTENSION RATES IN OSA IDENTIFIED USING AMERICAN ACADEMY OF SLEEP MEDICINE (AASM) HYPOPNEA CRITERIA, BUT MISCLASSIFIED BY **MEDICARE (CMS) HYPOPNEA DEFINITION**

Budhiraja, R.¹ Javaheri, S.¹ Berry, R. B.² Parthasarathy, S.³ Ouan, S. $F.^1$

¹Harvard Medical School, Boston, MA, ²University of Florida, Gainesville, FL, ³University of Arizona, Tucson, AZ.

Introduction: The impact of not treating OSA identified using AASM standards (hypopneas scored using a minimum 3% O₂ desaturation or arousal), but misclassified by CMS standards (hypopneas scored only if minimum 4% O₂ desaturation) remains unclear. This analysis determined the ~5 year incident hypertension rates using the new 2018 ACC/AHA blood pressure (BP) guidelines in these individuals.

Methods: Data were analyzed from all Sleep Heart Health Study exam 2 study participants (N=1219) who were normotensive (BP≤120/80) at exam 1. The apnea hypopnea index (AHI) at exam 1 was classified into 4 categories of OSA severity: <5, $5 \le 15$, $15 \le 30$ and ≥30/hour using both the AASM or CMS definitions. Three definitions of hypertension were used: Elevated BP (>120/80), Stage 1 (>130/80) and Stage 2 (>140/90) to determine incidence rates at exam 2.

Results: Five year follow-up data were available for 476 participants classified as having OSA (AHI ≥5) by AASM criteria, but not by CMS standards at exam 1. Incident hypertension rates in these misclassified participants for ACC/AHA defined BP categories were 15% (Elevated BP), 15% (Stage 1) and 6%(Stage 2). 4% of normotensive participants used hypertensive medications. Overall incidence rate of at least an elevated BP was 40% (191/476) in those with OSA defined using AASM, but not by CMS criteria and 17% (191/1219) of the overall population at risk. In comparison to those with incident hypertension and OSA identified by CMS standards, BMI (27.7 vs 30.1 kg/ m^2 , p<.001) and % men were lower (45 vs 58%, p=.012), but age and race were not different.

Conclusion: Use of the CMS hypopnea definition as a component of the AHI resulted in the failure to identify a significant number of individuals with OSA who eventually developed hypertension and could have benefited from earlier diagnosis and treatment. Support: HL53938

NON INTRUSIVE AND UNATTENDED SLEEP ANALYZER EFFECTIVELY SCREENS PATIENTS SUSPECTED OF SLEEP APNEA: A COMPARISON WITH POLYSOMNOGRAPHY

Edouard, P.¹ Campo, D.¹ Bartet, P.¹ Marais, L.¹ Petitjean, M.² Roisman, G.² Bruyneel, M.³ Escourrou, P.²

¹WITHINGS, issy-les-moulineaux, FRANCE, ²Hôpital Antoine-Béclère, Clamart, FRANCE, ³Hôpital Saint-Pierre, Bruxelles, BELGIUM.

Introduction: Sleep Apnea Syndrome (SAS) is largely underdiagnosed due to the cost and availability of Polysomnography (PSG). We aimed at evaluating the diagnosis of SAS with the WITHINGS Sleep Apnea Detector (SAD), a non-intrusive pressure and sound sensor placed under the mattress.

Methods: 118 patients (67 F, 49 years, BMI 33kg/m²) suspected of SAS had an in-laboratory PSG together with Sleep Apnea Detector. From the pressure signal, Sleep Apnea Detector derives respiratory and cardiac signals and movements. From the microphone, snoring and snorting are detected. These features are used to detect sleep periods with a Random Forest classifier and apnea and hypopnea events with a Convolutional Neural Network. The Total Sleep Time (TST) and Apnea Hypopnea Index (AHI) deduced (TST_{sad}, AHI_{sad}) are compared with the PSG results scored according to AASM rules (TST_{psg}, AHI_{psg}). AHI and TST were compared using bias and Mean Absolute Error (MAE). Sensitivity, specificity, likelihood ratios (LR) and AUROC were calculated for AHI thresholds of 15 and 30/hr.

Results: The average (SD) TSTpsg was 367 (61) minutes. Sleep Apnea Detector overestimated TST by 25 minutes, 7.0% of the average duration in the sample. The precision is acceptable, with a MAE=53 minutes. Average AHI_{psg} was 32.5 (30.1) and AHI_{sad} 32.8 (29.9). The bias was 0.3 (95% CI [-2.7, 3.3]), MAE=10.3. The sensitivity (Se₁₅) and specificity (Sp₁₅) and their 95% confidence intervals were Se₁₅=88.0% [79.0, 94.1] and Sp₁₅=88.6% [73.3, 96.8]. Positive and negative LR were respectively LR⁺₁₅=7.70 and LR⁻₁₅=0.136. AUROC₁₅=0.926. At the 30 threshold, Se₃₀=86.0% [73.3, 94.2] and Sp₃₀=91.2% [81.8, 96.7]. Positive and negative LR were LR⁺₃₀=9.75 and LR⁻₃₀=0.153. AUROC₃₀=0.954.

Conclusion: Sleep Apnea Detector has excellent sensitivity and specificity, low bias and good precision. Thus it can be used as an unattended SAS screening device in patients likely to suffer from SAS. **Support:** WITHINGS

0580

HABITUAL SLEEP PATTERN, ANXIETY AND DEPRESSION ARE PREDICTIVE OF EXCESSIVE DAYTIME SLEEPINESS IN A LARGE-SCALE CLINICAL SAMPLES OF OBSTRUCTIVE SLEEP APNEA

Chang, S.¹ Huang, W.² Liu, Y.³ Lee, P.⁴

¹Department of Neurology, Taipei City Hospital, Songde branch, Taipei, TAIWAN, ²Department of Computer Science and Information Engineering, National Taiwan University, Taipei, TAIWAN, ³Department of Multimedia Technology Development, MediaTek Inc., Hsinchu, TAIWAN, ⁴Center of Sleep Disorder, National Taiwan University Hospital, Taipei, TAIWAN.

Introduction: Excessive daytime sleepiness (EDS) is a common symptom that patients with obstructive sleep apnea (OSA) seek

medical attention for. Prevalence ranged from 20% to 60%. Previous studies reported factors associated with EDS included age, body mass index (BMI), depression, and OSA severity. In most studies, the sample size was small, participants having specific co-morbidities, and the definitions of EDS was heterogeneous. Moreover, the association between anxiety, depression, habitual sleep pattern, and EDS has not been widely studied. Therefore, the present study aims to investigate the prevalence of EDS and associated factors, especially anxiety, depression, and habitual sleep pattern, in a large-scale clinical sample.

Methods: Data was prospectively collected from 8,081 adult patients who underwent initial overnight polysomnography (PSG) for the first time were referred from 2009 to 2016. Patients with total recording time less than 240 minutes and missing data were excluded. Data collected include demographics, anthropometrics, co-morbidities, and self-reported habitual sleep patterns. Subjective sleepiness was assessed by the Epworth Sleepiness Scale (ESS) with EDS defined as ESS ≥10. Anxiety and depression were assessed by the Hospital Anxiety and Depression Scale (HADS). The stepwise forward Logistic regression was used to identified predictors for EDS.

Results: In 5,780 (82.6%) patients with OSA (apnea-hypopnea index, AHI \geq 5/h), mean age was 63.9 \pm 0.2 yr, BMI was 27.7 \pm 0.1, and ESS was 10.4 \pm 0.1 and 80.5% were male. Prevalence of EDS in all OSA patients was 55.1% where the patients with severe OSA had higher prevalence (59.8%) than that in mild (49.5%) and moderate OSA (51.8%). Anxiety (OR: 2.036, 95% CI:1.153-1.502), depression (OR: 1.159, 95% CI:1.01-1.33), and short sleep (<6hr/night) (OR:1.316, 95% CI:1.32,1.70) were top three risk factors for EDS. Other risk factors for EDS included AHI, arousal index, % total sleep time with SpO2<90%, %REM, smoking while hypnotic use and long sleep (\geq 8hr/night) were associated with lower risk.

Conclusion: Anxiety, depression, short sleep are predictive and OSA severity are predictive of EDS while long sleep was associated with lower risk.

Support: National Taiwan University (NTU-EPR-104R8951-1; 105R8951-1; 106R880301), Center of electronics technology integration (NTU-107L900502, 108L900502) by the Ministry of Education in Taiwan and MediaTek Inc (201802034RIND).

0581

CHARACTERISTICS OF US WOMEN VETERANS WITH SLEEP APNEA: RESULTS OF A NATIONAL SURVEY OF VA HEALTHCARE USERS

Zhu, R.¹ Carlson, G.² Kelly, M.³ Song, Y.^{4,5} Fung, C. H.^{4,6} Mitchell, M. N.⁷ Josephson, K. R.⁷ Zeidler, M. R.^{4,6} Badr, M. S.^{8,9} Alessi, C. A.^{4,6} Washington, D. L.¹ Yano, E. M.² Martin, J. L.^{4,6} ¹Department of Medicine, David Geffen School of Medicine, University of California, Los Angeles, Los Angeles, CA, ²VA Health Services Research & Development Center for the Study of Healthcare Innovation, Implementation, and Policy, VA Greater Los Angeles Healthcare System, Los Angeles, CA, ³VA Greater Los Angeles Healthcare System, Los Angeles, CA, ⁴Geriatric Research Education and Clinical Center, VA Greater Los Angeles Healthcare System, North Hills, CA, 5School of Nursing at the University of California, Los Angeles, Los Angeles, CA, ⁶Department of Medicine, David Geffen School of Medicine at the University of California, Los Angeles, Los Angeles, CA, ⁷Geriatric Research Education and Clinical Center, VA Greater Los Angeles Healthcare System, Los Angeles, CA, 8Department of Internal Medicine, Wayne State University School of Medicine, Detroit, MI, ⁹John D. DIngell VA Medical Center, Detroit, MI.

SLEEP, Volume 43, Abstract Supplement, 2020

Introduction: Sleep apnea (SA) is the most commonly diagnosed sleep disorder among patients in the US Veterans Administration (VA). The dramatic rise in women receiving VA care makes it essential to understand the presentation and treatment of SA in women Veterans. We performed a nationwide survey about sleep among US women Veterans and compared characteristics of respondents with and without a self-reported history of SA diagnosis and treatment.

Methods: A survey was mailed to a random sample of 4000 women VA healthcare users. The survey included demographics, Insomnia Severity Index (ISI), Patient Health Questionnaire-4 (PHQ-4 depression/anxiety), Primary Care-Post-Traumatic Stress Disorder (PC-PTSD), RLS symptom presence, SA symptoms (snore loudly, observed breathing pauses), diagnosis of SA, and use of PAP therapy (APAP, BPAP, CPAP). We compared women with and without SA, and (among those with SA) women who did and did not use PAP, using Chi-square and t-tests.

Results: 1,498 completed surveys were returned (mean age 51.6 years, range 18-105 years, 62% non-Hispanic White). 200 respondents (13.4%) reported diagnosed SA. Women with SA were older (p<.001), likely to be employed (p=.013), more likely to snore loudly (p<.001) and to have breathing pauses while asleep (p<.001). They also had higher ISI (p<.001), were more like to report RLS (p<.001) nightmares (p=.027), and had higher PHQ-4 (p<.001) and PC-PTSD (p<.001) scores. Among women with SA, 130 (65%) used PAP. Loud snorers (p<.001) and those with observed breathing pauses were more likely to use PAP (p<.001).

Conclusion: One in 7 women who receive VA care report diagnosed SA. Women with SA had more mental health symptoms and comorbid sleep problems. Most reported using PAP therapy, although the amount of use is unknown. Those with SA symptoms were more likely to use PAP. Future work is needed to understand barriers to diagnosis and treatment of SA among women Veterans. **Support:** Funding: VA Quality Enhancement Research Initiative RRP12-189 (Martin); NIH/NHLBI K24 HL143055 (Martin).

0582

EXAMINING OSA SCREENING AND TREATMENT FOR INDIVIDUALS ON A PTSD AND ALCOHOL DISORDER RESIDENTIAL TREATMENT UNIT

Colvonen, P. J.¹ Rivera, G.² Haller, M.² Norman, S.²

¹University of California, San Diego, San Diego, CA, ²San Diego VA Hospital, San Diego, CA.

Introduction: Obstructive sleep apnea (OSA) is highly co-occurring with both alcohol use disorder (AUD) and posttraumatic stress disorder (PTSD) and has been shown to interfere with both PTSD and AUD outcomes. However, OSA often goes undiagnosed and untreated in residential treatment facilities. Our study aimed to assess the feasibility of incorporating OSA screening and treatment onto a substance abuse residential rehabilitation treatment program (SARRTP). Further, we examine the relationship between adherence rates of CPAP on PTSD outcomes.

Methods: Participants were 35 consecutive veterans admitted to the SARRTP PTSD track who consented to screening. Veterans were on the unit for 4-6 weeks. OSA was diagnosed using Nox T3 recorders, a Type-3 portable OSA screener (using Apnea Hypopnea Index \geq 5). Insomnia Severity Index and PTSD checklist were given at pre- and post-treatment.

Results: 64.7% of Veterans screened positive for OSA. 11.8% were previously diagnosed with OSA, but did not use a CPAP machine; 17.6% were previously diagnosed and were using a CPAP

machine; and 35.3% were newly diagnosed with OSA. Individuals with untreated OSA had significantly more days drinking in the last 30 days (M = 21.17 days, SD = 11.41) compared to no OSA/ Treated OSA group (M = 8.82 days, SD = 10.92). There was no difference in change in PCL scores from baseline to post-treatment by the no-OSA/high compliance group and the low compliance group. **Conclusion:** Taken together, OSA screening on the unit was accepted by the participants, feasible, and effective in diagnosing OSA. OSA screening and treatment should be considered as necessary on SUD and PTSD units. We did not find that OSA adherence predicted change in PTSD score, this is most likely due to veterans receiving their CPAP late into their stay on the unit. Future studies will need to examine OSA treatment on long term treatment outcomes.

Support: Support: This work is supported by UCSD Academic Senate Grant and a Veterans Affairs RR&D CDA (11K2Rx002120-01) to Peter Colvonen.

0583

OBESITY-ASSOCIATED SLEEP HYPOVENTILATION SYNDROME AND ADVERSE POST-OPERATIVE BARIATRIC SURGERY OUTCOMES

Chindamporn, P.¹ Bena, J.² Wang, L.² Zajichek, A.² Milinovich, A.² Kaw, R.³ Kashyap, S.⁴ Cetin, D.⁵ Aminian, A.⁶ Kempke, N.¹ Foldvary-Schaefer, N.¹ Aboussouan, L. S.¹ Mehra, R.¹ ¹Sleep Disorders Center, Neurological Institute, Cleveland Clinic, Cleveland, OH, ²Department of Quantitative Health Sciences, Cleveland Clinic, Cleveland, OH, ³Medicine Institute, Cleveland Clinic, Cleveland, OH, ⁴Endocrinology, Cleveland Clinic, Cleveland, OH, ⁵Obesity Medicine Specialist, Bariatric Metabolic Institute, Cleveland Clinic, Cleveland, OH, ⁶Bariatric and Metabolic Institute, Department of General Surgery, Cleveland Clinic, Cleveland, OH.

Introduction: Although obesity hypoventilation syndrome (OHS) is associated with right ventricular dysfunction and increased mortality, its contribution to post-bariatric surgery risk remains unclear due to non-systematic OHS assessments. We hypothesize that patients with obesity-associated sleep hypoventilation (OASH) have increased adverse post-bariatric surgery outcomes than those without.

Methods: Patients undergoing polysomnography (PSG) prior to bariatric surgery at the Cleveland Clinic from 2011-2018 were retrospectively examined. OASH was defined by body mass index (BMI) \geq 30kg/m² and either PSG-based end-tidal CO2 \geq 45mmHg or serum bicarbonate \geq 27mEq/L. The following were considered individually and as a composite outcome: ICU stay, re-intubation, tracheostomy, discharge disposition or 30-day readmission. Allcause mortality was also examined. Outcomes were compared using two-sample t-test or Wilcoxon rank sum test and Chi-square or Fisher exact test. A multivariable logistic regression model included age, sex, BMI, apnea hypopnea index(AHI) and diabetes to examine OAHS and the composite outcome. All-cause mortality was compared using Kaplan-Meier estimation and hazard ratios from Cox proportional hazards models. SAS software (version 9.4) was used with overall significance level of 0.05.

Results: The sample comprised 1665 patients: age 45.2 ± 12 years, 20.4% male, BMI= 48.7 ± 9 kg/m², and 63.6% Caucasian. OASH prevalence was 68.5%. OAHS patients were older and more likely to be male with higher BMI, AHI and HbA1c. Although some individual outcomes were higher in OASH vs. non-OASH, findings were not statistically significant: re-intubation (1.5%vs.1.3%, p=0.81) and 30-day readmission

(13.8% vs.11.3%, p=0.16). The composite outcome remained significantly associated with OAHS in the multivariable model: OR=1.36, 95%CI:1.005,1.845. Mortality was 2% in OASH and not significantly higher than non-OAHS (HR=1.39, 95%CI:0.56,3.42).

Conclusion: In this largest sample to date of systematically phenotyped OASH in patients undergoing bariatric surgery, we identify increased post-operative morbidity in those with OASH. Further study is needed to identify whether peri-operative treatment of OASH improves surgical outcomes. **Support:**

0584

DETECTING SLEEP DISORDERED BREATHING USING SUB-TERAHERTZ RADIO-FREQUENCY MICRO-RADAR

Korotun, M.¹ Weizman, L.² Drori, A.² Zaccaria, J.² Goldstein, T.¹ Litman, I.² Hahn, S.¹ Greenberg, H.¹

¹Northwell Health, New Hyde Park, NY, ²Neteera Technologies Ltd., Jerusalem, ISRAEL, ³Northwell Health, New Hyde Park, NY.

Introduction: New sensor technologies are entering sleep testing at a rapid pace; NeteeraTM developed a novel sensor and algorithm for sleep apnea detection utilizing a contact-free, radar-based sensor system. The system utilizes a high-frequency, low-power, directional micro-radar which operates at ~120GHz and a sampling rate of 2500Hz as well as algorithms which are able to detect both pulse and respiratory activity of subjects during sleep.

Methods: Adult subjects undergoing diagnostic PSG for clinical purposes were simultaneously assessed with the novel micro-radar system with sensors under the mattress. Disordered breathing events (DBEs) were scored from the PSG using AASM scoring guidelines and were compared with those detected by the micro-radar sensor. Test data were grouped into three sets: 1. Single under mattress sensor; 2. Two under mattress sensors on each side of the bed (to improve signal capture); 3. After software optimization. The micro-radar sensor detected DBEs but software to describe the type of DBEs (obstructive apnea/central apnea/hypopnea) is still under development. Detection rate of DBEs was compared between the two methodologies and the development sets.

Results: n=22 (12 F, 10 M), Age= 50.8 ± 12.4 years, BMI= 35.32 ± 7.37 kg/m². Diagnostic PSG AHI: 19.7 ±29.4 /hr, T₉₀= 15.8 ± 25.7 %. Percent DBEs missed by the micro-radar sensor: 1st set= 14.6 ± 10.6 %; 2nd set= 9.4 ± 8.3 %; 3rd set= 1.2 ± 2.6 %. Number of DBEs assessed for each set was 646, 1144, 125 events, respectively. With each successive set, the detection rate improved.

Conclusion: A novel micro-radar, non-contact sensor technology can be used to detect DBEs during sleep. Detection rate improved with utilization of two sensors per bed and software optimization. Future software development is expected to improve detection rate and facilitate breathing event classification into obstructive apneas/ central apneas/hypopneas.

Support: None.

0585

C-REACTIVE PROTEIN IMPROVES THE ABILITY TO DETECT CARDIOMETABOLIC RISK IN MILD-TO-MODERATE SLEEP APNEA

Vgontzas, A. N.¹ Puzino, K.¹ Fernandez-Mendoza, J.¹ Criley, C.¹ He, F.¹ Krishnamurthy, V. B.¹ Basta, M.³ Bixler, E. O.¹ ¹Penn State College of Medicine, Hershey, PA, ²Penn State College of Medicine, Hershey, PA, ³University of Crete, Heraklion, GREECE. **Introduction:** Mild-to-moderate obstructive sleep apnea (OSA) affects 15-40% of the adult general population. However, it remains unclear when and how best to treat mild-to-moderate OSA. It has been shown that mild-to-moderate OSA in general random samples is associated with incident hypertension. The aim of this study was to compare the relative utility of apnea/hypopnea index (AHI) versus a biomarker of inflammation, C-reactive protein (CRP), in identifying the presence and severity of hypertension and insulin resistance (IR).

Methods: A clinical sample of 148 adults (53.79 ± 12.45) with mild-to-moderate OSA (AHI between 5 and 29 events per hour) underwent 8-hour polysomnography, a clinical history and physical examination, including measures of blood pressure, body mass index (BMI), fasting blood glucose, insulin and CRP plasma levels. Hypertension was defined by previous diagnosis, past or present treatment, or blood pressure \geq 140/90. IR was defined by homeostatic model assessment. Individuals with diabetes and/or on diabetes medication were excluded from analyses with IR. All analyses were conducted controlling for age, gender and BMI.

Results: CRP levels (OR=2.62, 95% CI=1.35-5.04, p=0.004), age (OR=1.75, 95% CI=1.11-2.75, p=0.016), and BMI (OR=2.74, 95% CI=1.20-6.26, p=0.017) were independently associated with greater odds for hypertension, whereas AHI (OR=1.33, 95% CI=0.61-2.92, p=0.477) was not. Additionally, CRP levels (β =0.21; p=0.04) and BMI (β =0.24; p=0.02) were independently associated with higher IR, while AHI (β =-0.03; p=0.75) was not. There was a trend for this association to be stronger in non-obese patients.

Conclusion: These preliminary findings suggest that including a measure of inflammation improves the ability for clinicians to detect cases of mild-to-moderate OSA with true cardiometabolic risk. CRP may be a simple, easy-to-use biomarker that can improve prognosis assessment and clarify which treatment option is best for patients with mild-to-moderate OSA.

Support: Department of Psychiatry, Penn State College of Medicine

0586

ADVANCED TREE MODELS TO PREDICT MODERATE-TO-SEVERE OBSTRUCTIVE SLEEP APNEA

KIM, S. Yang, K.

Sleep Disorders Center, Soonchunhyang Univ. Cheonan Hospital, Cheonan, KOREA, REPUBLIC OF.

Introduction: The aim of this study was to develop a predicting model for the moderate-to-severe obstructive sleep apnea (OSA) by using advanced tree models.

Methods: We retrospectively investigated the medical records of patients who undertaken overnight polysomnography (PSG) at our sleep disorders center. We divided the data to a training set (70%) and a test set (30%), randomly. We made a random forest and a XGBoost model to predict the moderate-to-severe OSA (apnea hyponea index [AHI] \geq 15/h) by using the training set, and then applied each models to the test set. To compare the fitness of the models, we used an accuracy, and an area under curve (AUC).

Results: Finally, 1,426 patients (AHI \leq 5:AHI \geq 15= 464:962) were enrolled. The random forest model showed an accuracy of 0.79, and AUC of 0.82. In the random forest model, the sleep apnea scale of the sleep disorders questionnaire (SA-SDQ), age, neck circumference, male sex, body mass index (BMI), hypertension, and hyperlipidemia appeared in order of a variance importance. The XGBoost model showed an accuracy of 0.75 and AUC of 0.79.

Conclusion: The random forest model to predict moderate-tosevere OSA showed better performance compared to the XGBoost model. The further study for validation is required. **Support:** None

0587

CONVENIENCE VS ACCURACY: NEGATIVE PREDICTABILITY OF THE HOME SLEEP TEST IN CLINICAL PRACTICE

Morse, C. D.¹ Meissner, S.² Kodali, L.² ¹Cayuga Medical Center, Ithaca, NY, ²Pulmonary and Sleep Services of Cayuga Medical Associates, Ithaca, NY.

Introduction: Sleep apnea is a serious disorder associated with numerous health conditions. In clinical practice, providers order screening home sleep testing (HST) for obstructive sleep apnea (OSA); however, there is limited research about the negative predictive value (NPV) and false negative rate of this test. Providers may not understand HST limitations; therefore, what is the NPV and false negative rate in clinical practice?

Methods: A retrospective study of non-diagnostic HST is conducted in a Northeastern US rural community sleep clinic. The study population includes adult patients \geq 18 years old who underwent HST from 2016-2019. The non-diagnostic HST result is compared to the gold standard, the patient's nocturnal polysomnogram (NPSG). The results provide the NPV (true negative/total) and false negative (true positive/total) for the non-diagnostic HST.

Results: We identified 211 potential patients with a mean age of 43 years, of which 67% were female. Of those, 85% (n=179) underwent NPSG, with the others declining/delaying testing or lost to follow up. The non-diagnostic HST showed 15.6% NPV for no apnea using AHI<5 and 8.4% NPV using respiratory disturbance index (tRDI)<5. The false negative rate for AHI/tRDI was 84.4% and 91.6%, respectively. The AHI for positive tests ranged from 5-89 per hour (mean AHI 14.9/tRDI 16/hour), of which OSA was identified with an elevated AHI (\geq 5) ranging from 54.2% mild, 21.8% moderate, and 8.4% severe.

Conclusion: The high false negative rate of the HST is alarming. Some providers and patients may forgo NPSG after non-diagnostic HST due to a lack of understanding for the HST's limitations. Knowing that the non-diagnostic HST is a very poor predictor of no sleep apnea will help providers advise patients appropriately for the necessity of the NPSG. The subsequent NPSG provides an accurate diagnosis and, therefore, an informed decision about pursuing or eschewing sleep apnea treatment. **Support:** none

0588

RING PULSE OXIMETER FOR SCREENING OF MODERATE TO SEVERE OSA: A PILOT STUDY

Xue, J. Zhao, R. Li, J. Zhao, L. Zhou, B. Dong, X. Han, F. Peking University Peoples' Hospital, Beijing, CHINA.

Introduction: To evaluate the utility of the ring pulse oximeter for screening of OSA in adults.

Methods: 87 adults were monitored by a ring pulse oximeter and PSG simultaneously during a nocturnal in-lab sleep testing. 3% oxygen desaturation index (ODI₃); Mean oxygen saturation(MSpO₂), Saturation impair time below 90% (SIT90) derived from an automated algorithm of the ring pulse oximeter. Meanwhile, the parameters of PSG were scored manually according to the AASM Manual. Correlation and receiver operator characteristic curve analysis were used to measure the accuracy of ring pulse oximeter and its diagnostic value for moderate to severe OSA (AHI≥15).

Results: Among the 87 participants, 18 cases were AHI<5, 17 cases were diagnosed with mild OSA (AHI:5-14.9), 25 cases were diagnosed with moderate OSA (AHI:15-29.9) and 27 cases were diagnosed with severe OSA (AHI≥30). There was no significant difference between PSG and ring pulse oximeter in regard to ODI. $(23.4\pm23.5 \text{ vs } 24.7\pm21.7)$, and SIT90 (1.54%, range 0.14%-8.99%)vs. 3.20%, range 0.60%, 12.30%) (P>0.05], Further analysis indicated that two parameters from the oximeter correlated well with that derived from PSG (r=0.889, 0.567, respectively, both p<0.05). Although MSpO₂ correlated significantly (r=0.448, P<0.05), the difference was remarkable [95.9%, range 94.0% to 97.0% vs. 94.5%, range 93.3% to 95.7%, p<0.05]. Bland-Altman plots showed that the agreement of these three parameters was within the clinical acceptance range. The ROC curve showed that the sensitivity and specificity of the ring pulse oximeter when the oximeter derived $ODI_{2} \ge 12.5$ in the diagnosis of moderate to severe OSA were 82.7% and 74.3%, respectively.

Conclusion: The pilot study indicated that ring pulse oximeter can detect oxygen desaturation events accurately, therefore to be used as a screening tool for moderate to severe OSA.

Support: The study was supported by the National Natural Science Foundation of China (No. 81420108002 and NO. 81570083).

0589

ARRHYTHMIA DETECTION IN OBSTRUCTIVE SLEEP APNEA (ADIOS)

*Geil, E. S.*¹ *Ramos, A. R.*¹ *Abreu, A. R.*¹ *Lambrasko, L. K.*² *Dib, S. I.*¹ *Wallace, D. M.*³ *Junco, B.*⁴ *Torre, B. C.*⁴ *Chediak, A. D.*¹ *Chaturvedi, S.*⁵

¹UHealth Sleep Medicine Program, Miller School of Medicine, Miami, FL, ²Department of Medicine, Miller School of Medicine, Miami, FL, ³Bruce W. Carter VA Medical Center, Miami, FL, ⁴Department of Neurology, Miller School of Medicine, Miami, FL, ⁵Department of Neurology, University of Maryland, Baltimore, MD.

Introduction: Obstructive sleep apnea (OSA) is a recognized risk factor for ischemic stroke; however, there is a paucity of studies devoted to modifying stroke risk factors in patients with OSA. We aimed to evaluate the prevalence and treatment of stroke risk factors in newly diagnosed OSA patients.

Methods: We evaluated consecutive patients with an OSA diagnosis made within 12 months and CHADS² score of >2, consistent with high risk for atrial fibrillation. The patients completed polysomnography, sleep questionnaires, and systematic assessments for demographic variables, vascular risk factors, and medication use. Participants also completed up to four weeks of ambulatory cardiac monitoring. A six-month follow-up visit screened for new hospitalizations associated to vascular events and use of new anticoagulants or antiplatelet therapy.

Results: The sample consisted of 87 patients, mean age 59 ± 8 years, 53% women, and 69% of Hispanic/Latino background. The mean BMI was 35 ± 9 . Hypertension was seen in 57% and diabetes mellitus in 33% of the sample. The mean apnea-hypopnea index was 41 ± 27 events/hour. Atrial fibrillation was detected in 3% of the sample through prolonged monitoring. At six-month follow-up, 9% of the sample was hospitalized due to stroke, transient ischemic attack, or coronary artery disease, while 13% reported use of anticoagulants and 38% antiplatelet therapy.

Conclusion: In this high risk sample of OSA patients, there was a high prevalence of cerebrovascular events and use of medical treatment for secondary stroke prevention. Future studies evaluating the treatment of vascular risk factors in OSA can provide strategies to minimize stroke occurrence. **Support:** Boehringer Ingelheim

0590

RELIABILITY OF SIMPLE SLEEP EVALUATION DEVICE AT SPLIT-NIGHT POLYSOMNOGRAPHY

Adachi, T.¹ Koba, S.² Hanyu, A.¹ Kato, M.¹ Morita, M.¹ Kawamoto, T.¹ Ida, H.¹ Watanabe, Y.¹ Shinke, T.² ¹Sleep Medicine Center, Showa University East Hospital, Tokyo,

JAPAN, ²Department of Medicine, Division of Cardiology, Showa University School of Medicine, Tokyo, JAPAN.

Introduction: Watch-PAT is a sleep evaluation device that measures the peripheral blood volume continuously with a probe attached to a fingertip and does not use an electroencephalogram or a nasal cannula. There has been no report on the usefulness of watch-PAT to determine the apnea diagnosis and continuous positive airway pressure (CPAP) use effects in split-night sleep study.

Methods: The consent of the study was obtained. Watch-PAT was simultaneously worn on a patient admitted for split-night polysomnography. The apnea-hypopnea index (AHI) obtained from PSG and the pAHI gained from the watch-PAT were measured when not using CPAP and when using CPAP respectively. And also we examined whether the reduction rates of AHI and pAHI could be correlated.

Results: 38 subjects (32 men, age 55 \pm 13 years old). BMI 28.3 \pm 5.7 kg / m². When CPAP was not used, AHI was 57.2 \pm 23.3 / h and pAHI was 50.8 \pm 20.3 / h (r = 0.93, p < 0.0001), when CPAP was used, AHI was 5.2 \pm 4.5 /h and pAHI was 6.2 \pm 4.5 h (r = 0.82, p < 0.0001), AHI reduction rate was 90.4 \pm 8.0% and pAHI reduction rate was 85.4 \pm 14.6% (r = 0.76, p < 0.0001).

Conclusion: It was suggested that Watch-PAT had a good correlation with AHI at split night-sleep study.

Support: None

0591

THE SIGNIFICANCE OF BODY MASS INDEX IN VARYING SLEEP APNEA LEVELS FOR MEN AND WOMEN

Jones, A. M.¹ Rogers, A. E.² Hertzberg, V. S.² Bliwise, D. L.³ Lewis, T. T.³

¹Georgia State University Byrdine F. Lewis School of Nursing and Health Professions, Atlanta, GA, ²Emory University Nell Hodgson Woodruff School of Nursing, Atlanta, GA, ³Emory University, Atlanta, GA.

Introduction: A BMI increase, in men and women, is associated with an increased severity and progression of OSA. This study will examine the impact of BMI on varying levels of OSA severity and progression.

Methods: Participants, divided by sex, included 2728 (47%) men and 3076 (53%) women over the age of 40 that were in the Sleep Heart Health Study (SHHS). Participants were separated into 1 of 10 groups based on initial OSA levels at SHHS time point 1 (SHHS1) and ending OSA levels at SHHS time point 2 (SHHS2) as measured by RDI. A Kruskall-Wallis test examined the BMI median differences in the groups. Post-hoc tests, including pairwise comparisons and Wilcoxon rank sum test with Holm adjustment, were conducted to further examine results.

Results: Significant differences existed between certain groups (Men: Chi-Square=146.87, p<.001, df=9; Women: Chi-Square=128.59, p<.001, df=9). For men and women, those in the group with normal OSA levels at SHHS1 and SHHS2 had significant BMI differences compared to those in all 9 other groups where mild, moderate, or severe OSA levels exist at SHHS1 or SHHS2. Additionally, in men, BMI is significantly different for those with normal or mild OSA levels at SHHS2 compared to those with moderate or severe OSA levels at SHHS2. Also, a significant BMI difference exists in men maintaining mild OSA levels throughout SHHS compared to those maintaining severe OSA levels.

Conclusion: Although BMI is a known influential factor in OSA progression, this study demonstrated that those maintaining normal OSA levels over time have a significant BMI difference compared to those reaching even mild OSA levels over time. Additional implications were also found for men. These findings may coincide with recent research suggesting that one needs to progress only to moderate OSA levels to reach a tipping point of significantly increasing and impacting many health risks.

Support: Robert Wood Johnson Foundation Future of Nursing Scholars Program

0592

PREVALENCE OF SLEEP APNEA IN PATIENTS WITH TRACHEOBRONCHOMALACIA

Chopra, S.¹ Luthra, S.¹ Dalal, L.¹ Blattner, M.¹ August, J.¹ Thomas, R.¹ Heckaman, E.¹

¹BIDMC, Boston, MA, ²BIDMC, Boston, MA.

Introduction: Tracheobronchomalacia (TBM) is a pathologic weakness in the trachea and bronchi leading to excessive dynamic narrowing of the airway. A relationship between sleep disordered breathing (SDB) and TBM has been observed before. SBD may be an important contributor to development or progression of TBM. The objective was to determine the Prevalence and characteristics of sleep disordered breathing in patients with tracheobronchomalacia.

Methods: We performed a retrospective chart review of patients who have been diagnosed with tracheobronchomalacia and who also underwent a polysomnogram (PSG) at the AASM - accredited Sleep Center of Beth Israel Deaconess Medical Center.

Results: In our 24 patient cohort of TBM, 71% were females, mean age 55 years (SD \pm 12.3 years) and mean BMI 31.7 kg/m² (SD \pm 9.4 kg/m²). In patients with TBM we found a sleep apnea prevalence of 62.5% (n= 15), defined as an apnea-hypopnea index>5/hour (hour) with a desaturation greater than 4%. Of the 15 patients, 73.3% (n = 11) had mild sleep apnea, 20% (n = 3) had moderate sleep apnea, 6.6% (n = 1) had severe sleep apnea, defined per the AASM criteria with oxygen desaturation greater than 4%. The TBM cohort had a mean sleep efficiency of 72.7% (SD \pm 22.2%) with a mean REM of 16.3% (SD \pm 9.8%). Other characteristics included a median AHI 3% of 19.9/ hour (95% CI 3.9 - 25.0), median AHI 4% of 5.5/hour (95% CI 3.9 - 9.3), Respiratory disturbance index of 22/hour (95% CI 15.1 to 28.4). No unique challenges for treatment with positive airway pressure were noted.

Conclusion: Sleep apnea may be more common in patients with tracheobronchomalacia and could be regularly screened. **Support:** none

HYPOXEMIA DURING SLEEP DISORDERED BREATHING AND CARDIOVASCULAR DISEASE: A COMPARISON OF DIFFERENT OXYGEN DESATURATION MEASURES

Mazzotti, D. R.¹ Leppänen, T.² Sands, S.³ Töyräs, J.⁴ Wellman, A.³ Kulkas, A.² Redline, S.³ Karhu, T.² Azarbarzin, A.³ ¹University of Pennsylvania, Philadelphia, PA, ²University of Eastern Finland, Kuopio, FINLAND, ³Brigham and Women's Hospital and Harvard Medical School, Boston, MA, ⁴The University of Queensland, Brisbane, AUSTRALIA.

Introduction: The apnea-hypopnea index has been used to characterize obstructive sleep apnea (OSA) severity. However, this metric is limited in providing information about cardiovascular disease (CVD) risk. Recent studies proposed alternative metrics that capture frequency, duration, depth, and combinations of duration and depth of hypoxemia. This study provides a systematic evaluation of the association between conventional or novel nocturnal hypoxemia metrics and the incidence of CVD and CV mortality in the Sleep Heart Health Study (SHHS).

Methods: We used data from 5,042 participants of the SHHS. Over 10.7 years, there were 1,312 (26.0%) incident CVD events and 359 (7.1%) CV deaths. We calculated standardized (z-scored) values of eight nocturnal hypoxemia indices, including conventional (e.g., oxygen desaturation index) and novel metrics (e.g., hypoxic burden, respiratory event-related area under desaturation curve and desaturation severity, corresponding to alternative quantitative measurements looking at the shape of each desaturation event). The association between each metric and incidence of CVD or CV mortality was evaluated using Cox proportional hazards models. Age, sex, body mass index, race, ethnicity, smoking, total sleep time, number of respiratory events, and prevalent CVD at baseline were used as covariates. Hazard ratios (HR) are presented as the effect of one standard deviation increase in each correponding metric.

Results: In unadjusted models, all nocturnal hypoxemia indices were associated with increased incidence of CVD and CV mortality. In adjusted models, longer average desaturation duration was associated with lower CVD incidence (HR[95%CI]=0.93[0.86-0.99];p=0.034), higher hypoxic burden with increased CV mortality (HR[95%CI]=1.22[1.04-1.43];p=0.017), and higher % sleep time with oxygen saturation less than 90% (Tlt90%) with increased CV mortality (HR[95%CI]=1.12[1.00-1.26];p=0.040).

Conclusion: Different metrics of nocturnal hypoxemia derived from polysomnography were associated with CV risk in the SHHS. However, after covariate adjustment, only shorter average desaturation duration, and higher hypoxic burden and Tlt90% were independent CV risk factors.

Support: AASM Foundation (194-SR-18,188-SR-17); American Heart Association (19CDA34660137); NIH (U01HL53940,U 01HL53941,U01HL63463,U01HL53937, U01HL53938,U01H L53916,U01HL53934,U01HL63429,U01HL53931,HL114473, P01HL094307,HL134015,R35HL135818,1R21HL145492-01,R01HL102321,R01HL128658); The State Research Funding (KUH: 5041767, 5041768; TUH: VTR3242, VTR3228, EVO2089), Academy of Finland (313697, 323536), Business Finland (5133/31/2018), Respiratory Foundation of Kuopio Region, Tampere Tuberculosis Foundation, Research Foundation of Pulmonary Diseases, Foundation of Finnish Anti-Tuberculosis Association.

0594

CAN A DEEP CONVOLUTIONAL NEURAL NETWORK EXTRACT DIAGNOSTIC INFORMATION ON OBSTRUCTIVE SLEEP APNEA FROM IMAGES?

Tsuiki, S.¹ Nagaoka, T.² Fukuda, T.¹ Sakamoto, Y.³ Almeida, F. R.⁴ Nakayama, H.¹ Inoue, Y.¹ Enno, H.²

¹Institute of Neuropsychiatry, Tokyo, JAPAN, ²Rist Inc., Kyoto, JAPAN, ³Kyoto University, Kyoto, JAPAN, ⁴The University of British Columbia, Vancouver, BC, CANADA.

Introduction: Lateral cephalometric radiography is a simple way to provide craniofacial soft/hard tissue profiles specific for patients with obstructive sleep apnea (OSA) and may thus offer diagnostic information on the disease. We hypothesized that a machine learning technology, a deep convolutional neural network (DCNN), could make it possible to detect OSA based solely on lateral cephalometric radiographs without the need for either large amounts of subjective/laboratory data or skilled analyses.

Methods: In this diagnostic study, a DCNN was developed (n=1,258) and tested (n=131) using data from 1,389 lateral cephalometric radiographs obtained from individuals diagnosed with severe OSA (n=867; apnea hypopnea index >30/ hour) or non-OSA (n=522; apnea hypopnea index < 5) at a single center for sleep disorders from March, 2006 to February, 2017. Three kinds of data sets were prepared by changing the area of interest using a single image; original image without any modification (Full Image), image containing a facial profile, upper airway, craniofacial soft/hard tissues, and image containing part of the occipital region (upper left corner of the image; Head Only). A radiologist and an orthodontist also performed a manual cephalometric analysis of the Full Image for comparison. Observers were blinded to the patient groupings. Data analysis was performed from April, 2018 to August, 2019. When the predictive score obtained from the DCNN analysis exceeded the threshold (0.50), the patient was judged to have OSA. The primary outcome was diagnostic accuracy in terms of area under the receiver-operating characteristic curve.

Results: The sensitivity/specificity was 0.87/0.82 for Full Image, 0.88/0.75 for Main Region, 0.71/0.63 for Head Only, and 0.54/0.80 for the manual analysis. The area under the curve was the highest for Main Region (0.92): 0.89 for Full Image, 0.70 for Head Only, and 0.75 for the manual analysis.

Conclusion: A DCNN identified individuals with OSA with high accuracy. This is a useful approach that does not require any laborious analyses in a primary care setting or in remote areas where an initial specialized OSA diagnosis is not feasible.

Support: This study was supported in part by the Japan Society for the Promotion of Science (grant numbers 17K11793, 19K10236).

0595

OBSTRUCTIVE SLEEP APNEA CHARACTERISTICS IN YOUNGER VERSUS OLDER WOMEN

Budhiraja, R. Limbekar, N. Quan, S.

Brigham and Women's Hospital, Harvard Medical School, Boston, MA.

Introduction: There are few large studies of obstructive sleep apnea (OSA) comparing the clinical and polysomnographic characteristics between men and women, and between younger and older women.

Methods: The current study involves retrospective analyses of data from the Apnea Positive Pressure Long-term Efficacy Study (APPLES), a prospective multicenter randomized controlled trial in persons with OSA. The mean age of the 1105 participants was 51.6 ± 12 years (range 18-83 years). The participants included 723 men (65.4%) and 382 women (34.6%). Of all women, 25% were <=45 years of age (likely pre-menopausal) and 50% were above >=53 years of age (likely post-menopausal). We used these 2 groups to define younger women and older women, respectively.

Results: The overall mean body mass index (BMI) and apnea hypopnea index (AHI) were 32.2 ± 7.1 Kg/m2 and 40.1 ± 25.2 (range 6-156/hour), respectively. Women had a higher BMI and Epworth Sleepiness Scale (ESS) than men but a lower AHI and lower arousal index (AI). The Hamilton Rating Scale for Depression (HAMD) score was also significantly higher in women. Younger men and women had no other PSG differences including total sleep time (TST), Sleep Efficiency (SE), Sleep Onset Latency (SOL). Older women had lower AHI, higher TST and higher SE than older men. Morningness-Eveningness Questionnaire (MEQ) scores were lower in women than in men in all age groups (suggestive of eveningness). Compared to the older women, younger women had higher BMI, HAMD score and ESS, but lower MEQ score. On PSG, younger women had higher TST and SE but similar SOL, AHI and AI.

Conclusion: In this cohort with OSA, the prevalence of sleepiness and depression is higher in women than in men despite a lower AHI and lower AI. Sleepiness and depression are more common in younger compared to older women with OSA despite similar AHI and AI and a higher SE. Women with OSA demonstrate more 'eveningness' compared to men with OSA, as do younger women compared to older women.

Support: Contract 5UO1-HL-068060 from the National Heart, Lung and Blood Institute

0596

PROVIDERS RARELY ASSESS OBSTRUCTIVE SLEEP APNEA SYMPTOMS AMONG PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Donovan, L. M.¹ Keller, T.¹ Stewart, N. H.² Spece, L. J.¹ Au, D. H.¹ Feemster, L. C.¹

¹VA Puget Sound Health Care System, Center for Veteran-Centered and Value-Driven Care, Seattle, WA, ²University of Kansas, Kansas City, KS.

Introduction: Professional societies recommend providers assess sleep symptoms in COPD, but it is unclear if this occurs. We aimed to evaluate OSA symptom assessment and documentation among patients with COPD, and the patient and provider characteristics associated with this assessment.

Methods: We conducted a cross-sectional study of adults aged \geq 40 years with clinically diagnosed COPD and no prior diagnosis of OSA. We selected patients receiving care at two academic general internal medicine clinics between 6/1/2011 - 6/1/2013. We abstracted charts to assess how often OSA symptoms such as snoring, somnolence, witnessed apneas, or gasping/choking arousals were documented as present or absent. We performed multivariable mixed-effects logistic regression to assess associations of patient and primary care provider (PCP) factors with assessment of OSA symptoms. Patient factors included demographics, body mass index, comorbidities, healthcare utilization, and severity of COPD, and PCP factors including demographics, degree, and years of experience.

Results: Of 523 patients with COPD, only 26 (5.0%) had documentation of OSA symptom assessment within a one-year period. In mixed effects models, only referral to general pulmonary clinic was associated with the assessment of OSA symptoms (OR: 4.56, 95% CI 1.28-15.52). Among the 26 individuals who had OSA symptoms assessed, 9 (34.6%) reported snoring, 15 (57.7%) reported daytime somnolence, 2 (7.7%) reported gasping/choking arousals, and 5 (19.2%) reported witnessed apneas. Among those assessed for OSA symptoms, providers referred 11 (42.3%) for formal sleep consultation.

Conclusion: Providers rarely document OSA symptoms for patients with COPD in primary care clinic, but assessment is greater among those with pulmonary specialty consultation. Given time constraints in primary care, external facilitation of sleep symptom assessment may improve symptom recognition and receipt of appropriate services.

Support: NIH 5K23HL11116-05, VA Center of Innovation for Veteran-Centered and Value-Driven Care.

0597

PRECISION OF SLEEP-DISORDERED BREATHING EVENT CLASSIFICATION USING SIMULATED HOME SLEEP APNEA TESTING IN PATIENTS WITH SPINAL CORD INJURY, OR DISEASE

Zeineddine, S.^{1,2} Sankari, A.^{1,2} Arvai, k.^{1,2} Salloum, A.^{1,2} Abu Awad, Y.³ Martin, J. L.^{4,5} Badr, M. S.^{1,2} ¹John D Dingell VAMC, Detroit, MI, ²Wayne State University, Detroit, MI, ³Concordia University, Montreal, QC, CANADA, ⁴VA Greater Los Angeles Healthcare System, Los Angeles, CA,

⁵David Geffen School of Medicine at UCLA, Los Angeles, CA.

Introduction: Sleep-disordered breathing (SDB) is highly prevalent among patients with spinal cord injury or disease (SCI/D). In-laboratory polysomnography (PSG) is difficult for these patients due to functional limitations and the physical construction of most sleep laboratories. Our objective was to evaluate the concordance between simulated HSAT and PSG in identifying SDB severity and subtypes of respiratory events in this patient population.

Methods: Within a larger study, 33 Veterans with SCI/D completed one night of in-laboratory PSG. Limited-channel HSAT was simulated by extracting 5 channels from PSG signals to include nasal pressure, thermistor, thoracic and abdominal belts, and oxygen saturation.

Results: Mean age of patients was 59.8 ± 10.9 years; 87.9% were male, and the average BMI was 28.1 ± 6.3 . The mean Apnea-Hypopnea Index (AHI) from PSG was 35.5 ± 22.7 . The mean Respiratory Event Index (REI) based on simulated HSAT was 22.5 ± 18.6 . Thirty-one patients (93.9%) had SDB defined as AHI \geq 5/hour. Simulated limited-channel HSAT accurately identified 32 out of 33 patients (96.96%). When SDB was further classified into mild (AHI 5-15 events/hr), moderate (AHI 15-30 events/hr), and severe (AHI>30/hr), simulated HSAT consistently underestimated the severity of underlying SDB. Spearman correlation between estimating AHI (PSG-HSAT) and subtypes of respiratory events was primarily accounted for by the difference in the number of hypopneas (r=0.72, -0.021 and -0.001 for hypopneas, obstructive and central apneas, respectively).

Conclusion: Our findings support the diagnostic utility of HSAT in SCI/D patients with SDB; however, HSAT underestimation of SDB may lead to difficulties in optimizing therapy. The misclassification of SDB severity is mainly driven by the number of hypopneas. Classification of hypopneas as obstructive or central

may shed further light on the nature of this difference. Further research on the usability of HSAT devices in this patient population is needed.

Support: VA Rehabilitation Research and Development Service (RX002116; PI Badr and RX002885; PI Sankari) and NIH/ NHLBI (K24HL143055; PI: Martin)

0598

BLURRED BINARIES: THE CLINICAL MANAGEMENT OF OBSTRUCTIVE SLEEP APNEA IN TRANSGENDER PATIENTS

Peters-Mathews, B. Lee, M. Sabzpoushan, A. Virginia Mason Medical Center, Seattle, WA.

Introduction: Transgender patients require careful clinical assessment to identify the effects of hormones on their risk of obstructive sleep apnea. Testosterone increases sleep apnea risk and assigned males at birth may develop the condition earlier. Estrogen and progesterone are known to reduce sleep apnea risk. Depending on the use of supplemental hormones and surgical status, the risk of sleep apnea may be altered in a transgender patient. Comorbid conditions, including mood disorders and obesity, may further impact sleep. This topic has not been well-studied, and this pilot project identifies special needs that exist in transgender populations.

Methods: This retrospective chart review included 25 subjects who identify as transgender who have been managed at an urban sleep disorders center from 2017 to 2019. The case series was assessed to identify characteristics that impact the diagnosis and treatment of obstructive sleep apnea (natural and supplemental hormonal effects, surgical effects, comorbid conditions, etc.).

Results: The average age of the cohort was 34 years (range 16 to 76). Fourteen subjects were assigned females at birth and identify as men and 11 subjects were assigned males at birth and identify as women. Preferred pronoun usage was concordant with gender identity in 21 subjects, discordant in 1 subject, and gender-neutral (they/them) was used by 3 subjects. Based on 24 subjects, the average BMI was 35.5 (range 23.5 to 53.1). The measured neck circumference was <16 inches in 9 subjects, >17 inches in 6 subjects, and a risk factor for sleep apnea based on birth-assigned sex but not gender identity in 4 subjects. Hormone therapy was used by 24 subjects to enhance their gender identities. When documented, 6 subjects had mastectomies and 2 also had total hysterectomies. Anxiety or depression had been diagnosed in 20 subjects. Testing revealed sleep apnea in 18 subjects. Loss to follow up affected 9 subjects.

Conclusion: Transgender patients deserve respectful evaluation and careful consideration regarding risk factors for obstructive sleep apnea that may be impacted by gender-affirming hormonal therapy or surgery, and weight gain. Higher rates of mood disorders and loss to follow up may put these patients at long-term risk.

Support: N/A

0599

THE ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP **APNEA AND CANCER INCIDENCE AND MORTALITY:** A SYSTEMATIC REVIEW AND META-ANALYSIS

Sutherland, R. Platt, J.

University of Calgary, Cumming School of Medicine, Community Health Sciences, Calgary, AB, CANADA.

Introduction: Sleep related breathing disorders (SRBD) are common (20% prevalence) in the general population and can

have multiple health consequences. There is growing evidence that chronic hypoxia - a key consequence of sleep apnea - is a common feature in solid tumour tissue, therapeutic resistance, tumour progression, and metastasis. However, there is conflicting evidence regarding the association between sleep apnea, cancer incidence, or mortality. A review of all available literature and subsequent metaanalysis was done to clarify these relationships.

Methods: A thorough literature search was completed using Medline, EMBASE and Web of Science databases. The search resulted in 7222 studies. 1551 duplicates were removed. 5552 studies were removed after abstract screening, and full text review was done on 119 studies, yielding 12 full retrospective cohort studies. The risk of bias was assessed using the Newcastle-Ottawa Scale. Review and data extraction were done in duplicate.

Results: In the pooled analysis, 9 studies totalling 2,358405 subjects with OSA and 3.97% cancer incidence and 2,442794 subjects without OSA and 3.35% cancer incidence. A random effects model with inverse-variance weighting analysis yielded an unadjusted OR = 1.32 (95% CI: 0.76 - 2.30). After 2 studies with a moderate risk of bias were removed the pooling yielded an OR = 1.89 (95%)CI: 0.99 - 3.50). Heterogeneity was high at 99.9% p-value less than 0.01. Meta-regression was then done to assess for the cause(s) of heterogeneity sex, age, or BMI were not significant contributors. A review of 3 studies, which included cancer mortality, was done. Hazard ratios in 2 studies suggested OSA increased the risk of cancer mortality. Hazard ratios also increased with increasing OSA severity.

Conclusion: Sleep apnea significantly increases cancer mortality and is positively associated with increasing severity. Meta-analysis demonstrated an 86% increase in the unadjusted odds of cancer in those with sleep apnea. However, this result was borderline nonsignificant with high heterogeneity. Further studies may be helpful in determining the true associations between sleep apnea and cancer. Support: None.

0600

AUTOMATED OXIMETRIC VERSUS STANDARD SLEEP POLYGRAPHY SCORING

Skjodt, N. M.¹ Pahwa, V.² Platt, R. S.²

¹Canadian Centre for Behavioural Neuroscience, Lethbridge, AB, CANADA, ²Sagatech Electronics Inc., Calgary, AB, CANADA.

Introduction: Agreement between automated standard respiratory event scoring and a novel, validated, and patented oximetry-based algorithm was assessed.

Methods: The standard apnea-hypopnea index (AHI) was derived by adding apneas (flow drop $\geq 90\%$ for 10 to 30 s) and hypopneas (flow drop $\geq 30\%$ for 10 to 60 s with oxygen saturation (SpO2) dropping $\geq 3\%$). The novel oxygen index (ODI4) was derived by scoring events where SpO2 dropped in each of three successive samples and cumulatively by $\geq 4\%$. Agreement was assessed by Bland-Altman analysis

Results: AHI versus ODI4 and Bland-Altman plots showed a high prevalence of AHI > ODI4 when AHI was< 30/h. Negative difference outliers were frequent when mean index difference was > 30/h. There was a bias of 2.83/h in the difference between AHI and ODI4 with upper and lower confidence limits of 22.0/h and -16.3/h.

Conclusion: Standard respiratory event scoring overestimates respiratory disturbance compared to a novel oximetric index. Standard automated scoring frequently over scores events when basal flow amplitude is low.

Support: None.

USE OF THE EPWORTH SLEEPINESS SCALE, THE NOSAS, AND THE STOP-BANG QUESTIONNAIRE TO IDENTIFY PATIENTS WITH MODERATE-TO-SEVERE OBSTRUCTIVE SLEEP APNEA

YIN, S.^{1,2,3} XU, H.^{1,2,3} ZHANG, C.^{1,2,3} ZOU, J.^{1,2,3} GUAN, J.^{1,2,3} YI. H.^{1,2,3}

¹Department of Otolaryngology Head and Neck Surgery and Center of Sleep Medicine, Shanghai Sixth People's Hospital Affiliated to Shanghai Jiao Tong University, Shanghai, CHINA, ²Shanghai Key Laboratory of Sleep Disordered Breathing, Shanghai, CHINA, ³Otolaryngological Institute of Shanghai Jiao Tong University, Shanghai, CHINA.

Introduction: A variety of scales and questionnaires regarding sleep and sleep-related disorders have been widely used in scientific research and clinical practice, as important tools for differential diagnosis and rapid screening of complex sleep disorders, especially obstructive sleep apnea (OSA). However, the diagnostic efficacy of different scales and questionnaires for patients with different severity of OSA and of different demographic characteristics has not been clearly described. In this study, we evaluated the ability of the most popular scales, including the Epworth Sleepiness Scale (ESS), the NoSAS, and the STOP-BANG questionnaire in predicting moderate-to-severe obstructive sleep apnea (OSA) by gender.

Methods: This cross-sectional study screened 2,031 consecutive subjects referred with suspected OSA from 2012 to 2016. Anthropometric measurements, polysomnographic data, ESS, NoSAS scores and STOP-BANG scores were recorded. Receiver operating characteristic curve analyses were performed, and the final predictive models were verified in a validation cohort.

Results: A total of 1,840 adults were finally included. The STOP-BANG questionnaire afforded a better diagnostic accuracy than did the ESS, with different cutoffs for the two genders: 3 in males and 1 in females. A predictive model based on STOP-BANG yielded an area under the curve (AUC) of 0.918 (0.897-0.935), a sensitivity of 79.89%, and a specificity of 89.19%, in males; and an AUC of 0.951 (0.914-0.975), a sensitivity of 80.52%, and a specificity of 95.92%, in females. In the validation cohort, the sensitivity and specificity were respectively 85.44 and 93.00% in males and respectively 83.02 and 87.60% in females.

Conclusion: The STOP-BANG questionnaire was moderately effective when used to screen for moderate-to-severe OSA. A STOP-BANG-based predictive model afforded excellent diagnostic efficacy, which could be applied in clinical practice. However, gender differences must be considered.

Support: This study was supported by Grants-in-aid from Shanghai Municipal Commission of Science and Technology (Grant No.18DZ2260200).

0602

DAYTIME SLEEPINESS IN HEART FAILURE WITH PRESERVED VERSUS REDUCED EJECTION FRACTION

Schütz, S. G. Nguyen-Phan, A. Konerman, M. Hummel, S. Chervin, R. D.

University of Michigan, Ann Arbor, MI.

Introduction: Sleep apnea is common in patients with heart failure, though often not associated with significant daytime sleepiness in heart failure with reduced ejection fraction (HFrEF). The clinical presentation of sleep apnea in patients who have heart failure with

borderline or preserved ejections fraction (HFbEF and HFpEF, respectively) is not well characterized.

Methods: Eighty patients with heart failure were identified retrospectively in data from University of Michigan Sleep Disorders Laboratories. Heart failure was categorized as heart failure with reduced ejection fraction (HFrEF)/systolic heart failure, heart failure with borderline ejection fraction (HFbEF) or heart failure with preserved ejection fraction (HFpEF)/diastolic heart failure. Clinical information and Epworth Sleepiness Scale (ESS) scores were extracted from medical records. A subset of subjects underwent a diagnostic polysomnogram. ANOVA was used to compare clinical characteristics in subjects with different heart failure types. Results: ESS scores trended higher in 49 subjects with HFpEF (ESS mean 10.9±4.7 [sd]) compared to 9 with HFbEF (ESS 8.0±3.4) and 22 with HFrEF (ESS 8.4 ± 5.0) (p=0.058). Among the 40 subjects who underwent diagnostic polysomnography, no statistically significant difference emerged in apnea-hypopnea index between subjects with HFpEF, HFbEF, and HFrEF (p=0.43). No significant differences emerged for the central apnea index (p=0.16), despite magnitudes of discrepancy that suggested a larger sample size might show different

results: CAI in participants with HFrEF showed a mean of $9.0\pm14.6/h$, compared to $0.1\pm0.1/h$ in HFbEF and $3.1\pm6.3/h$ in HFpEF.

Conclusion: Among these patients with HFpEF, HFbEF, and HFrEF, subjects with HFpEF showed a trend towards increased subjective daytime sleepiness, though overall apnea and central apnea severity did not differ between groups. Further examination of clinical phenotypes in larger cohorts may help guide care in heterogeneous heart failure populations.

Support: National Institutes of Health grant NS107158

0603

DIVIDED ATTENTION STEERING SIMULATOR COMPARED TO OTHER DAYTIME SLEEPINESS TESTS IN SLEEP APNEA

Penzel, T.¹ Henning, S.¹ Glos, M.¹ Huang, Y.¹ Fietze, I.¹ ¹Charite University Hospital, Berlin, GERMANY, ²Charite University Hospital, Berlin, GERMANY.

Introduction: Sleep apnea is often associated with daytime sleepiness. A test which may be similar to driving situations is the divided attention steering Simulator (DASS) as a surrogate for testing sleepiness at the wheel. In this study we compared DASS parameters against Epworth sleepiness score (ESS) and psychomotor vigilance test (PVT) in subjects with sleep apnea.

Methods: In 16 healthy subjects without sleep disorders and in 38 patients with sleep apnea, we tested daytime sleepiness and Performance using different methodologies. We applied the DASS twice, the PVT twice, the testbattery for attentional Performance (TAP), and the MSLT during one day after polysomnography.

Results: Sleep apnea patients had a longer response time according to DASS (p<0.05) and a larger average deviation from midline of the road (p<0.05) and left the road more frequently (p<0.05). Response time in DASS correlated well with PVT (p<0.001). We found no significant association between ESS and DASS parameters. We no correlation between MSLT sleep latency and DASS parameters.

Conclusion: The DASS is very well to mirror reaction time as determined by PVT. However, the DASS does not reflect sleepiness as determined by MSLT or ESS. The study confirms the finding that reaction time testing, even using different tools, does not relate very

well with perceived subjective sleepiness by ESS nor by objective sleepiness as assessed by multiple sleep latency test. Different measures do form a complex Picture which might lead to falling asleep when driving. All tests do show some limitations regarding this assessment.

Support: This study was supported by Charite University funds.

0604

COMORBIDITIES AND ADMISSION RATES IN INPATIENTS UNDERGOING SLEEP STUDIES

Johnson, K. G.¹ Ravikumar, N.¹ Scuderi, N.² Sharma, A.³ Rastegar, V.¹ Visintainer, P.¹

¹University of Massachusetts Medical School- Baystate, Springfield, MA, ²University of Massachusetts-Amherst, Amherst, MA, ³Baystate Medical Center, Springfield, MA.

Introduction: Uncontrolled sleep-disordered breathing (SDB) and hypoventilation, which are common in COPD, CHF and obesity hypoventilation patients can lead to death and readmissions. It is unknown whether inpatient sleep studies to diagnose and optimize treatment improve care and prevent readmissions.

Methods: All patients > 18 years old with sleep studies while inpatient at Baystate Medical Center between October 2015 and September 2017 were included. Patient characteristics, comorbidities, sleep study diagnoses, and treatment recommendations were evaluated. Admission (inpatient or observation) and death rates were determined for 1-year before admit date and 1-year after discharge date of index admission.

Results: 326 adult inpatients had 120 portable and 304 in-laboratory tests performed. Average age was 62.9±14.4, mean BMI was 37.2±12.3 and 56% were male. Principal diagnoses were CHF (50%), COPD (39%), both COPD and CHF (20%) and obesity hypoventilation (27%). 31 used PAP and 71 used oxygen prior to admission. Sleep diagnoses included OSA (73%), central sleep apnea (CSA) (29%), treatment emergent CSA (8%), hypoxia (48%), hypoventilation (41%), and normal or non-diagnostic (6%). Treatment recommendations included CPAP (25%), BiPAP (18%), BiPAP ST (3%), ASV (4%), iVAPS (22%), oxygen only (5%) and further titration (20%). The average length of stay was 11.6 ± 9.6 days. There was no difference in the percentage of patients who had an admission before or after their sleep study (53% vs 56%, respectively). In addition, no difference was seen in the median number of admissions before and after the sleep study (median=1.0, IQI=0-2, p=0.77). 90-day readmission rate was 19%. 14% died.

Conclusion: SDB, hypoxia and hypoventilation were common in inpatients evaluated with sleep studies with PAP therapy recommended in most patients. Further research is needed to determine whether inpatient testing and subsequent treatment can result in decreased readmissions and death. **Support:** None

0605

COMPARISON OF SLEEP SPECIALIST VS PRIMARY CARE DRIVEN HOME SLEEP APNEA TESTING IN ROUTINE PRACTICE

He, K. Mendez, M. Atwood, C. W. Sleep Medicine, VA Pittsburgh Healthcare System, Pittsburgh, PA.

Introduction: Home sleep apnea testing (HSAT) has largely supplanted diagnostic polysomnography. Primary care (PC) driven HSAT utilization is common especially in rural settings that lack sleep

specialist (SS) support. There have been no studies comparing appropriateness of HSAT utilization in veterans managed by SS vs. PC. **Methods:** We use hub and spoke model to manage patients with OSA. SS selects testing for hub and PC utilizes HSAT for spoke

patients. Testing is interpreted by SS. Patients referred for HSAT using WatchPAT over 4 months were compared on test failure rate, adherence to AASM guidelines for OSA diagnosis, adherence to HSAT use criteria, and diagnostic success rate (AHI \geq 5) in high risk patients (STOPBANG \geq 5) without significant comorbidities or HSAT contraindications compared to all comers.

Results: There were 125 hub and 170 spoke patients included in the analyses. Baseline characteristics were similar between sites (gender, age, BMI, Epworth sleepiness scale, neck size, STOPBANG, pace-maker dependence, and medication use affecting HSAT). Spoke patients had slightly higher prevalence of comorbidities (hypertension, cardiac arrhythmia, heart failure, COPD, stroke, and long acting opioid use). Complete HSAT failure (no data) was 2% and technical failure (monitoring time <4 hours) was 13% at both sites. Unnecessary studies primarily to confirm OSA in those previously diagnosed on therapy seeking to establish care were 3% (hub) and 21% (spoke). HSAT done in patients without significant comorbidities was 77% (hub) and 68% (spoke). Adherence to HSAT use criteria was 74% at both sites. Diagnostic success rate of prespecified and all comers was 65% vs. 60% at hub and 86% vs. 64% at spoke sites.

Conclusion: Adherence to AASM guidelines and HSAT use criteria was overall fair with low failure rates. Further improving HSAT protocol for SS and PC with the aim to improve diagnostic success rate and minimize unnecessary studies should be pursued. **Support:**

0606

COMPARATIVE EFFECTIVENESS OF SLEEP APNEA SCREENING TOOLS DURING INPATIENT REHABILITATION FOR MODERATE TO SEVERE TBI

Richardson, R.¹ Schwartz, D.² Drasher-Phillips, L.³ Ketchum, J.⁴ Calero, K.² Dahdah, M.⁵ Monden, K.⁶ Bell, K.⁷ Hoffman, J.⁸ Magalang, U.⁹ Bogner, J.¹⁰ Whyte, J.¹¹ Zeitzer, J.¹² ¹James A. Haley Veterans Hospital, Tampa, FL, ²Medicine Service, James A. Haley Veterans' Hospital, Tampa, FL, ³Research Department, James A. Haley Veterans Hospital, Tampa, FL, ⁴Research Department, Craig Hospital, Denver, CO, ⁵Baylor Scott & White Institute for Rehabilitation, Dallas, TX; Baylor Scott & White Medical Center, Dallas, TX, 6Craig Hospital, Denver, CO, ⁷Department of Physical Medicine and Rehabilitation, UT Southwestern Medical Center, Dallas, TX, ⁸Department of Rehabilitation Medicine, University of Washington School of Medicine, Seattle, WA, 9Division of Pulmonary, Critical Care, and Sleep Medicine and Neuroscience Research Institute, The Ohio State University Wexner Medical Center, Columbus, OH, ¹⁰Department of Physical Medicine and Rehabilitation, Ohio State University, Columbus, OH, ¹¹Moss Rehabilitation Research Institute, Albert Einstein Healthcare Network, Philadelphia, PA, 12Psychiatry and Behavioral Service, Stanford University, Palo Alto, CA.

Introduction: Recent studies highlight prevalent obstructive sleep apnea after moderate to severe TBI during a time of critical neural repair. The purpose of this study is to determine the diagnostic sensitivity, specificity and comparative effectiveness of traditional sleep apnea screening tools in TBI neurorehabilitation admissions. **Methods:** This is a prospective diagnostic comparative effectiveness trial of sleep apnea screening tools (STOPBANG, Berlin, MAPI [Multi-Apnea Prediction Index]) relative Level 1 polysomnography at six TBI Model System Inpatient Rehabilitation Centers. Between 05/2017 and 02/2019, 449 of 896 screened were eligible for the trial with 345 consented (77% consented). Additional screening left 263 eligible for and completing polysomnography with final analyses completed on 248. The primary outcome was the Area Under the Curve (AUC) of screening tools relative to total apnea hypopnea index \geq 15 (AHI, moderate to severe apnea) measured at a median of 47 days post-TBI (IOR 29-47).

Results: Participants were primarily young to middle age (AGE IQR 28,40,59), male (81%), white (74%), and had primarily severe TBI (IQR GCS 3,6,14). A subset (26%) had a history of military service. Results revealed that the Berlin high risk score (ROC-AUC=0.63) was inferior to the MAPI (ROC-AUC = 0.7802) (p=.0211, CI: 0.0181, 0.2233) and STOPBANG (ROCAUC = 0.7852) (p=.0006, CI: 0.0629, 0.2302); both of which had comparable AUC (p=.7245, CI: -0.0472, 0.0678). Findings were similar for AHI≥30 (severe apnea); however, no differences across scales was observed at AHI>5. The pattern was similar across TBI severity subgroups except for delirium or post-traumatic amnesia status wherein the MAPI outperformed the Berlin and STOPBANG. Youden's Index to determine risk yielded lower sensitivities but higher specificities relative to non-TBI samples.

Conclusion: This study is the first to provide clinicians with data to support a choice for which sleep apnea screening tools are more effective during inpatient rehabilitation for moderate to severe TBI (STOPBANG, MAPI vs Berlin) to help reduce comorbidity and possibly improve neurologic outcome.

Support: PCORI (CER-1511-33005), GDHS (W91YTZ-13-C-0015; HT0014-19-C-0004)) for DVBIC, NIDILRR (NSDC Grant # 90DPTB00070, #90DP0084, 90DPTB0013-01-00, 90DPTB0008, 90DPT80004-02).

0607

CLINICAL PHENOTYPES OF OSA IN DIVERSE HISPANICS/LATINOS: RESULTS FROM THE HISPANIC COMMUNITY HEALTH STUDY/STUDY OF LATINOS

Gonzalez, K. T.¹ Tarraf, W.² Wallace, D. M.³ Stickel, A.⁴ Schneiderman, N.⁵ Redline, S.⁶ Patel, S. R.⁷ Gallo, L. C.⁸ Mossavar-Rahmani, Y.⁹ Daviglus, M.¹⁰ Zee, P. C.¹¹ Talavera, G. A.⁸ Sotres-Alvarez, D.¹² Gonzalez, H. M.¹ Ramos, A. R.¹³ ¹Department of Neurosciences and Shiley-Marcos Alzheimer's Disease Research Center, University of California, San Diego, La Jolla, CA, University of California San Diego, CA, ²Wayne State University, Detroit, MI, ³University of Miami, Miami, FL, ⁴Department of Neurosciences and Shiley-Marcos Alzheimer's Disease Research Center, University of California, San Diego, La Jolla, CA, La Jolla, CA, ⁵University of Miami, Department of Psychology, University of Miami, FL, ⁶Harvard Medical School, Harvard University, MA, ⁷University of Pittsburgh, School of Medicine, University of Pittsburgh, PA, 8 Institute for Behavioral and Community Health, Graduate School of Public Health, San Diego State University, San Diego, California, San Diego, CA, ⁹Albert Einstein College of Medicine, Department of Epidemiology & Population Health, Bronx, NY, ¹⁰Institute for Minority Health Research, University of Illinois at Chicago, College of Medicine, Chicago, Illinois, Chicago, IL, ¹¹Northwestern University, Feinberg School of Medicine, Department of Neurology, Chicago, IL, ¹²University of North Carolina, Department of Biostatistics, University of North Carolina, NC, ¹³University of Miami, Miller School of Medicine, Miami, FL.

Introduction: Recent work on US non-Latino Whites and Europeans from clinical samples used obstructive sleep apnea (OSA) symptoms to generate OSA phenotypes for individuals with moderate-severe OSA and proposed between 3-5 clusters. Validating these clusters in a diverse Hispanic/Latino community-based population with different biopsychosocial characteristics is crucial for early OSA identification and more personalized treatment.

Methods: This work is based on baseline data from The *Hispanic Community Health Study/Study of Latinos* (HCHS/SOL). HCHS/ SOL is a prospective cohort study designed using a multisite (Bronx, NY, Chicago, IL, Miami, FL, San Diego, CA) multistage probability sample. The subpopulation of interest included adults 18-74 years (unweighted n=1,623) meeting criteria for moderatesevere OSA symptoms (\geq 15 Apnea-Hypopnea index (AHI) events per hour). We performed latent class analysis (LCA) using 15 common OSA symptoms to identify phenotype clusters.

Results: Average age was 52.4 ± 13.9 years and 34.1% were female. Mean AHI was 33.8 ± 22.5 events per hour. Fit statistics and clinical significance suggested that a three-class solution provided best fit to the data. The symptom profiles were consistent with (1) a *Minimally Symptomatic* group (46.8%), (2) a *Disturbed Sleep* group (38.1%), and (3) a *Daytime Sleepiness* group (15.1%). Validation analyses using alternative hierarchical and partitioning algorithms also suggested support for a three-class solution.

Conclusion: Sleep apnea phenotypes among diverse Hispanics/ Latinos were consistent with recent findings from *the Sleep Apnea Global Interdisciplinary Consortium*. However, we found notable differences in the prevalence of these clusters relative to Whites. This suggests that other biopsychosocial factors may be contributing to OSA phenotypes among Hispanics/Latinos. Identification of OSA phenotypes in Hispanics/Latinos could inform better sleep interventions and therapeutics and help better align public health resources.

Support: 5R01AG048642-05; R21AG056952; R21HL140437.

0608

CONCORDANCE BETWEEN CURRENT AASM AND CMS SCORING CRITERIA FOR OBSTRUCTIVE SLEEP APNEA IN HOSPITALIZED PERSONS WITH TBI: A NIDILRR AND VA MODEL SYSTEM STUDY

Richardson, R.¹ Dahdah, M.² Almeida, E.³ Ricketti, P.⁴ Silva, M.⁵ Calero, K.⁶ Magalang, U.⁷ Scwhartz, D.⁶

¹James A. Haley Veterans Hospital, Tampa, FL, ²Baylor Scott & White Institute for Rehabilitation, Dallas, TX; Baylor Scott & White Medical Center, Dallas, TX, ³Research Department, Craig Hospital, Denver, CO, ⁴, Morsani College of Medicine, Division of Pulmonary and Sleep Medicine, University of South Florida, Tampa, FL, ⁵Mental Health and Behavioral Sciences Service, James A Haley Veterans' Hospital, Tampa, FL, ⁶Medicine Service, James A. Haley Veterans' Hospital, Tampa, FL, ⁷Division of Pulmonary, Critical Care, and Sleep Medicine and Neuroscience Research Institute, The Ohio State University Wexner Medical Center, Columbus, OH.

Introduction: The objective of this study was to compare OSA, demographic, and TBI characteristics across the American Academy of Sleep Medicine (AASM) and Centers for Medicare and Medicare (CMS) scoring rules in moderate to severe TBI undergoing inpatient neurorehabilitation.

Methods: This is a secondary analysis from a prospective clinical trial of sleep apnea at six TBI Model System study sites (n=248).

SLEEP, Volume 43, Abstract Supplement, 2020

Scoring was completed by a centralized center using both the AASM and CMS criteria for OSA. Hospitalization and injury characteristics were abstracted from the medical record and demographics obtained by interview by trained research assistants using TBI Model System standard procedures.

Results: OSA was prevalent using the AASM (66%) and CMS (41.5%) criteria with moderate to strong agreement (weighted kappa = 0.64 (95%CI = 0.58, 0.70). Significant differences were observed for participants meeting AASM and CMS criteria (Agreement Group; AG) compared to those meeting criteria for AASM but not CMS (Disagreement Group; DG). At AHI \geq 5, the DG (n=61) had lower Emergency Department Glasgow Coma Scale Scores consistent with greater injury severity (median 5 vs. 13, p = 0.0050), younger age (median 38 vs 58, p<0.0001), and lower BMI (median 24.8 vs 22.1, p = 0.0007) compared to the AG (n=103). At AHI \geq 15, female gender and but no other differences were noted possibly due to the smaller sample size.

Conclusion: The underestimation of sleep apnea using CMS criteria is consistent with prior literature; however, this is the first study to report the impact of the criteria in persons with moderate to severe TBI during a critical stage of neural recovery. Management of comorbidities in TBI has become an increasing focus for optimizing TBI outcomes. Given the chronic morbidity after moderate to severe TBI, the impact of CMS policy for OSA diagnosis for persons with chronic disability and young age are considerable.

Support: PCORI (CER-1511-33005), GDHS (W91YTZ-13-C-0015; HT0014-19-C-0004)) for DVBIC, NIDILRR (NSDC Grant # 90DPTB00070, #90DP0084, 90DPTB0013-01-00, 90DPTB0008, 90DPT80004-02).

0609

SLEEP PHENOTYPES IN MIDDLE-AGED AND OLDER HISPANICS/LATINOS. RESULTS FROM THE HISPANIC COMMUNITY HEALTH STUDY/STUDY OF LATINOS (HCHS/SOL)

Wu, *B.*¹ Tarraf, *W.*² Wallace, D. M.³ Stickel, A.¹ Schneiderman, N.⁴ Redline, S.⁵ Patel, S. R.⁶ Gallo, L. C.⁷ Mossavar-Rahmani, Y.⁸ Daviglus, M.⁹ Zee, P. C.¹⁰ Talavera, G. A.⁷ Sotres-Alvarez, D.¹¹ Gonzalez, H. M.¹ Ramos, A. R.¹²

¹Department of Neurosciences and Shiley-Marcos Alzheimer's Disease Research Center, University of California, San Diego, La Jolla, CA, University of California San Diego, CA, ²Institute of Gerontology & Department of Healthcare Sciences, Wayne State University, Detroit, Michigan, Wayne State University, MI, ³University of Miami, Miller School of Medicine, University of Miami, FL, ⁴University of Miami, Department of Psychology, University of Miami, FL, ⁵Harvard Medical School, Harvard University, MA, ⁶University of Pittsburgh, School of Medicine, University of Pittsburgh, PA, ⁷Institute for Behavioral and Community Health, Graduate School of Public Health, San Diego State University, San Diego, California, San Diego, CA, 8 Albert Einstein College of Medicine, Department of Epidemiology & Population Health, Bronx, NY, 9Institute for Minority Health Research, University of Illinois at Chicago, College of Medicine, Chicago, Illinois, Chicago, IL, ¹⁰Northwestern University, Feinberg School of Medicine, Department of Neurology, Chicago, IL, ¹¹University of North Carolina, Department of Biostatistics, Chapel Hill, NC, ¹²University of Miami, Miller School of Medicine, Miami, FL.

Introduction: Identifying sleep phenotypes in the diverse and understudied US Hispanic/Latino population is critical to developing interventions and mitigating distal clinical outcomes (e.g. dementias).

Methods: Using latent class analyses (LCA), we identify empirically derived and clinically meaningful sleep phenotypes using data on community dwelling middle-aged/older adults (ages \geq 45-years) from the HCHS/SOL (2008-2011) - Investigation of Neurocognitive Aging (n=6,377). Sleep variables used included Apnea/Hypopnea Index (AHI), percent time SpO2<90%, Epworth Sleepiness Scale (ESS), Women's Health Initiative Insomnia Rating Scale (WHIIRS), self-reported average sleep duration, restless legs symptoms, napping frequency, and sleep quality.

Results: Mean (M) age was 56.4 ± 8.1 years, and 54.7% were female. Average AHI, ESS, WHIIRS, and sleep duration were 8.7±13.1, 6.0±5.0, 7.6±5.5, and 7.8±1.4, respectively, and 25.8% had zero percent time SpO2 <90%. Fit statistics indicated that a four-class solution provided the best data fit. The derived classes, adjusting for age, sex, income, and acculturation, corresponded with four clinically meaningful groups: (1) 28.8% were asymptomatic [(M) AHI=0.8; (M) ESS=5.6; (M)WHIIRS=7.6; (M) sleep duration=7.8; 0% SpO2<90%=74.1%], (2) 25.7 % were *asymptomatic mild sleep apnea* [(M) AHI=6.2; (M) ESS=3.8; (M) WHIIRS=2.9; (M) sleep duration=7.8; 0% SpO2<90%=8.8%], (3) 19.4% were symptomatic sleep apnea [(M) AHI=25.6; (M) ESS=8.5; (M) WHIIRS=7.2; (M) sleep duration=7.7; 0% SpO2<90%= 0.5%], and (4) 26.1% were insomnia [(M) AHI=5.7; (M) ESS=6.7; (M) WHIIRS=13.0; (M) sleep duration=7.8; 0% SpO2<90%=10.3%]. Classification into groups 3 and 4 were primarily driven by elevated AHI and WHIIRS scores, respectively. The distribution of scores in the derived groups suggest variations relative to current clinical thresholds.

Conclusion: We identified 4-groups using LCA in a communitybased sample of diverse U.S. Hispanic/Latino adults. Better characterization of sleep phenotypes for Hispanics/Latinos can help in developing targeted interventions studies and ameliorate health disparities.

Support: 5R01AG048642-05; R21AG056952; R21HL140437.

0610

PREVALENCE OF POSITIONAL OBSTRUCTIVE SLEEP APNEA BASED ON 3% VS 4% OXYGEN DESATURATION USING HOME SLEEP APNEA TESTING

Mandal, M. Rengan, R. Rani, S. Ramzy, J. Vega Sanchez, M. Jaffe, F. D'Alonzo, G. Shariff, T. Chatila, W. Weaver, S. Krachman, S.

Lewis Katz School of Medicine at Temple University Hospital, Philadelphia, PA.

Introduction: Approximately 30% of patient with obstructive sleep apnea (OSA) have positional OSA [non-supine apnea-hypopnea index (AHI) < 5 events/hr]. However, the prevalence is based on variable definitions for hypopneas related to the degree of oxygen desaturation. In addition, use of a home sleep apnea test (HSAT) to identify positional OSA is limited. We hypothesized that in patients evaluated with an HSAT, using a definition for hypopneas based on 4% compared to 3% oxygen desaturation will significantly decrease the percentage diagnosed with positional OSA.

Methods: Fourteen patients with positional OSA based on a non-supine respiratory event index (REI) < 5 events/hr were included. The initial diagnosis was determined based on a hypopnea

definition of $\ge 3\%$ oxygen desaturation. The studies were reanalyzed using a hypopnea definition of $\ge 4\%$ oxygen desaturation.

Results: Fourteen patients [9 (64%) males, 46±14 yrs, BMI 31±6 kg/m², ESS 7±5, REI 9±3 events/hr, mean SaO₂ 94±2%, lowest SaO₂ 81±6%, %TST SaO₂ < 90% 4±6%] were identified with positional OSA (supine REI 16±7 events/hr, non-supine REI 3±1 events/hr) using a hypopneas definition of \geq 3% oxygen desaturation. When reanalyzed using a hypopnea \geq 4% oxygen desaturation there was a significant decrease in the REI to 7±2 events/hr (p<0.001). Three patients (21%) no longer were considered to have OSA. These patients were younger (32±14 vs. 50±11yrs, p=0.03) and had less severe OSA (REI 6±1 vs. 9±3 events/ hr (p=0.04), but there was no difference in BMI (32±11 vs. 31±5 kg/m², p=0.9) or mean and lowest SaO₂ (96±0.4 vs. 94±2%, p=0.13, and 82±8 vs. 81±6%, p=0.9, respectively).

Conclusion: In patients with mild positional OSA, using a hypopnea definition of at least 4% vs. 3% oxygen desaturation on a HSAT will have a significant effect on the overall REI and often exclude patients who would otherwise be treated for OSA. **Support:** None.

0611

SCREENING FOR OBSTRUCTIVE SLEEP APNEA IN THE BARIATRIC SURGERY POPULATION

*Kreitinger, K.*¹ *Lui, M. M.*² *Owens, R.*¹ *Schmickl, C.*¹ *Grunvald, E.*¹ *Horgan, S.*¹ *Malhotra, A.*¹

¹UC San Diego, San Diego, CA, ²The University of Hong Kong, Hong Kong, HONG KONG.

Introduction: Obstructive sleep apnea (OSA) is prevalent in the bariatric surgery population and has been associated with increased perioperative risk, especially if OSA is moderate-severe (apnea-hypopnea index \geq 15/h). Consequently, screening for OSA is recommended as part of the preoperative evaluation. Several screening tools for OSA have been developed; however, some tools lack validation and their relative performance is unclear. The purpose of this study was to compare four existing screening tools (Epworth Sleepiness Scale (ESS), STOP-BANG, NO-OSAS, and No-Apnea) with regards to the ability to identify patients with moderate-severe OSA among bariatric surgery patients.

Methods: We retrospectively reviewed data from Jan 2015 to Mar 2019 for adult patients presenting consecutively to UC San Diego for first-time bariatric surgery who had undergone a home or in-lab sleep study (within one year of the initial encounter for bariatric surgery), which is our standard of care. We compared the accuracy of the four screening tools for detecting moderate-severe OSA based on the area under the receiver operating characteristic curves (AUC). Subgroup analyses were explored based on sex, BMI, and ethnicity (Hispanic/Latino vs non-Hispanic/Latino).

Results: Of the 214 patients (83.2% female, median age 39 years) included in the study, 45.4% had moderate-severe OSA. STOP-BANG (AUC 0.75 [95%CI: 0.68 to 0.81]) and NO-OSAS (AUC 0.76 [95%CI: 0.69 to 0.82]) had similar performance (p 0.62); both performed significantly better than the ESS (AUC 0.61 [95%CI: 0.54 to 0.68]; p 0.02 for both). STOP-BANG and NO-OSAS tended to perform better in the female vs male subgroup, but this finding did not reach statistical significance.

Conclusion: STOP-BANG and NO-OSAS are superior to the ESS when screening bariatric surgery patients for moderate-severe OSA. In future analyses we will further explore if adjustments of standard cut-offs improve test characteristics (i.e. sensitivity/specificity) when screening bariatric surgery patients (analyses ongoing). **Support:** None.

0612

VALIDATION OF NEMS-BG SCORE IN SCREENING OF PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Gharraf, H. Baess, A.

Faculty of Medicine, Alexandria, EGYPT.

Introduction: Obstructive sleep apnea (OSA) is one of the most prevalent diseases worldwide. Diagnosis of OSA is still a dilemma despite our well known disease-related impact on human body health. Under-diagnosis of OSA is still a problem despite the well-established clinical and laboratory criteria of diagnosis. Over-diagnosis may lead to exhaustion of our limited health-care resources. Therefore, an efficient screening tool that is well validated and easily applied, will be an ideal solution for the over or under-diagnosis of OSA. NAMES-BG score was suggested to efficiently screen for OSA depending on neck circumference, airway classification, comorbidities, Epworth sleepiness scale, snoring score, body mass index and gender

Methods: The aim of the work was to validate NEMS-BG score in screening of patients with obstructive sleep apnea (OSA).patients and methods. This is a retrospective single center clinical study. Records of patients with documented obstructive sleep apnea were collected. Those records were collected from the database of department of chest diseases in Alexandria Main University Hospital (AMUH). The records of included patients included demographic data, clinical findings, anthropometric measures and polysomnographic records. Statistics were formulated to validate the sensitivity and specificity of this score in our cohort of patients. The significance of the results were at the 5% level of significance.

Results: The cutoff value for the composite NAMES tool was calculated at \geq 3 points. In the validation group, NAMES demonstrated similar test characteristics to the Berlin questionnaire, and sensitivity was statistically significantly better than that seen with the Epworth scale. The addition of BMI and gender to the tool improved screening characteristics

Conclusion: The NAMES assessment is an effective, inexpensive screening strategy for moderate to severe OSA. **Support:** no support

0613

THE EFFECTS OF DIABETES ON OBSTRUCTIVE SLEEP APNEA SEVERITY AND PROGRESSION: A PROPENSITY SCORE ANALYSIS

Li, Q.¹ Keenan, B. T.² Punjabi, N. M.³ Maislin, G.² Kuna, S. T.^{2,4} ¹Department of Respiratory and Critical Care Medicine, Ruijin Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, CHINA, ²Division of Sleep Medicine, Department of Medicine, University of Pennsylvania, Philadelphia, PA, ³Division of Pulmonary and Critical Care Medicine, Johns Hopkins University School of Medicine, Baltimore, MD, ⁴Sleep Medicine Section, Crescenz Veterans Affairs Medical Center, Philadelphia, PA.

Introduction: Obstructive sleep apnea (OSA) is associated with increased glucose intolerance and insulin resistance, and commonly coexists with type 2 diabetes mellitus (T2DM). Research suggests an independent association between these two conditions. However, more research into the role of T2DM in the development of OSA, or vice versa, is needed.

Methods: Leveraging data from 139 participants with T2DM from the Sleep AHEAD cohort and 5,085 participants without T2DM

from the Sleep Heart Health Study (SHHS), we conducted two complementary propensity score (PS) subclassification analyses to estimate the effect of T2DM on the oxygen desaturation index (ODI), both at baseline and over 4-5 years of follow-up. PS models included age, sex, race, body mass index, neck circumference, waist circumference, total cholesterol, HDL cholesterol, triglycerides, Epworth Sleepiness Scale, and comorbid hypertension or cardiovascular disease. Models evaluating ODI progression also included baseline ODI.

Results: The PS subclassification analysis identified 109 participants with T2DM and 480 without T2DM, balanced with respect to baseline covariates, for evaluation of the effect of T2DM on baseline ODI. On average, those with T2DM had a 9 events/hour greater ODI compared to those without (24.3 [20.8, 27.9] vs. 15.3 [13.6, 16.9] events/hour; p<0.0001). Among patients with baseline ODI≥5, a second PS subclassification identified 99 with T2DM and 227 without for evaluating the effect of T2DM on ODI progression. No difference in change in ODI was observed between those with and without T2DM (mean [95% CI] difference -0.25 [-10.7, 10.2] events/hour; p=0.963).

Conclusion: Using two robust PS subclassification designs to minimize selection bias, we evaluated the effect of T2DM on baseline ODI and ODI progression in adults with OSA after 4-5 years. Those with T2DM had more severe baseline ODI, but there were no meaningful differences in ODI progression. Results further our understanding of the association between these coexisting conditions.

Support: NIH grant HL070301

0614

DIURNAL PATTERNING OF AUTONOMIC MEASURES IN SLEEP APNEA AND PAROXYSMAL ATRIAL FIBRILLATION AND RESPONSE TO CONTINUOUS POSITIVE AIRWAY PRESSURE THERAPY

Sandhu, A.¹ Wang, L.² Bena, J.² Kaffashi, F.³ Loparo, K.³ Aylor, J.¹ Nawabit, R.¹ Chung, M.⁴ Van Wagoner, D.⁵ Walia, H.¹ Mehra, R.¹ ¹Sleep Disorders Center, Neurological Institute, Cleveland Clinic, Cleveland, OH, ²Lerner Research Institute, Cleveland Clinic, Cleveland, OH, ³School of Engineering, Case Western Reserve University, Cleveland, OH, ⁴Heart and Vascular Institute, Cleveland Clinic, Cleveland, OH, ⁵Department of Molecular Cardiology, Lerner Research Institute, Cleveland Clinic, Cleveland, OH.

Introduction: Diurnal patterning of autonomic function in paroxysmal atrial fibrillation (PAF) and sleep disordered breathing (SDB) is unknown. We hypothesize heart rate variability (HRV) as surrogates of autonomic function, exhibit diurnal differences in PAF relative to SDB severity and treatment.

Methods: We leveraged the Sleep Apnea and Atrial Fibrillation Biomarkers and Electrophysiologic Atrial Triggers (SAFEBEAT,NCT02576587) study focused on participants with PAF and SDB (apnea hypopnea index,AHI≥15,3% oxygen desaturation hypopnea). Attended 16-channel polysomnography (PSG) and continuous ECG monitoring (Heartrak Telemetry®) for 7-21 days was performed at baseline and after 3-months of continuous positive airway pressure (CPAP). Linear mixed-effects models (least square means,95%CI) were used to assess relationships between daily average HRV measures (frequency domain:LF,HF,LF/HF;time domain:MNN,RMSSD,SD1,SD ratio and novel non-linear:DFA-alpha measures) with SDB (AHI),%sleep time with SaO2<90%(TRT<90): per 5-unit

increase),effect of 3-month CPAP relative to sleep-wake and statistical interaction of sleep-wake. Analyses were conducted using SAS version v.9.4, Cary, NC.

Results: The analytic sample was comprised of 33 cases with PAF and SDB:61.1±11.7 years,62.5% male, BMI:33.9±7.2kg/ m²,75% Caucasian,AHI 15.1 (IQR: 4.4,29.4) and 68.8% on atrioventricular nodal blocking medications. AHI was associated with frequency (HF:0.08[0.01.0.16] and LF/HF:-0.11[-0.20, -0.01]), time (SD1:0.08[0.02,0.14] and SD ratio: 0.09[0.04,0.14]) and non-linear (DFA-alpha1: -0.02[-0.036,-0.003]) domain measures during wake, but not sleep. Significant sleep-wake and AHI as well as TRT<90 interactions relative to HRV measures were observed (p≤0.001). Only SD ratio was associated with TRT<90 (0.12[0.03,0.24]). Baseline to follow-up CPAP time domain measures were altered mainly during wake versus sleep with MNN increased 0.13: [0.08,0.19],p<0.001; RMSSD increased 0.13 [0.08,0.19], p<0.001; SD1 increased 30% [0.09,0.55], p=0.004; SD ratio increased 20% [0.01,0.43], p=0.033,and also frequency domain: HF increased 33%[0.03,0.72], p=0.028.

Conclusion: SDB defined by AHI--more so than nocturnal hypoxia--was associated with surrogate autonomic measures impacted by CPAP intervention during wake and not sleep in PAF. SDB-related autonomic influences in PAF appear to be more pronounced during wakefulness suggesting long-term potentiation-like influences.

Support: This study was supported by the National Heart, Lung and Blood Institute (NHLBI) [Grant R01 HL108493] and National Institutes of Health (NIH) National Center for Research Resources [Grant UL1 RR024989]

0615

THE PREVALENCE OF COMMON SLEEP DISORDERS IN YOUNG ADULTS: A POPULATION-BASED STUDY

Eastwood, P. R.¹ Ward, S. V.² Bucks, R. S.³ Maddison, K.¹ Smith, A.⁵ Huang, R.⁶ Pennell, C. E.⁷ Hillman, D. R.¹ McArdle, N.¹ ¹University of Western Australia, Centre for Sleep Science, Perth, AUSTRALIA, ²University of Western Australia, Centre for Genetic Origins of Health and Disease, Perth, AUSTRALIA, ³University of Western Australia, School of Psychological Science, Perth, AUSTRALIA, ⁴University of Western Australia, Centre for Sleep Science, Perth, AUSTRALIA, ⁵Curtin University, School of Physiotherapy and Exercise Science, Perth, AUSTRALIA, ⁶Perth Childrens Hospital, Department of Endocrinology, Perth, AUSTRALIA, ⁷University of Newcastle, Newcastle, AUSTRALIA.

Introduction: Adult sleep disorders are associated with adverse health effects including reduced quality of life and increased mortality. However, there is a paucity of data on sleep disorders in young adulthood.

Methods: We undertook a cross-sectional observational study of 1,227 young adults participating in the Western Australian Pregnancy (Raine) Study (2012-2014) to describe the prevalence of common sleep disorders in young adults. We used in-laboratory polysomnography (PSG) and validated survey methods, including the Epworth Sleepiness Scale (ESS), Pittsburgh Sleep Symptom Questionnaire-Insomnia and International Restless Legs Syndrome Study Group criteria. 1,146 completed a core questionnaire, 1,051 completed a sleep-focused questionnaire and 935 had analysable PSG data.

Results: Participants had a mean age of 22.2 years and male to female ratio of 1 to 1.1. The prevalence of OSA [apnoea hypopnoea

index (AHI): \geq 5events/hour] was 20.8% (95%CI: 18.2 to 23.6) and this was usually of mild severity (AHI: \geq 5 to<15events/hour, 17.1%). OSA syndrome (AHI \geq 5 events/hour and ESS \geq 11) was found in 2.8% (95%CI: 1.9 to 4.1). Chronic insomnia was present in 16.6% (95%CI: 14.7 to 19.4). Restless legs syndrome was present in 2.9% (95%CI: 2.0 to 4.0) and abnormal periodic leg movements during sleep (>5 movements/hour) in 9.1% (95%CI: 7.3 to 11.1). In those participants who had complete data on all sleep-related assessments (n=841), at least one sleep disorder was present in 42.6%. **Conclusion:** Sleep disorders are very common in young adults. Health practitioners should be aware of these high prevalences, as early identification and treatment can improve quality of life and may reduce later morbidity and mortality. **Support:**

0616

VALIDATION OF A SLEEP HEADBAND FOR DETECTING OBSTRUCTIVE SLEEP APNEA

Guillot, A.¹ Moutakanni, T.¹ Harris, M.² Arnal, P. J.² Thorey, V.¹ ¹Dreem Algorithms Team, Paris, FRANCE, ²Dreem Science Team, New York, NY.

Introduction: Polysomnography (PSG) is the gold-standard to diagnose obstructive sleep apnea (OSA). OSA severity diagnosis is defined by the apnea-hypopnea index (AHI) defined as the number of apnea and hypopnea events measured per hour of sleep. The Dreem2 headband (DH) is a self-administered, easy to use device that measure EEG, breathing frequency, heart rate and sound at-home. In our study, we assessed the performance of the DH to automatically detects OSA compared to 3 sleep's experts scoring on PSG.

Methods: 41 subjects (8 females, 42.6 ± 13.7 y.o.) having a suspicion of OSA performed a night at-home wearing both a PSG and the DH. Each PSG record was scored for apnea and hypopnea events by 3 independent trained sleep experts following AASM guidelines. The deep learning approach DOSED, was trained on the DH signals using the manual apnea scoring. 10-fold cross-validation was used to provide predictions for each of the 41 subjects with the DH.

Results: We observed an average AHI expert's scoring of 13.6 ± 10.1 CI[10.5, 16.5] compared to 12.9 ± 10.3 CI[9.6, 15.8] for the DH. Both, the correlation between the 3 scorers (r= 0.88, p < 0.001) and the DH and the scorers (r=0.79, p< 0.001) were significant. The specificity and sensitivity to detect mild OSA (AHI \leq 5) was 84.4 % and 96.4 % for the DH and 86.5 % and 86.0% for the scorers.

Conclusion: The results show that the DH using deep learning can detect OSA with an accuracy similar to the sleep experts. The use of DH paves the way for longitudinal monitoring of patients with a suspicion of OSA and its accessibility could lead to better screening of the general population.

Support: This Study has been supported by Dreem sas.

0617

OPTIMIZATION OF OSA SCREENING IN A BLACK POPULATION

Fossum, M. Najimi, N. Whitesell, P. Howard University, Washington, DC.

Introduction: Racial disparities in the prevalence and severity of Obstructive Sleep Apnea (OSA) in Black Americans may play an important contributory role in the increased burden of cardiometabolic disease experienced by this population. Effective screening for OSA could permit early recognition and treatment, thereby preventing future the adverse cardiovascular outcomes which contribute to a decreased life expectancy. The STOP-BANG questionnaire is a validated screening tool for OSA as demonstrated in many populations and settings. Unfortunately, Blacks have typically been underrepresented in studies evaluating the sensitivity and specificity of the tool. Potentially unique biologic and psychosocial factors may affect the phenotypic expression of OSA and influence the performance of the STOP-BANG. Data are needed regarding the reliability of the tool for this population and potentially modifications to optimize its usefulness.

Methods: Data were from 204 patients evaluated at the Howard University Hospital Sleep Disorder Center between April 2018 and June 2019. Records were reviewed for demographic information, body mass index (BMI), neck circumference, and medical history, including the presence of snoring, tiredness/sleepiness, observed apneas, and hypertension and results of sleep study testing. Data were analyzed using SPSS software. Subcomponents of the STOP-BANG scoring criteria were adjusted to improve performance.

Results: Mean age was 48.5. 62% of subjects were female and mean BMI was 40.2 The mean ESS was 9.9. The mean AHI was 27.7 with a prevalence of mild or greater OSA (AHI >5) of 80%. Scoring of the STOP-BANG using a criterion of 3 or greater as indicating increased risk demonstrated a sensitivity of 90%, specificity of 8%, PPV of 80%, and NPV of 17%. Performance was worse for women then men. Multiple adjustments to scoring were evaluated to improve accuracy.

Conclusion: Similar to its performance in other populations, the STOP-BANG demonstrated high sensitivity but very poor specificity and overall poor accuracy in a Black population referred for Sleep Medicine Consultation to an urban university hospital. Performance was worse for women than men. Modifications are presented to try to improve performance and the clinical utility for this population.

Support: Howard University RCMI

0618

WHAT ARE THE BENEFITS OF REMOTE MONITORING POLYSOMNOGRAPHY

Yagi, T.¹ Chiba, S.^{1,2} Ito, H.²

¹Ota Memorial Sleep Center, Kanagawa, JAPAN, ²The Jikei University Hospital, Tokyo, JAPAN.

Introduction: The use of information and communication technology (ICT) for sleep testing is mainly aimed at improving the accuracy of out-of-center sleep testing (OCST) by remote monitoring. In this study, as the first achievement in Japan, we report the results of our sleep medical clinic and hospital unit. For the diagnosis of sleep disorders, monitoring polysomnography (PSG) attending sleep technologist is the gold standard and is positioned as Type I. On the other hand, diagnosis using OCST has become acceptable because many patients can be diagnosed quickly and cost can be reduced.When using Type II devices that measure electroencephalogram at home, the measurement accuracy is inevitable, including poor recording, because it is performed in a nonmonitoring situation. As an attempt to improve this situation, our clinic and hospital unit have established a remote monitoring PSG system that can be upgraded from Type II to Type I level by remote monitoring by a sleep technologist to ensure recording accuracy. Methods: During the period from April 2004 to December 2017, a total of 286 remote monitoring PSGs were performed by dedicated

sleep technologists at the Ota Memorial Sleep Center for patients admitted to a private room at Ota General Hospital.

Results: The breakdown of the reasons for requesting remote monitoring tests is about 30% of patients scheduled to undergo surgerysuch as palatine tonsillectomy or soft palate plastic surgery the next day, and 24% of hospitalized patients with risky complications %, 17% of patients expected to have a high probability of nighttime seizures and abnormal behavior, and 15% were physically disabled or paralyzed.

Conclusion: Our remote monitoring PSG system is effectively used in the clinic for the general hospital for patients who need nighttime safety management and nursing management. **Support:** non

0619

TO RELY OR NO TO RELY: UNDERSTANDING THE DEMOGRAPHICS AND POLYSOMNOGRAPHIC FEATURES OF FALSE NEGATIVE HOME SLEEP APNEA TESTING

Bollu, P.¹ Gurung, P.¹ Mehta, T.¹ Monegro, A.¹ Manjamalai, S.¹ Goyal, M.¹ Thakkar, M.¹ Sahota, P.¹

¹University of Missouri, Columbia, MO, ²University of Missouri, Columbia, MO.

Introduction: The current gold standard for a definitive diagnosis of OSA is an in-center Polysomnography (PSG). Home Sleep Apnea Testing(HSAT) has become an important tool in identifying high-risk populations. One of the limitations of the study is the lack of Electroencephalographic (EEG) data. This prevents the inclusion of Respiratory Effort Related Arousals (RERAs). We attempted to identify the patients whose HSAT showed an REI of less than 5 but are at risk for having sleep apnea based on the presence of airflow and thoraco-abdominal fluctuations.

Methods: Patients in this study were those that underwent HSAT from September 2016 till June of 2019. The studies reviewed and interpreted by board certified Sleep Specialists. Studies were done using nox-T3 sleep monitor and Nomad portable Home Sleep Testing type III devices-Both are type 3 Portable Monitors. Only those patients whose REI in their HSAT less than 5 were included in this study. All these patients had multiple **airflow fluctuations** in their HSAT that raised the suspicion for the presence of RERAs. None of these patients had significant hypoxemia in the HSAT.Airflow fluctuations were defined by the presence of fluctuations in the signal in the airflow channel along with increasing thoracoabdominal channels. Those patients with REI of less than 5 and without airflow fluctuations were excluded from the study.

Results: A total of 178 patients were recommended to undergo an in-center polysomnogram. Of those, 92 patients completed their polysomnogram with 59 patients ending up with a diagnosis of sleep apnea while 33 did not suggesting a false negative rate of 64.13%. Of those who were positive, 39 were females while 20 were males. Both groups did not differ significantly. Females had a median BMI of 32.9(28.19 for males), a median ESS of 11(8 in males) and a median RDI of 14.8(13.25).

Conclusion: Our study shows that both Home Sleep apnea testing can have a high proportion of false-negative results in patients exhibiting thoraco-abdominal and airflow fluctuations. The interpreting physicians should understand the limitations of the HSAT and should have a low threshold to recommend an in-center polysomnogram. **Support:** None.

0620

CHARACTERISTICS DISTINGUISHING SPECIAL OPERATIONAL FORCES (SOF) PERSONNEL FROM NON-SOF PEERS WITH TBI: RETROSPECTIVE ANALYSIS FROM THE VA TBI MODEL SYSTEMS

Kretzmer, T.¹ Bajor, L.¹ Silva, M. A.¹ Eapen, B.³ McKenzie-Hartmann, T.⁴ Garcia, A.⁴ Belanger, H.⁵ Richardson, R.¹ ¹Mental Health and Behavioral Sciences Section (MHBSS), James A. Haley Veterans' Hospital, Tampa, FL, ²Mental Health and Behavioral Sciences Section (MHBSS), James A. Haley Veterans' Hospital, Tampa, FL, ³7 Physical Medicine and Rehabilitation Section, South Texas Veterans Health Care System, San Antonio, TX, ⁴Defense and Veterans Brain Injury Center, James A. Haley Veterans' Hospital, Tampa, FL, ⁵Defense and Veterans Brain Injury Center (DVBIC), United States Special Operations Command MacDill AFB, Tampa, FL, ⁶Mental Health and Behavioral Sciences Section (MHBSS), James A. Haley Veterans' Hospital, Tampa, FL.

Introduction: Special Operations Forces (SOF) is an umbrella term which encompasses over a dozen specialized communities across all military branches. Little is known about potential differences in demographic and health characteristics, including sleep, between SOF vs. non-SOF service members. We leveraged existing longitudinal studies of those with history of TBI to examine differences between SOF and non-SOF in the dataset.

Methods: We conducted a retrospective analysis of data from the VA TBI Model Systems, a multi-center longitudinal study of outcomes following TBI rehabilitation. Participants were included if SOF status was known (N = 261). Differences between groups on variables of interest were then classified as "Immaterial", "Minor," and "Important" based on either prevalence (categorical data) or degree of difference (continuous data).

Results: Of included participants, 68 (26%) were identified as SOF and 193 (74%) as non-SOF. SOF were more highly educated and more likely to have history of mild TBI. There were multiple "important" differences in co-morbidity prevalence. SOF participants were more likely to be diagnosed with sleep apnea (36% SOF vs 12% non-SOF). They were also more likely to have been diagnosed with chronic pain, a cardiac condition, high blood cholesterol, and/ or osteoarthritis.

Conclusion: SOF participants differed from non-SOF in a multiple important ways, suggesting this is a different and medically complex population. The most striking finding was that SOF personnel had a significantly greater rate of sleep apnea, relative to non-SOF. The mechanism underlying this difference is not known but may relate to training, blast exposure, weapons use, and mission demands. Further investigation regarding mechanisms, prevalence, and treatment of OSA in the SOF community is needed.

Support: This research was sponsored by VHA Central Office VA TBI Model Systems Program of Research; Subcontract from General Dynamics Information Technology (W91YTZ-13-C-0015; HT0014-19-C-0004).

0621

UTILIZATION OF THE ARES TO PREDICT OSA AMONG BLACKS USING HOME-BASED WATCHPAT RECORDING

Rogers, A.¹ Seixas, A.² Moore, J.² Zizi, F.² Williams, S.²

Gyamfi, L.² Pichardo, Y.² Jean-Louis, G.²

¹St. John's University, Queens, NY, ²NYU Grossman School of Medicine, New York, NY.

Introduction: In two waves of data we collected in Brooklyn New York, we observed blacks were at high risk for obstructive sleep apnea (OSA). In the NIH-funded study 'Metabolic Syndrome Outcome Study (MetSO), blacks enrolled from primary-care settings had a 59% risk of OSA. Similarly, blacks surveyed in churches and barbershops had a 43% risk of OSA. While these studies showed higher than expected risk as noted in the general population (29%), it remains uncertain how many of those blacks would be diagnosed with OSA in that population. The purpose of this study was to explore the rate of OSA using the WatchPat device in a community-based setting.

Methods: Data were collected from an NIH-funded study 'Peer-Enhanced Education to Reduce Sleep Ethnic Disparities, designed to navigate blacks at risk of OSA to receive timely diagnosis and treatment using peer-delivered linguistically and culturally tailored sleep health education. Blacks were screened for OSA using the Apnea Risk Evaluation System (ARES) Questionnaire; a score ≥ 6 denoted moderate-high OSA risk. Individuals were asked to wear the WatchPAT 200 for one night during a week-long sleep assessment. WatchPat 200 measures SaO2 to determine respiratory-related arousals, defined as an Apnea-Hypopnea Index (AHI) ≥ 5 , which is used to identify and diagnose OSA. We used SPSS 25.0 to perform logical regression analysis to assess associations between ARES and WatchPat AHI.

Results: A sample of 111 blacks provided valid ARES and WatchPat data for the present analyses. Of the sample, the mean age was 62.26 (SD=13.52 years; female = 55%); 49% reported annual income >20K and 79.5% reported a high school education. Moreover, 27% reported high blood pressure, 13%, diabetes, and 65% were overweight/ obese. Multivariate-adjusted logical regression analyses indicated that blacks at risk for OSA were 66% more likely to receive an OSA diagnosis based on WatchPat AHI data (OR = 1.662, p < 0.01). The model adjusted for age, sex, income, and education.

Conclusion: The present study demonstrated that blacks at risk for OSA at the community level have a significant likelihood of receiving an OSA diagnosis using home-based recordings.

Support: NIH Support (T32HL129953, RO1MD007716, K01HL135452 and K07AG052685).

0622

IN-DEPTH SURVEILLANCE OF CENTRAL SLEEP APNEA IN PATIENTS WITH STABLE HEART FAILURE

Ibrahim, S. Wharton, R. Harmon, E. Bonner, H. Davis, E. Cho, Y. Mazimba, S. Kwon, Y.

University of Virginia, Charlottesville, VA.

Introduction: Central sleep apnea (CSA) is unique sleep breathing phenotype in patients with advanced chronic heart failure (HF) and portend poor prognosis. The prevalence of CSA in HF patients under contemporary therapy is uncertain.

Methods: We reviewed consecutive HF patients on optimal medical therapy who underwent clinically indicated diagnostic in-lab polysomnography at a single academic center. Age, sex and BMI matched patients without HF were selected from sleep clinic as a control. Patients with atrial fibrillation were excluded from this study. Apnea subtypes were determined after careful scoring and confirmation by sleep physicians. 'Any CSA' was defined by central apnea index (CAI) >5 and >1/hr. 'True CSA' was defined if met both CAI \geq 5/ hr and > obstructive apnea index (OAI). Obstructive sleep apnea (OSA) was defined if apnea hypopnea index >15 and OAI>CAI. Multivariate analysis was performed using logistic regression adjusting for age, sex, HF and systolic dysfunction as appropriate.

Results: In patients with HF (N=95, mean age 59, female: 50%), CSA was low and was comparable to control group (N=94) (HF

vs. Non-HF; CSA: 5.3 vs. 4.3%, P=NS; Any CSA 14.7 vs. 17%, P=NS). Only 3 patients with HF had true CSA. In contrast, OSA was common in both groups regardless of obesity status (52.3 vs. 55.3%). In patients with HF, Cheyne Stokes respiration was more frequent in patients with Any CSA vs. without Any CSA (13.3 vs. 3.8%, p=0.04). In multivariate analysis, presence of OSA, but not HF, was associated with Any CSA in entire cohort (Any CSA OR: 3.1 [1.3, 8.1], p=0.02). In patients with HF, male sex was associated with Any CSA (OR: 5.3 [1.1, 40.8], p=0.05). Exclusion of patients with high BMI did not change the results.

Conclusion: CSA was rare in patients with stable HF on contemporary optimal medical therapy. **Support:** None

Support: None

0623

RESPIRATORY PROFILING OF COMMUNITY-DWELLING INDIVIDUALS MANY YEARS AFTER POLIO INFECTION

Li, X.¹ Wang, M.¹ Wang, J.¹ Sun, T.¹ Sun, Y.¹ Dong, X.² Li, J.² Zhao, L.² Zhang, X.² Lv, C.¹ Strohl, K.³ Han, F.²

¹Binzhou Medical University Hospital, binzhou, CHINA, ²Peking University People's Hospital, Beijing, CHINA, ³Case Western Reserve University, Cleveland, OH.

Introduction: To determine the presence of respiratory impairment in community-living subjects with a history of poliomyelitis.

Methods: In a study conducted from July 2013-January 2014, we used a national database to recruit individuals (212 males, 86 females) from north and south provinces in China with a known prior poliomyelitis infection >25 years previously. 298 subjects (96.8%) completed overnight oximetry to collect the number/hr of drops in oxygen saturation >4% (ODI₄) and Epworth Sleepiness Scale (ESS); many completed a metabolic and lipid panel, arterial blood gas analysis, chest x-ray (CXR), spirometry and maximal voluntary ventilation (MVV). Some completed risk profiling for OSA.

Results: Age was 47.8 ± 6.7 years (M±SD) and, when known, the age of infection was 2.3 ± 1.8 yrs. As defined by ODI₄ ≥ 5/ hr, the frequency of sleep-disordered breathing (SDB) was 37.2%; 9% (n = 27) had an ODI₄ ≥15/hr. Those with vs. those without SDB differed by male gender (81% vs 65%, p = 0.004) and BMI (25.9 vs 23.0kg/m², p < 0.001). ESS was within the normal range, but was higher in those with 6.8 ± 5.0 vs. without 5.2 ± 4.0 (p < 0.01) SDB. Spirometry and MVV revealed no differences among groups. Scoliosis on the CXR was present 26.1% of those with and 14.4% of those without SDB. ODI₄ correlated weakly with body mass index (r = 0.40). Glucose levels or hyperglycemia were not different. A triglyceride level > 1.7 mmol/L was present in 47.2% of those with and 21.3% of those without SDB (p < 0.001).

Conclusion: Mild (28%) and to a smaller extent moderate-severe (9%) SDB is present many years after surviving poliomyelitis in infancy. In most, sleepiness is low but scoliosis is often present, and associations to hyperlipidemia and obesity are not reflected in fasting glucose levels.

Support: no

0624

VOICE OF THE PATIENT: A PATIENT-CENTERED EXPLORATION OF FACTORS INFLUENCING OBSTRUCTIVE SLEEP APNEA CARE

Puravath, F. M.¹ Ash, T.² Rottapel, R.³ Spadola, C.⁴ Bandana, S.⁵ Schonberg, M.⁶ Redline, S.³ Bertisch, S.⁷

¹University of Texas at Houston, Houston, TX, ²Brown University, Providence, RI, ³Brigham and Women's Hospital Harvard Medical School, Boston, MA, ⁴Florida Atlantic University, Boca Raton, FL, ⁵University of Sydney, Sydney, AUSTRALIA, ⁶Harvard Medical School, Boston, MA, ⁷Brigham and Women's Hospital, Harvard Medical School, Boston, TX.

Introduction: Despite widely available efficacious treatments for obstructive sleep apnea (OSA), patients commonly report frustration in accessing and adhering to treatments. Sparse research has explored factors influencing OSA care from the patient perspective, which may limit provision of patient-centered care: care responsive to patient preferences, needs, and values. To this end, we conducted qualitative research to identify factors, voiced by patients, that influence OSA treatment initiation and adherence.

Methods: We performed semi-structured interviews with 15 patients previously diagnosed with OSA from Boston, MA and a national patient portal (MyApnea.Org). Patients were asked about barriers and facilitators to their diagnosis and treatment as well as about their preferences and values that informed their treatment decisions. Interviews were audio-recorded and transcribed. A qualitative content analysis was performed to identify themes. After developing a codebook, interviews were coded. Codes were then audited and finalized by study team consensus.

Results: Our sample was aged 25-74 years; 71% identified as female. Among participants, 57.1% identified as White, 14.3% Black, 14.3% Asian, and 14.3% Other. Major themes were broadly classified as (1) facilitators (provision of useful information on treatment options, participation in shared decision-making, continued clinician support); (2) barriers (inconvenience of treatment, difficulty of habit formation, treatment side effects, competing comorbid conditions); (3) motivators (value of improving chronic health, family support, positive treatment effects); (4) contextual factors (insufficient knowledge/awareness of OSA, navigating healthcare systems, access to informational resources). Awareness of OSA symptoms and treatments, and ongoing support were cited as the most common factors influencing the patient experience.

Conclusion: This formative research highlights that diverse factors impact the OSA evaluation and treatment patient experience. Further research should test interventions that promote effective patient-centered care for OSA, such as shared decision-making tools.

Support: Brigham and Women's Hospital Research Institute Patient-Centered Comparative Effectiveness Research Center Grant

0625

THE EFFECTS OF TOPICAL NASAL STEROIDS ON CONTINUOUS POSITIVE AIRWAY PRESSURE COMPLIANCE IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Segsarnviriya, C. Mahakit, P.

Department of Otolaryngology, Phramongkutklao Hospital, Bangkok, THAILAND.

Introduction: Continuous positive airway pressure (CPAP) for treatment of moderate to severe obstructive sleep apnea can produce troublesome nasal symptoms (i.e. congestion, rhinorrhea, sneezing, itching) that may affect the continuity and decrease the compliance of CPAP. Topical nasal steroids are often prescribed to reduce these side effects, although recent studies are scarce

supporting any benefit of this treatment for CPAP-induced nasal side effects.

Methods: 80 patients who were previously diagnosed with OSA and pretreatment nasal symptoms were enrolled. All of them were selected for CPAP treatment and divided randomly into two groups. The study group was prescribed Fluticasone furoate nasal spray 55 ug daily before bedtime. The patients' compliance to CPAP was recorded by the memory card in CPAP device. Total nasal symptom score was assessed using a questionnaire by direct interview. The follow-up was performed in 30 and 90 days after treatment.

Results: The compliance to CPAP (Percent days with usage, Average time usage and Percent of days with usage ≥ 4 hours) increased in both groups with higher significance in topical nasal steroid group (P-value=0.002, 0.001, 0.002) after 90 days of treatment. There was no difference in nasal symptom between both groups after 30 days of treatment. However, addition of topical nasal steroid resulted in decreased rhinorrhea and congestion symptom (P-value <0.001, <0.001) after 90 days of treatment.

Conclusion: The addition of topical nasal steroid decreased the frequency of nasal symptoms (esp. rhinorrhea and congestion) in OSA patients initiating CPAP therapy and increased the compliance to CPAP after 90 days of treatment.

Support: Keywords: Obstructive sleep apnea, Continuous positive airway pressure, CPAP, Topical nasal steroid

0626

WITHDRAWN

0627

COGNITIVE PERCEPTIONS IMPACT SHORT-TERM CPAP ADHERENCE IN ASIAN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Png, C. A.¹ Liang, J.¹ Mok, Y.¹ Chang, J.¹ ¹Changi General Hospital, Singapore, SINGAPORE, ²Changi General Hospital, Singapore, SINGAPORE.

Introduction: Adherence for the gold standard Continuous Positive Airway Pressure (CPAP) treatment for obstructive sleep apnea (OSA) is poor worldwide. Studies have explored factors impacting CPAP adherence but data is limited for Asian patients where cultural and social norms differ. This study aimed to examine the role of disease and treatment-related perceptions in short-term CPAP adherence among patients from a multi-ethnic Southeast Asian country.

Methods: 34 patients with newly diagnosed OSA were recruited from Changi General Hospital, a 1000-bed tertiary hospital in Singapore between September 2018 and February 2019. Psychological factors of self-efficacy, risk-perception and outcome expectancies were assessed with the Self-Efficacy Measure for Sleep Apnea (SEMSA) questionnaire. The SEMSA questionnaire has been previously validated for the evaluation of adherence-related cognitions. Patients were administered the SEMSA questionnaire before commencement of CPAP treatment and 1 month after.

Results: 73.5% (25/34) of the patients were male (82.4% Chinese, 11.8% Malays, 2.9% Indians, 2.9% others). Mean age was 43.3 \pm 11.8 years, mean apnea hypopnea index (AHI) was 45.2 \pm 29.6 events/hr and mean CPAP usage at one month was 3.6 \pm 2.0 hours. 47% were adherent to CPAP, defined as average device use > 4 hrs/ day. Pre-treatment self-efficacy was significantly correlated with CPAP adherence (r = 0.498, P<0.01). Outcome expectancies and self-efficacy measured after one-month CPAP use were significantly

correlated with CPAP adherence (r = 0.702, P<0.01; r = 0.467, P<0.01, respectively). However, no association between risk perception and CPAP adherence was noted at either time points.

Conclusion: Consistent with previous literature in Western population, our study demonstrated that patients' cognitive perceptions of outcome expectancies and sense of self-efficacy have an impact on CPAP adherence in a Southeast Asian population. Strategies targeting these aspects would be important in designing patient education programs.

Support:

0628

AN OBSERVATIONAL REPORT OF CLINICAL OUTCOMES AMONG VETERANS WITH OBSTRUCTIVE SLEEP APNEA TREATED WITH HYPOGLOSSAL NERVE STIMULATION

*Raman, S.*¹ *Fernandez, A. D.*² *Mathew, E. V.*¹ *Hudgins, L. E.*¹ *Cornman, E.*¹

¹Hunter-Holmes McGuire Veterans Affairs Medical Center, Richmond, VA, ²VCU School of Medicine, Richmond, VA.

Introduction: The efficacy of positive airway pressure (PAP) therapy in the management of obstructive sleep apnea (OSA) is limited by inadequate patient adherence. Hypoglossal nerve stimulation (HNS) is an FDA approved PAP-alternate surgical treatment option that is available for patients with poor PAP tolerance. The purpose of this pilot study was to review outcome measures and adherence data for patients treated with HNS therapy in a military veteran population.

Methods: Methodology involved a retrospective chart review of 30 PAP intolerant cases referred for HNS treatment over the course of 2 years. The inclusion criteria were Body Mass Index (BMI) < 35 kg/m², Apnea Hypopnea Index (AHI) of 15-65/hour and drug induced sleep endoscopy (DISE) showing antero-posterior pharyngeal collapse. Qualified veterans proceeded to HNS implantation. Variables for analysis included BMI, Epworth sleep scale (ESS), DISE results, pre- and post-treatment AHI and adherence data.

Results: Of the 30 veterans, 17 proceeded to DISE. 15 had partial or complete AP collapse and 2 had concentric collapse. 12 veterans proceeded to HNS implantation. 6 veterans who completed HNS titration studies showed an average improvement of 77% from baseline AHI. There was significant decrease in mean AHI from 43.95/hour to 10.52/hour. The mean ESS decreased from 13 to 11. The mean compliance was 6.66 hours/night.

Conclusion: This limited veteran observational study supports that HNS is an effective treatment option for the management of PAP-intolerant patients with OSA. Our preliminary data suggests improved treatment adherence. Future prospective large-scale cohort studies should be considered.

Support: The authors declare no conflicts of interest nor any financial support

0629

FEASIBILITY OF OBSERVED BUT PHYSICALLY UNAIDED CONTINUOUS POSITIVE AIRWAY PRESSURE SET UPS

Hevener, W.^{1,2} Barnes, F.^{1,2} Munafo, D. A.^{1,2}

¹BetterNight, LLC, San Diego, CA, ²Sleep Data Services, LLC, San Diego, CA.

Introduction: We sought to determine if an observed but physically unaided CPAP set up, utilizing only a detailed set of CPAP

instructions without direct involvement of a Respiratory Therapist, could yield similar Medicare compliance rates when compared to the conventional method of hands-on instruction.

Methods: This was a single center, prospective trial. All patients were CPAP naïve. A total of 393 patients completed the physically unaided set ups utilizing a starter kit designed by Resmed (Airsense 10 Autoset, instructions and 3 mask options, P10,N20,F20). Patients were asked to set themselves up under observation with only verbal assistance provided if requested. No patient required conversion to conventional set up. 5287 completed the conventional set ups. We compared the success of observed but unaided CPAP set ups to the success rate for conventional CPAP set ups performed with physical assistance. The primary success endpoint was Medicare compliance within 90 days.

Results: The two groups were not significantly different with regard to gender, age, baseline AHI, and initial mask selection. The Medicare compliance of the unaided set ups was not significantly different from the conventional set ups (75% vs. 77% respectively, p=NS). Similarly, adherence at ten days (65% vs. 69%, p=NS) and thirty days (65% vs. 68%, p=NS) was no different between the two groups and the percentage of mask re-fits required at ten and thirty days was similarly no different.

Conclusion: We conclude that for the CPAP equipment used, observed but physically unaided CPAP set ups can yield similar Medicare compliance rates when compared to physically aided CPAP set ups. Further, we speculate that because these techniques yielded similar success rates the principal factor determining ultimate success is patient follow up and support once therapy is initiated. **Support:** None.

0630

COMPARISON BETWEEN THE USE OF APAP AND MANUAL TITRATION DURING SPLIT NIGHT POLYSOMNOGRAPHY FOR DIAGNOSIS AND TREATMENT OF OSA

Gharraf, H. S. Shaarawy, H. m. Faculty of Medicine, Alexandria, EGYPT.

Introduction: CPAP remains the gold standard treatment for OSA, CPAP titration can be done using manual titration or using APAP devices, CPAP titration can be done using full night or split night protocol. **The aim of the study** Is to compare between the use of APAP and manual titration to determine the needed CPAP pressure during split night polysomnography for diagnosis and treatment of OSA.

Methods: 100 patients with severe OSA were enrolled after exclusion of patients with heart failure or respiratory failure. After diagnostic polysomnography, patients were divided into 2 groups: group1 offered manual CPAP titration and Group2 offered APAP titration, the time for CPAP titration was at least 4 hours in both groups.

Results: both groups were matched as regard age, gender, BMI, sleep parameters and AHI (44.52 \pm 7.81/hour in group1 and 42.66 \pm 9.68/hour in group2 with no statistical significance, after CPAP titration AHI was significantly improved in both groups, the time needed to reach the therapeutic pressure was significantly lower in group2 than in group1, attended technician was needed only in group1.

Conclusion: Use of APAP was equal to manual titration in this group of patients with severe OSA, with decreased cost and lesser time to reach the therapeutic pressure, large multicenter trials are needed to modify the guidelines in view of using APAP in split night protocol for diagnosis and treatment of OSA. **Support:** no conflict of interest

SLEEP, Volume 43, Abstract Supplement, 2020

RESPIRATORY INFECTION RISK IN PAP USERS

Horvat, M. Gavidia, R. Dunietz, G. L. Braley, T. University of Michigan, Ann Arbor, MI.

Introduction: Although positive airway pressure (PAP) effectively treats obstructive sleep apnea (OSA), adherence remains suboptimal for many patients. One factor that may influence a patient's decision to use PAP is a prevailing concern that use of unsanitized PAP equipment may serve as a reservoir for pathogens that cause respiratory tract infections (RTI). Conversely, untreated OSA could also have long-term consequences, including impaired immune function, raising the possibility that PAP therapy could reduce RTI risk. The objective of this study is to compare RTI patterns before and after initiation of PAP in persons with OSA, and examine clinical characteristics that may influence RTI risk.

Methods: Patients with at least 2 years of continuous primary care at Michigan Medicine preceding and following their OSA diagnosis (N=480) were considered eligible for analyses. Medical charts were reviewed to determine pre- and post-PAP RTI frequency and characteristics during this time frame. Change in RTI frequency was examined with paired T-tests.

Results: Male:female ratio was 1:1, mean age 56 years, mean apnea hypopnea index was 10, N=85/480 had documented good PAP compliance (defined as \geq 4h/night, 70% of the nights). Among 480 patients, total RTI frequency pre and post PAP therapy were 170 and 144 respectively (p=0.11). Data collection is ongoing.

Conclusion: These preliminary data suggest that use of PAP is not associated with increased risk of respiratory infections. Further data regarding clinical characteristics that may influence RTI risk in OSA patients will be informed by ongoing data collection and planned multivariable analyses.

Support: Dr. Gavidia is supported by a Training in Clinical and Basic Neuroscience T32 (NIH/NINDS T32 NS 007222)

0632

THE CORRELATION BETWEEN SEVERITY OF OBSTRUCTIVE SLEEP APNEA AND PATIENT COMPLIANCE

Roberts, A. C.¹ Bastin, G.²

¹Parkview Health, Fort Wayne, IN, ²Marian University College of Osteopathic Medicine, Indianapolis, IN.

Introduction: Obstructive sleep apnea (OSA) is a prevalent disorder affecting 9-38% of the global population and is linked to multiple health complications. Continuous Positive Airway Pressure (CPAP) is regarded as the gold standard treatment for OSA, but its efficacy is limited by poor patient compliance. Studies have linked many clinical and lifestyle factors to CPAP adherence, but have produced conflicting outcomes. Based on the current literature, it is assumed patients diagnosed with severe OSA are more likely to be compliant with CPAP due to a greater improvement in quality of life. The goal of this study is to compare the compliance rate of CPAP for patients with mild, moderate, and severe OSA, as well as identify other potential predictors of CPAP compliance. Methods: This study is a retrospective chart review of 100 patients who were newly diagnosed with OSA and started on CPAP between January 1, 2017 and January 1, 2018. Baseline demographic data, past medical history, OSA severity, Epworth sleepiness scale, and compliance to CPAP therapy were recorded. Compliance was defined as CPAP usage greater than four hours per night for at least 21 days per month.

Results: Mean follow-up time after CPAP initiation was 3.19 months. Overall 77% of patients were compliant to CPAP therapy, of which 48% were males and 52% were females (P=0.48). CPAP compliance rates for mild OSA (79.3%), moderate OSA (73.7%), and severe OSA (78.8%) showed no significant difference for independence (P=0.83) or correlation with compliance (P=0.99). Only seasonal allergic rhinitis showed a positive association with CPAP adherence (P=0.031) and depression showed a negative association (P=0.027).

Conclusion: The level of OSA severity is not a significant predictor of short-term CPAP compliance among newly diagnosed patients.

Support: Parkview Physicians Group, Indiana School of Medicine - Fort Wayne, and the Dr. Luis and Anne B Schneider Foundation.

0633

VOLUME ASSURED PRESSURE SUPPORT IS AN EFFECTIVE TREATMENT IN PATIENTS WITH CENTRAL SLEEP APNEA SYNDROME

Levri, J. M.¹ Watanabe, N.¹ Peng, V.² Scharf, S. M.³ Diaz, M.² ¹University of Maryland Medical Center, Baltimore, MD, ²University of Maryland Medical Center, Baltimore, MD, ³University of Maryland, Baltimore, MD.

Introduction: Central sleep apnea syndrome (CSA) is commonly found in patients with congestive heart failure, brainstem disorders, and narcotic use. Various treatment modalities have been used with varied effectiveness in reducing the apneahypopnea index (AHI) and improving ventilation in patients with CSA. This study assessed whether Volume Assured Pressure Support (VAPS), a BiLevel mode of ventilation, is effective in treating CSA.

Methods: We performed а retrospective review of polysomnography (PSG) and VAPS titration studies on 11 patients at our institution: 7 patients had CSA with Cheyne-Stokes Respiration, 2 patients had CSA attributed to narcotic use, and 2 patients had primary CSA. CSA was diagnosed if more than 50% of the disordered breathing events were central. Five patients had failed a Continuous Positive Airway Pressure (CPAP) titration and then proceeded to VAPS while in 6 patients, VAPS was the initial treatment modality tried. We examined the effectiveness of VAPS in reducing AHI, improving oxygenation, and improving sleep architecture.

Results: Among the 11 patients, age was 63.0 ± 12.1 yo, BMI was 33.7 ± 4.5 , 7 were males, Epworth sleepiness score was 9.3 ± 4.9 . The following significant changes from baseline PSG to VAPS titration were observed: AHI: 59.1 ± 8.0 to 27.2 ± 9.9 (p<.01); Time $\leq 88\%$ O2 saturation (min): 48.1 ± 14.5 to 15.4 ± 6.1 (p<.05). Improvement in AHI was not related to gender, body mass index, narcotic use, or age. No significant changes in sleep architecture between the two studies were found. Ten (91%) patients had AHI \geq 30 on initial PSG. In 6 (55%) patients AHI was reduced to 22.2, while 4 (36%) patients did not achieve an AHI < 30 with VAPS.

Conclusion: VAPS is an effective mode of treating CSA in the majority of patients.

Support: NA

VOLUME-ASSURED PRESSURE SUPPORT IS EFFECTIVE TREATMENT FOR OBSTRUCTIVE SLEEP APNEA PATIENTS WHO FAILED CPAP TITRATION

Watanabe, N.¹ Levri, J.² Peng, V.¹ Scharf, S. M.³ Diaz, M.¹ ¹University of Maryland Medical Center, Baltimore, MD, ²University of Maryland Medical Center, Baltimore, MD, ³University of Maryland, Baltimore, MD.

Introduction: Obstructive sleep apnea (OSA) is a common disease, often treated using continuous positive airway pressure (CPAP). In many cases, patients fail an attended CPAP titration study, often due to inadequate control of AHI, and treatment-emergent central apneas as CPAP is increased. Here, we report our experience using volume-assured pressure support (VAPS) for these patients.

Methods: We retrospectively reviewed records of 45 adults who had OSA diagnosed on polysomnography (PSG) in whom CPAP titration had failed. In these patients, VAPS-AE (adjustable expiratory pressure) titrations were performed. Patients with central sleep apnea on baseline PSG were excluded.

Results: Reasons for CPAP titration failure included: treatment emergent central apneas (25), failure of maximum CPAP pressure to treat OSA (18), and persistent hypoxia (2). Average age was 57.9 ± 13.1 , BMI was 40.2 ± 8.7 , 26 males, Epworth sleepiness score was 10.7 ± 7.9 . The following significant changes from baseline PSG to VAPS titration were observed: AHI: 65.3 ± 29.3 to 22.3 ± 16.1 (p<.001) events/hour. Time < 88% saturation: 63.7 (median) to 6.9(median) min (p<.001). The number of patients with AHI<15 was 0 on PSG and 16 (36%) on VAPS-AE, while the number of patients with AHI<30 was 7 (16%) on PSG and 32 (71%) on VAPS-AE. Improvement in AHI was not related to gender, age, or narcotic use, but was correlated with BMI: Δ AHI = 12.2 - (1.4 * BMI); p=.05. VAPS resulted in improved sleep architecture: slow wave sleep increased (medians: 1.4% to 19.6% total sleep time (TST)) (p<.001), REM sleep increased (medians 6.4% to 13.6% TST) (p<.01).

Conclusion: For OSA patients for whom CPAP titration failed, titration with VAPS-AE was an effective treatment for many patients. **Support:** N/AS

0635

EFFECTS OF DONEPEZIL IN PATIENTS WITH RESIDUAL EXCESSIVE SLEEPINESS OF OBSTRUCTIVE SLEEP APNEA: A DOUBLE BLIND; RANDOMIZED PLACEBO; CONTROLLED STUDY

Sousa, K. M.¹ Piovezan, R. D.¹ e Silva, L.¹ De melo, C.¹ Poyares, D.¹ Tufik, S.¹

¹Univesidade Federal de Sao Paulo, Sao Paulo, BRAZIL, ²Univesidade Federal de Sao Paulo, Sao Paulo, BRAZIL.

Introduction: Residual excessive sleepiness (RES) is presented by 6% of obstructive sleep apnea patients despite effective CPAP therapy. Few interventions have been tested for this condition and are focused on daytime stimulants. Recently, cholinergic activity decline was suggested as a potential mechanism in the pathophysiology of RES. This study aimed to investigate the effects of donepezil, an anticholinesterase inhibitor, in patients with RES.

Methods: This double-blind, randomized, placebo-controlled crossover study included participants with RES (35-65 years). Neuropsychiatric disorders, alcoholism, smoking, shiftwork, psycho-active drugs, other sleep disorders were exclusion criteria. Participants were assigned to one intervention arm (donepezil 5 mg for 15 days followed by donepezil 10mg for 15 days or placebo in the morning).

After a 20-day wash-out, the same procedure was repeated following the crossover design. Somnolence measured by the Epworth sleepiness scale (ESS) and Maintenance of Wakefulness Test (MWT) were the primary endpoints. PSG, cognitive (trail test, continuous performance test) and Beck's depression scale parameters were secondary endpoints. General Linear Models for repeated measures compared interventions responses. Cohen's d measured effect sizes. Adverse events (AEs) were assessed by questionnaire.

Results: The study enrolled eight individuals. ESS was lower in the donepezil arm than in the placebo arm $(8.9\pm4.4 \text{ vs } 15.7\pm4.1, \text{ p}<0.05)$. Effect size for ESS was high (d 1.61). Other endpoints were not different among study arms. Randomization order didn't affect the results. No AEs were reported.

Conclusion: Donepezil improved subjective sleepiness in individuals with RES. To our knowledge, this is the first study to report the effects of a cholinergic intervention in patients with RES. Effect size was high for self-reported sleepiness, which may impact on quality of life and risk of disability in people with RES. Agents acting on the cholinergic system are potential targets for treating RES.

Support: Acknowledgements Brazilian National Council for Scientific and Technological Development (CNPq) This study is supported by AFIP (Associacao Fundo Incentivo a Pesquisa).

USING AI TO PREDICT FUTURE CPAP ADHERENCE AND THE IMPACT OF BEHAVIORAL AND TECHNICAL INTERVENTIONS

Hevener, W.¹ Beine, B.¹ Woodruff, J.¹ Munafo, D.¹ Fernandez, C.² Rusk, S.² Nygate, Y.² Glattard, N.² Piper, D.² Sheedy, C.² Simpson, M.² Turkington, F.² Shokoueinejad, M.³ ¹Sleep Data Diagnostics, San Diego, CA, ²EnsoData Research, EnsoData, Madison, WI, ³Department of Biomedical Engineering, University of Wisconsin, Madison, WI.

Introduction: Clinical management of CPAP adherence remains an ongoing challenge. Behavioral and technical interventions such as patient outreach, coaching, troubleshooting, and resupply may be deployed to positively impact adherence. Previous authors have described adherence phenotypes that retrospectively categorize patients by discrete usage patterns. We design an AI model that predictively categorizes patients into previously studied adherence phenotypes and analyzes the statistical significance and effect size of several types of interventions on subsequent CPAP adherence.

Methods: We collected a cross-sectional cohort of subjects (N = 13,917) with 455 days of daily CPAP usage data acquired. Patient outreach notes and resupply data were temporally synchronized with daily CPAP usage. Each 30-days of usage was categorized into one of four adherence phenotypes as defined by Aloia et al. (2008) including Good Users, Variable Users, Occasional Attempters, and Non-Users. Cross-validation was used to train and evaluate a Recurrent Neural Network model for predicting future adherence phenotypes based on the dynamics of prior usage patterns. Two-sided 95% bootstrap confidence intervals and Cohen's d statistic were used to analyze the significance and effect size of changes in usage behavior 30-days before and after administration of several resupply interventions.

Results: The AI model predicted the next 30-day adherence phenotype with an average of 90% sensitivity, 96% specificity, 95% accuracy, and 0.83 Cohen's Kappa. The AI model predicted the number of days of CPAP non-use, use under 4-hours, and use over 4-hours for the next 30-days with OLS Regression R-squared values of 0.94, 0.88, and 0.95 compared to ground truth. Ten resupply interventions were associated with statistically significant increases in adherence, and ranked by adherence effect size using Cohen's d. The most impactful were new cushions or masks, with a mean post-intervention CPAP adherence increase of 7-14% observed in Variable User, Occasional Attempter, and Non-User groups.

Conclusion: The AI model applied past CPAP usage data to predict future adherence phenotypes and usage with high sensitivity and specificity. We identified resupply interventions that were associated with significant increases in adherence for struggling patients. This work demonstrates a novel application for AI to aid clinicians in maintaining CPAP adherence. **Support:**

0637

COMPARISON OF AHI AND ESS OUTCOMES BETWEEN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA UNDERGOING SLEEP SURGERY VERSUS UPPER AIRWAY STIMULATION

Huntley, C.¹ Doghramji, K.¹ Tschopp, K.³ Tschopp, S.³ Jardin, P. B.⁴ Heiser, C.⁵ Schwab, R.⁶ Thaler, E.⁶ Jenks, C.⁷ Walia, H.⁸ Kominsky, A.⁸ Kezirian, E.⁹ Waxman, J.¹⁰ Lin, H.¹¹ Boon, M.¹ ¹Thomas Jefferson University, Philadelphia, PA, ²Thomas Jefferson University, Philadelphia, PA, ³Kantonsspital Baselland, Liestal, SWITZERLAND, ⁴Clinica Universitaria de Navarra, Navarra, SPAIN, ⁵Technical University of Munich, Munich, GERMANY, ⁶University of Pennsylvania, Philadelphia, PA, ⁷University of pennsylvania, Philadelphia, PA, ⁸Cleveland Clinic, Cleveland, OH, ⁹University of Southern California, Los Angeles, CA, ¹⁰Wayne State University, Detroid, MI, ¹¹Wayne State, Detroid, MI.

Introduction: Single or multi-level soft tissue surgical interventions are common options for CPAP-intolerant patients with OSA. Upper Airway Stimulation (UAS) is an alternative option using an implantable hypoglossal nerve stimulator. We compared patient outcomes between traditional sleep surgery (TSS) and UAS.

Methods: We selected patients who underwent TSS (including palate, oropharynx, tongue, and/or epiglottis-based procedures) for OSA and also met general UAS criteria (BMI≤35, AHI between 15-65, absence of palate concentric collapse during DISE, if available) for chart review. UAS outcomes were collected from the ADHERE international registry. For both groups, post-op AHI was collected, including full-night UAS efficacy studies. Data are presented as mean and standard deviation.

Results: The TSS group (n=284) and UAS group (n=541) were predominantly male and overweight. The TSS group was younger than UAS (47±12 vs 60±11 years, p<0.001). At baseline, both groups had severe OSA with AHI of 34±14 and 36±15 (p=0.23) and excessive daytime sleepiness with ESS of 12±5 and 12±6, (p=0.38), respectively. TSS follow-up was 169±151 days vs 392±181 days for UAS, which was significantly different. UAS had significantly larger decrease in AHI than TSS (-21/h±18 vs -16/h±16, p<0.0001). Both groups had a large decrease in ESS, however, the UAS group had a slightly smaller decrease, (-6±5 vs -5±5, p=0.01). Using the Sher response criteria of 50% AHI reduction and ≤ 20 events/hour, UAS had a 70% response rate vs 51% for TSS.

Conclusion: This study represents largest and first international, multicenter comparison of UAS to traditional surgical interventions for OSA, albeit with limitations of potential differential patient selection. While both TSS and UAS show similar improvement in symptoms, upper airway stimulation has a larger reduction of AHI with higher rates of therapy response than traditional sleep surgery

Support: ADHERE data assistance from Inspire Medical (Minneapolis, MN)

0638

EVALUATION OF AN ORAL DRUG PYRIDOSTIGMINE BROMIDE IN PATIENTS WITH MILD TO MODERATE OBSTRUCTIVE SLEEP APNEA

FAN, J.¹ Wu, H.² Chen, G.² Lv, Q.² Shi, C.³ Ma, X.⁴ Gao, H.⁵ Palling, D.¹

¹Pfantastic Med Res, Cresskill, NJ, ²Emergency General Hospital, Beijing, CHINA, ³Meitan University, Beijing, CHINA, ⁴Liang Xiang Hospital, Beijing, CHINA, ⁵Air Force Medical Center, Beijing, CHINA.

Introduction: A randomized, double-blind, cross-over, placebocontrolled clinical study with pyridostigmine bromide (PYD) in obstructive sleep apnea (OSA) patients ranging from mild to moderate disease was conducted to evaluate its clinical efficacy and safety.

Methods: Six diagnosed male patients with averages of age 48 yr (38 - 57 yr), BMI 28 (26 - 33), AHI 19.2 (15 - 26.2), minimum oxygen saturation (Min Sa₀₂) 81% (75 - 87%) were enrolled to the study. The study consisted one-night acclimatization period followed immediately by a 2-night double-blind treatment period when subjects received either a single dose of PYD (a cholinesterase inhibitor, 90 mg) or placebo before sleep. Subjects were required to maintain in a supine position, and monitored by a standard polysomnography all the time. Sleep questionnaires (The SMH Sleep Questionnaire and ESS) were taken daily immediately after sleep and at the evening to evaluate the sleep satisfaction and the day-time quality, respectively. Safety of the drug was monitored and evaluated.

Results: Reductions of AHI (28.1%, p < 0.01), apnea index (37.2%, p < 0.05), % of total apnea/hypopnea time (36.4%, p < 0.05) were observed in the treatment group compared with the placebo between 2-7 hours of sleep. Min Sa₀₂ was increased, no change, or decreased by PYD in 3, 2, or 1 subject(s), respectively. PYD was generally well tolerated with minimum minor incidents. Subjects reported to have more satisfied sleep and more clear-headed in the treatment night, and more energy, more concentrated and less sleepy during the daytime following the treatment night.

Conclusion: This study demonstrated the initial effectiveness of the PYD treatment for OSA, indicating that it may provide a new treatment option if the efficacy can be maintained in a large-scale clinical trial.

Support: N/A

0639

LONG TERM ORAL APPLIANCE THERAPY DECREASES STRESS SYMPTOMS IN UPPER AIRWAY RESISTANCE SYNDROME PATIENTS

Godoy, L. B. Sousa, K. M. Palombini, L. O. Guimarães, T. M. Poyares, D. Tufik, S. Togeiro, S.

Departamento de Psicobiologia - Universidade Federal de São Paulo, São Paulo, BRAZIL.

Introduction: Upper Airway Resistance Syndrome (UARS) is suspected in individuals with excessive daytime sleepiness, fatigue and sleep fragmentation due to increased respiratory effort. UARS can negatively impact daytime function and decrease quality of life. Cognitive impairment in UARS patients has not been well stablished yet. The objective of the study was to evaluate the long-term effects of a mandibular advancement device (MAD) on cognitive function in patients with UARS compared with placebo.

Methods: This study was a randomized placebo-controlled clinical trial. Thirty UARS patients were randomized in two groups: placebo and MAD groups. UARS criteria were the presence of sleepiness (Epworth Sleepiness Scale ≥ 10) and/or fatigue (Modified Fatigue Impact Scale ≥ 38) associated with an apnea/hypopnea index (AHI) ≤ 5 and a respiratory disturbance index (RDI) ≥ 5 events/hour of sleep, and/or flow limitation in more than 30% of total sleep time. All patients completed the Rey Auditory-Verbal Learning Test (RAVLT), the Logical Memory Test, the Stroop Color Test, the Trail Making Test, the Digit Symbol Substitution Test and Inventory of Stress Symptoms (Lipp test). Cognition protocol was defined based on most used neuropsychological tests in the literature. Evaluations were performed before and after 1.5 years of treatment.

Results: There was no statistically significant difference in RAVLT, Logical Memory Test, Stroop Color Test, Trail Making Test, Digit

Symbol Substitution Test before and after 1.5 year of treatment in both groups. The Lipp test score decreased at alarm phase (p = 0.05) and at resistance phase (p = 0.01) after 1.5 year of MAD treatment compared to placebo.

Conclusion: Mandibular advancement device was effective in decreasing stress symptoms in alarm and resistance phases of Lipp test in UARS patients after 1.5-years of MAD treatment.

Support: The authors would like to thank for the support by grants from Associação Fundo de Incentivo a Pesquisa (AFIP), Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP), Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) and Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES).

0640

INITIAL SLEEP CENTER EVALUATION AND FOLLOW UP IMPROVES POSITIVE AIRWAY PRESSURE (PAP) THERAPY ADHERENCE WHEN COMPARED TO DIRECT REFERRALS WITHOUT SLEEP PHYSICIAN FOLLOW UP: A RETROSPECTIVE STUDY

*Aljarod, T.¹ Tran, L.² Al Ikhwan, M.³ Prasad, B.³.*3 ¹University of Illinois at chicago, Chicago, IL, ²Universitry of Illinois at Chicago, Chicago, IL, ³University of Illinois at Chicago, IL.

Introduction: Obstructive sleep apnea (OSA) affects 26% of adults and positive airway pressure (PAP) is the gold-standard of therapy. Factors affecting PAP adherence—use >4 hours in a 24-hour period—have been studied extensively. We compared of the three months (or other time frame) PAP adherence between patients seen by a sleep specialist prior to OSA diagnosis versus patients referred directly for OSA testing by non-sleep specialist providers. The goal of the study was to understand the impact of sleep consultation on PAP adherence.

Methods: Direct referral (DR) patients underwent polysomnography (PSG) and received PAP devices prior to the sleep clinic visit. In contrast, sleep center patients (SC) had a sleep clinic visit with a sleep physician or APRN prior to PSG.

Eighty-four patients were included in this study, 42 DR and 42 SC patients. Exclusion criteria were age <18 years old, absence of baseline PSG, and lack of 90-day compliance data. Covariates included demographics, body mass index (BMI), AHI, nadir oxygen saturation, demographics, and Epworth Sleepiness Scale (ESS) score. Objective PAP adherence for first 90 days was the primary outcome.

Results: Age (p=0.1), ESS (p=0.3), BMI (p=0.6), and AHI (p=0.9) were not significantly different between the groups. SC patients had greater PAP adherence (4.77 hours, 95%CI: 4.1 to 5.4) compared to DR patients (3.61 hours, 95%CI: 2.88 to 4.33, p=0.02). SC patients were also 8 times more likely to follow up in clinic within 1 year of starting PAP treatment (Likelihood Ratio 8.25, p=0.004).

Conclusion: While possibly more time-efficient for patients, direct referrals may ultimately result in lower PAP adherence due to missed opportunities for receiving education about OSA and PAP therapy. This is consistent with findings from a previous meta-analysis demonstrating that educational interventions improve PAP adherence. Moving forward, we will continue encouraging directly referred patients to follow up in the sleep center after PSG.

Support: None

EFFECTS OF WEIGHT LOSS DURING LONG-TERM SOLRIAMFETOL TREATMENT ON CARDIOMETABOLIC INDICES

Malhotra, A.¹ Strollo, P.² Pepin, J.³ Schweitzer, P.⁴ Lammers, G.⁵ Hedner, J.⁶ Redline, S.⁷ Chen, D.⁸ Chandler, P.⁸ Bujanover, S.⁸ Menno, D.⁹ Strohl, K.¹⁰

¹Division of Pulmonary, Critical Care and Sleep Medicine, University of California San Diego Medical Center, La Jolla, CA, ²University of Pittsburgh/Veterans Administration Pittsburgh Health System, Pittsburgh, PA, ³Grenoble Alpes University Hospital, Grenoble, FRANCE, ⁴Sleep Medicine and Research Center, St. Luke's Hospital, Chesterfield, MO, ⁵Leiden University Medical Centre, Leiden, NETHERLANDS, ⁶Sahlgrenska University Hospital, Gothenburg, SWEDEN, ⁷Brigham and Women's Hospital, Boston, MA, ⁸Jazz Pharmaceuticals, Palo Alto, CA, ⁹Jazz Pharmaceuticals, Philadelphia, PA, ¹⁰Case Western Reserve University, Cleveland, OH.

Introduction: Increased prevalence of obesity has been reported in patients with narcolepsy and obstructive sleep apnea (OSA). Results from a 1-year open-label extension (OLE) study showed $\geq 5\%$ weight loss in 4.5%, 17.3%, and 32.4% of participants with narcolepsy or OSA receiving solriamfetol 75, 150, or 300mg/d. We examined whether clinically significant weight loss ($\geq 5\%$) in this population had favorable effects on biomarkers of cardiometabolic risk compared to no such weight loss.

Methods: We evaluated changes in weight and BP (parent study baseline to OLE week 40) and clinical laboratory assessments (OLE baseline to week 40) in participants with narcolepsy (n=124) or OSA (n=250) from the OLE study.

Results: Of 374 participants, 96 (25.7%) had \geq 5% weight loss and 34 (9.1%) had \geq 5% weight gain. Demographics were similar in those with and without $(n=278) \ge 5\%$ weight loss. From baseline to week 40, among participants with weight loss, there were decreases in percentage with high (ie, >ULN) serum glucose (36.6% to 28.1%) or triglycerides (26.6% to 21.9%), whereas among participants without weight loss, there was an increase in percentage with high glucose (43.3% to 50%) and no change in percentage with high triglycerides (37.1% to 37.2%). The percentage of participants with high total cholesterol was stable among participants with weight loss (22.3% to 22.9%) and increased (32.7% to 37.2%) in participants without weight loss. Participants with weight loss had mean±SD reductions in SBP (-2.6±11.4mmHg) and DBP (-1.0±9.0mmHg), whereas participants without weight loss had increases of +0.65±12.5mmHg and +1.2±8.7mmHg, respectively.

Conclusion: Among solriamfetol-treated participants with $\geq 5\%$ weight loss, there

were decreases in BP and percentage of participants with high glucose and triglycerides. Further research is required to examine prospective long-term effects of solriamfetol on specific biomarkers of cardiometabolic risk.

Support: Jazz Pharmaceuticals

0642

A NOVEL DAYTIME INTRA-ORAL NEUROMUSCULAR STIMULATION THERAPY IN SIMPLE SNORERS: OBJECTIVE IMPROVEMENT IN SNORING

Kotecha, B.

Nuffield Hospital, Brentwood, Essex, UNITED KINGDOM.

Introduction: The reduction in pharyngeal muscle tone in the upper airway is a pivotal factor in snoring and obstructive sleep apnoea (OSA). There is accumulative evidence that pharyngeal exercises can reduce snoring and OSA. We present a novel device SnooZeal® that uses daytime awake neuromuscular electrical stimulation (NEMS) as an application to induce toning of the tongue muscles. This study investigates objective changes in snoring and respiratory parameters with this device.

Methods: Prospective cohort study of 100 simple snorers was conducted. Objective snoring and respiratory parameters were recorded with 2 consecutive WatchPat sleep studies before and after treatment. SnooZeal® device was used for 20 minutes once a day for a 6 week period. Secondary outcome measures using visual analogue scale reporting of snoring by patient and Epworth Sleepiness Score (ESS) were recorded.

Results: Objective reduction of snoring was noted on the sleep studies in 95% with an average reduction of 48%. Subjectively, the visual analogue scale reported by partners similarly demonstrated reduction in 95% of the patients with an average reduction of 40%. **Conclusion:** This prospective cohort study demonstrates a notable improvement in both objective and subjective parameters of snoring and respiratory indices. SnooZeal® offers a novel approach to reduce snoring by utilising intra-oral neuromuscular electrical stimulation. This could be a preferred option for patients as it alleviates the need of using an oral device during sleep. **Support:**

0643

DAYTIME INTRA-ORAL NEUROMUSCULAR STIMULATION THERAPY ON PATIENTS WITH MILD OBSTRUCTIVE SLEEP APNOEA

Kotecha, B.

Nuffield Hospital, Brentwood, Essex, UNITED KINGDOM.

Introduction: Upper airway muscle stimulation can improve pharyngeal muscle tone and thus could be considered for treatment of obstructive sleep apnoea (OSA). We present a novel device SnooZeal® that uses daytime awake neuromuscular electrical stimulation (NEMS) as an application to induce oropharyngeal muscular training. This study demonstrates objective and subjective usefulness of SnooZeal® in improving Mild OSA.

Methods: Prospective cohort study of 40 patients with Mild OSA (Apnoea-Hypopnea Index of 5-15/hour) was conducted. Objective respiratory parameters were recorded with two consecutive WatchPat sleep studies before and after treatment with the SnooZeal® device. The device was used by the patients for 20 minutes every day for 6 weeks. Furthermore, visual analogue scale for the snoring was completed by the partner as was the Epworth Sleepiness Score (ESS) as secondary outcome measures over the duration of the study.

Results: A statistically significant improvement was demonstrated in this cohort with mean AHI (Apnoea-Hypopnea Index) dropping from 9.91/hour prior to treatment to 5.2/hour following the treatment. The Oxygen desaturation index also reduced from a mean of 7.9 to 4.7 and similarly, there was also a statistically significant drop in the ESS from 8.86 to 5.43.Objective reduction in snoring was also demonstrated on the sleep studies in 95% of patients with an average reduction of 48% which was reflected subjectively by 40% reduction as recorded on visual analogue scale by partners.

Conclusion: SnooZeal[®] device study in this cohort of mild OSA patients demonstrates encouraging results. It offers the advantage of not requiring a device in situ whilst asleep which many patients find annoying and intolerable and thus a useful treatment option

in patients with snoring and mild OSA. The device improves the muscle tone of the tongue and hence the upper airway dimensions during sleep.

Support:

0644

AN EDUCATIONAL AND BEHAVIORAL INTERVENTION FAILED TO IMPROVE PAP USE AMONG VETERANS WITH SPINAL CORD INJURIES AND DISEASES: RESULTS OF A RANDOMIZED CONTROLLED TRIAL

Martin, J. L.^{1,2} Sankari, A.^{3,4} Salloum, A.^{3,4} Zeineddine, S.^{3,4} Arvai, K.³ Henzel, M. K.^{5,6} Shamim-Uzzaman, Q.⁷ May, A.⁵ Fung, C. H.^{1,2} Mitchell, M. N.¹ Strohl, K. P.⁵ Badr, M. S.^{3,4} ¹VA Greater Los Angeles, North Hills, CA, ²University of California, Los Angeles, Los Angeles, CA, ³Wayne State University, Detroit, MI, ⁴John D. Dingell VA Medical Center, Detroit, MI, ⁵VA Northeast Ohio Healthcare System, Cleveland, OH, ⁶Case Western Reserve University, Cleveland, OH, ⁷VA Ann Arbor Healthcare System, Ann Arbor, MI, ⁸VA Greater Los Angeles, North Hills, CA.

Introduction: Sleep disordered breathing (SDB) is common among individuals with spinal cord injuries or diseases (SCI/D), many of whom are military Veterans, and physical limitations make use of positive airway pressure (PAP) therapy challenging. This study sought to test the effects of an educational and behavioral intervention to improve PAP adherence among Veterans with SCI/D over the first 3 months of use.

Methods: 63 Veterans (mean age=60.7(10.2) years; 92% male) with SCI/D (33 SCI, 30 SCD; 25 cervical involvement; 38 thoracic and below) and SDB (23 with AHI 5-15; 40 with AHI≥15) who received PAP treatment (CPAP or BPAP) were randomly assigned to receive a comprehensive 3-month intervention (INT) or an equal attention control (EAC). INT and EAC both included 1 face-to-face session (week 1) and 5 additional telephone sessions (weeks 2, 3, 4, 8 and 12). Main outcome measures were PAP use over the first 3 months: nights of use, nights of use ≥4 hours, and mean hours of use per night. Repeated measures ANCOVA models were used to test the differences between INT and EAC over the first 3 months of treatment.

Results: Number of nights with ≥4 hours of use in months 1-3 was 9, 7, and 6 nights in the INT and 8, 5 and 4 nights in the EAC (p's≥.37), respectively. There were no significant differences between INT and EAC for number of nights with any use (p's≥.24), or mean hours of use per night (p's≥.30). All 3 PAP use variables declined over time in both groups.

Conclusion: Sustained use of PAP therapy was difficult to achieve among those with SCI/D, and a 6-session behavioral intervention did not lead to significant improvements in use. Even when relatively high levels of initial use are achieved, this is difficult to sustain over time. Future studies should explore whether SCI/D patients experience significant symptom relief with PAP, and if so, whether home-based interventions or more intensive face-to-face PAP adherence programs will be effective.

Support: VA Rehabilitation Research and Development Service, Merit Review (1RX002116; PI: Badr); NIH/NHLBI K24 HL143055 (Martin).

0645

THE ASSOCIATION OF HYPOGLOSSAL NERVE STIMULATOR ADHERENCE AND INSOMNIA Sharma, M. Chacko, A. Rosenthal, M. Khan, M. The Ohio State Unviersity Wexner Medical Center Division of Pulmonary, Critical Care, and Sleep Medicine, Columbus, OH.

Introduction: The gold standard for treatment of Obstructive sleep apnea (OSA) is continuous positive airway pressure (CPAP). However, CPAP adherence is less than 50%. An alternative treatment is the hypoglossal nerve stimulator (HNS) which displaces the tongue anteriorly to treat upper airway obstruction. Difficulties tolerating HNS are related to stimulation discomfort. In this study, we investigated insomnia as a barrier to adherence.

Methods: Patients implanted and activated with HNS at The Ohio State University Wexner Medical Center between 2015 and 2019 were eligible. Patient usage data from the previous six months was obtained and subjects were asked to complete an Insomnia Severity Index (ISI). Participants were divided into adherent (defined as use ≥28 hours/week) and non-adherent (use <28 hours/week).

Results: 32 subjects were enrolled, 22 in the adherent group and 10 in the non-adherent group. There was a significant decrease in mean treatment AHI in both groups: 36.25 to 11.14 in the adherent group and 36.30 to 15.69 in the non-adherent group (p<0.0001). The mean ISI score in the adherent group was 6.84 which is consistent with no clinically significant insomnia and 8.67 in the non-adherent group consistent with subthreshold insomnia. However, there was not a statistically significant difference between the two groups (p=0.441). There was a statistically significant higher score for the question "Do you worry about your sleep problems?" in the non-adherent group (1.78, SD1.39 vs 0.74. SD 0.81) (p =0.018).

Conclusion: This study suggests that patients who have difficulty with sleep may have more difficulty with HNS adherence than those who do not. In particular the question stating "Do you worry about your sleep problems" had a statistically higher score in the non-adherent group. Prospective studies are needed to further explore a possible relationship between insomnia and HNS adherence.

Support: N/A

0646

EVALUATION OF INCREASED CPAP COMPLIANCE AFTER ELECTRONIC LEARNING VIDEO AIMED AT TROUBLESHOOTING COMMON CPAP RELATED PROBLEMS

Slota, K. A.¹ Wasey, W.² Dredla, B. K.¹ Castillo, P.¹ ¹Mayo Clinic, Jacksonville, FL, ²SIU Medicine, Springfield, IL.

Introduction: Obstructive sleep apnea(OSA) is a common diagnosis associated with immediate and long-term health consequences. Due to increasing awareness, the demand for treatment with CPAP is increasing. However, CPAP compliance remains a problem, and may be improved by initial patient education targeted at common CPAP problems

Methods: A 10 min video depicting common CPAP related problems was created in the Mayo Clinic Florida Simulation Center using standardized patients. Newly diagnosed patients with OSA requiring CPAP were randomized into two arms: (i) the intervention arm was shown the video and received standard of care; or (ii) in the "placebo" or standard of care alone. Our standard of care includes a review of the sleep study, discussion of diagnosis and recommendation for CPAP therapy along with Mayo OSA information packet, followed by compliance visit at 3 months. Compliance is defined as >4 hours of CPAP use per night gathered from machine's SD card. The two groups were compared for statistically significant difference in compliance at 3 months.

SLEEP, Volume 43, Abstract Supplement, 2020

Results: 47 patients diagnosed between 10/2018-5/2019 were included in the study(21:intervention, 26:placebo). 7 (33%) and 7 (26%) patients in the intervention and the placebo came back for follow up visit (p=0.63). Among them, median CPAP usage was 362.5 min (236.0, 480.0) in intervention arm vs 351.0 min (125.0, 466.0) in placebo group and the difference was not significant. Average nightly use of >than 4 hours was 12(57.1%) in the intervention group and 17(65.4%) in placebo group.

Conclusion: In this group addition of an educational video to standard of care did not show benefit in CPAP compliance. There was a tendency toward greater median nightly usage. Patients receiving the video had a higher likelihood of making their follow up appointment which is pivotal, as it provides an opportunity for further intervention and enhancement of adherence. **Support:** Mayo Clinic Jacksonville

0647

THE EFFECT OF CPAP ON THE BLOOD FLOW TO THE BRAIN: A PRELIMINARY REPORT

Alperin, N.¹ Hernandez-Cardenache, R.² Lee, S.³ Junco, B.³ Ramos, A. R.³

¹Department of Radiology, Miller School of Medicine, University of Miami, FL, ²Department of Psychiatry, Miller School of Medicine, University of Miami, FL, ³University of Miami, Miller School of Medicine, Miami, FL, ⁴University of Miami, Miller School of Medicine, Miami, FL.

Introduction: We aim to determine the effect of positive airway pressure (PAP) on total cerebral blood flow in a sample of middle-aged to older male patients with obstructive sleep apnea (OSA).

Methods: We evaluated consecutive treatment-naïve male (OSA) patients with Apnea hypopnea Index (AHI) \geq 15, from January to November of 2019. We obtained demographic variables, vascular risk factors, the Epworth sleepiness scale (ESS) and the Pittsburgh sleep quality index (PSQI). Brain magnetic resonance imaging (MRI) consisted of high resolution anatomical imaging and velocity encoded phase contrast MRI to measure volumetric blood flow rate to the brain, or the total cerebral blood flow (tCBF), by summation of the flow rate through the two internal carotid and vertebral arteries. Positive airway pressure at various pressures was provided during the MRI, while subjects were awake, starting at 0 cm H20, then 5 cmH20, 10, 15, and 20 cmH20. Each setting was applied for six minutes. Two subjects did not tolerate pressure setting of 20 cmH20.

Results: We had a total of 11 participants' age 40-73 years, 70% Hispanic/Latino background. The average ESS was 8.2 ± 6.0 , PSQI= 5.7 ± 4.9 , and AHI= 48.9 ± 25.5 . Five of the subjects had hypertension and a quarter had diabetes. In all subjects total CBF monotonically decreased with increasing PAP. The initial decreases in total CBF (PAP from 0 to 5 and 5 to 10) where significantly larger than the decrease in the higher pressure range. In two subjects the total decrease in tCBF was nearly 50%. The average tCBF was 682.3 mL/min at 0 cmH20 and 506.3 ml/min at 20 cmH20.

Conclusion: Incremental increases in PAP lead to substantial decrease in tCBF during wake. Substantial decrease in tCBF is possibly explained by decreasing end tidal CO2 pressure. The specific physiologic cause and the neurologic impact of prolonged decreased tCBF during CPAP therapy needs to be further investigated.

Support: Scientific Advisory Committee, Pilot grant, Miller School of Medicine; R21AG056952; R21HL140437; Jazz-Pharmaceutical.

0648

IMPACT OF POSITIVE AIRWAY PRESSURE (PAP) TROUBLESHOOTING CLINIC VISITS ON PATIENT SATISFACTION, PAP USE, MASK LEAK, AND PROLONGED USE

Bennett, K. L.

Michigan Medicine, Brighton, MI.

Introduction: Patients with sleep apnea are often prescribed positive airway pressure (PAP) treatment. Some patients have difficulty consistently using a PAP machine due to problems such as air leak, mask discomfort, and dry mouth. The purpose of this project is to evaluate the satisfaction and efficacy of a PAP Troubleshooting Clinic lead by a RN and a respiratory technician (RT). This clinic seeks to improve PAP compliance, increase PAP tolerability and increase PAP treatment efficacy

Methods: The PAP Troubleshooting Clinic consists of a RN and RT with specialized knowledge about sleep apnea and PAP treatment. During a 30 or 60-minute clinic visit, these providers review the patient's experience with PAP, assess mask fit, review PAP data and PAP settings, and recommend treatment adjustments for the primary Sleep Clinic provider's consideration. Outcomes assessment includes satisfaction (a telephone survey one week after the visit) and efficacy (30-day data on overall use, mask leak, and days with \geq 4 hours of use downloaded from PAP machines).

Results: To date, 58 patients have received care in the clinic and 56 (96.5%) patients reported they were satisfied/highly satisfied with the PAP Troubleshooting Clinic. From the sample, forty-three (74.1%) patients were compliant with mask use over 30 days. Twenty-four (55.81%) showed a greater than 10% reduction in mask leak after intervention. Ten patients (23.3%) achieved an improvement with a 10% increase in number of days with > than 4 hours of PAP use.

Conclusion: Patients and Sleep Clinic providers are very satisfied with the PAP Troubleshooting Clinic. Patients are referred to this clinic because they have significant issues with PAP usage and are at high risk of discontinuing use. Patients find the clinic helpful and encouraging, while sleep medicine physicians and APPs appreciate the assistance in helping patients succeed with PAP, especially during the 30-day time period where PAP compliance receives scrutiny from clinics and payers.

Support: A PAP Troubleshooting Clinic is an effective way to improve patient PAP Use, Mask Leak, Prolonged Use and Patient Satisfaction. Importantly, this new clinical model offers a valuable alternative to provide patients with the appropriate level of care.

0649

ULTRASOUND ASSESSMENT OF TONGUE MOVEMENT AS A PREDICTOR OF RESPONSE TO HYPOGLOSSAL NERVE STIMULATION (HGNS)

Korotun, M. Hahn, S. Quintero, L. Rajan, P. Iakovou, A. Mayo, P. Greenberg, H.

Northwell Health, New Hyde Park, NY.

Introduction: HGNS is an approved therapy for obstructive sleep apnea (OSA). Initial setting of HGNS voltage is based on observation of anterior tongue movement, which may not reflect opening of the retroglossal airway. We developed an ultrasonographic (US) technique to assess tongue movement with HGNS. We correlated US measures of tongue movement at the initial HGNS voltage setting with the AHI determined by PSG/HSAT on HGNS therapy. **Methods:** Eleven subjects implanted with INSPIRETM (HGNS) were enrolled at least one month post-implantation. Initial HGNS voltage was determined while awake and semi-recumbent and set to achieve visualized anterior tongue protrusion at a tolerable stimulation voltage. A curvilinear probe (5-2MHz) was placed longitudinally in the submental region at the midline with the indicator pointed anteriorly. Hyoid bone excursion (HBE) with stimulation was used as a marker of base of tongue movement. PSG or HSAT was performed to determine AHI with HGNS. Responders were defined as those with a reduction in AHI ≥50% and an AHI <20 events/hr.

Results: N=11, 6M, 5F, Age=66.5 \pm 18.4 years, BMI=27.9 \pm 2.7 kg/m². Pre-treatment AHI=38.8 \pm 13.4/hr, T-90%=10.5 \pm 16.7%. Mean HBE in responders=1.02 \pm 0.17cm vs 0.76 \pm 0.20cm in non-responders (p=0.006). Best subsets regression analysis performed using post-treatment AHI as the dependent variable and age, BMI, baseline AHI, HBE and HGNS voltage as independent variables showed that HBE (coef. -29.1, p=0.038) and BMI (coef. 2.6, p=0.018) were independent predictors of response.

Conclusion: We demonstrated that ultrasound assessment of HBE during HGNS may be a useful tool to predict response to therapy and guide HGNS settings. HBE, rather than voltage, predicted post-treatment AHI.

Support: None.

0650

IMPACT OF UPPER AIRWAY STIMULATION THERAPY ON 24 HOUR AMBULATORY BLOOD PRESSURE MONITORING

De, A. Bena, J. Wang, L. Aylor, J. Bhambra, R. Kaw, S. Lance, C. Mehra, R. Walia, H.

Cleveland Clinic, Cleveland, OH.

Introduction: Upper airway stimulation (UAS) is recommended treatment for moderate to severe obstructive sleep apnea (OSA) in select patients. Existing data have not focused on gold standard 24 hour ambulatory blood pressure monitor (ABPM) to elucidate the impact of UAS. We hypothesize that UAS reduces ABPM indices characterized using objective sleep-wake from actigraphy data over 12-month follow-up period.

Methods: A prospective sub-study of the Inspire ® post-approval study at the Cleveland Clinic was designed to examine the effect of UAS on 24-hour ABPM measures post-implantation by examining blood pressure (BP) at baseline, and-2, 6, and 12 months follow-up. Actigraphy data was contemporaneously collected. Paired T-tests were used to evaluate BP changes over time. Repeated measure correlations measured within-patient associations between BP and actigraphy measures. Results: Average age and BMI were 62.4 +/-12.9) years and 30.1 +/-3.3 kg/m², 73.3% males and all Caucasian. The mean baseline systolic, diastolic and mean arterial pressure (MAP) were 119.7+/-12.9 mmHg, 74.3+/-7.4 mmHg and 89.3+/-8.1 mmHg. There were no changes to number, type or dosage of BP medications. At 12 months, there were non-significant overall mean reduction in systolic [-0.55mmHg, p=0.75], diastolic [-0.73mmHg, p=0.63], and MAP [-0.55mmHg, p=0.71]. Mean sleeping systolic, diastolic and MAP changed by -4.36(p=0.34), -1.45 (p=0.57), -2.18 (p=0.50), respectively. Positive correlations above 0.25 (p<0.10) were observed between all dipping percentage measures and total sleep time. Negative correlations were seen between overall systolic, diastolic and MAP with sleep latency (-0.22, p=0.19, -0.35, p=0.031 and -0.29, p=0.075 respectively). No significant changes in BMI was observed, but average hours of usage decreased over time.

Conclusion: Although consistent reduction of BP measures were observed post-UAS implantation, findings were not statistically significant. It is unclear whether this is due to insufficient sample size or true lack of effect. Larger-scale clinical and mechanistic studies are needed to enhance understanding of UAS-related vascular influences.

Support: Funded: Inspire Medical Systems

0651

LONGITUDINAL EFFECTS OF CPAP ADHERENCE ON CHANGES IN PTSD SYMPTOMS AND SUBSCALES: THE IMPORTANCE OF HYPERAROUSAL ON ADHERENCE AND OUTCOMES

Colvonen, P. J.¹ Lizbeth, G.² Sarmiento, K.³

¹University of California, San Diego, San Diego, CA, ²San Francisco VA Medical Center, San Francisco, CA, ³University of California, San Francisco, San Francisco, CA.

Introduction: Comorbidity of posttraumatic stress disorder (PTSD) and obstructive sleep apnea (OSA) is staggeringly high, with rates of 75.7% when using criteria of apnea/hyponea index (AHI) > 5. Continuous positive airway pressure (CPAP) therapy can decrease PTSD symptoms, however, no study has used advanced mixed-modeling to examine which cluster of PTSD CPAP therapy effects.

Methods: Participants were 59 veterans with PTSD and undiagnosed OSA. Apnea/Hypopnea index (AHI) was scored according to AASM criteria. Auto-titration CPAP devices were prescribed with pressures empirically selected by the sleep physician based on BMI, overall AHI, and dominant event type; maximum pressures was set at 20cm H2O. Analyses used hierarchical linear modeling to examine changes in PTSD symptoms clusters as a function of CPAP use over 6-months. Measures include PTSD checklist (PCL) and clusters (reexperiencing, avoidance, and hyperarousal), percentage of nights CPAP used, weight, and BMI.

Results: Baseline scores were high: PCL (M=60.02; SD=15.03) and AHI (M=28.18 per hour; SD=20.35). Average number of nights CPAP use in the last 6-months was 59.3%, with 3.5 hours each night with clear adherent and non-adherent groups emerging. The adherent group showed a 15-point drop in PCL scores and the non-adherent group had a 3-point drop. More days of CPAP use in the last 6-months predicted larger decreases in hyperarousal (d=0.56) and re-experiencing (d=0.47) clusters, but not avoidance. The intercept was significant in the hyperarousal analyses suggesting individuals with higher hyperarousal at baseline had less CPAP adherence.

Conclusion: The need for PTSD clinicians to screen and refer for OSA is necessary, but may not be sufficient, in treating PTSD. Change in hyperarousal symptoms accounted for most of the effects from CPAP use. Higher hyperarousal symptoms at baseline predict lower CPAP adherence, suggesting a higher clinical need to address these individuals as they will get the most positive effects from CPAP.

Support: This work is supported by a VA RR&D CDA (11K2Rx002120-01) to Peter Colvonen.

0652

A NOVEL COGNITIVE-BEHAVIORAL THERAPY TO INCREASE PAP ADHERENCE IN VETERANS WITH POSTTRAUMATIC STRESS DISORDER: PRELIMINARY RESULTS

Kinoshita, L.¹ Blank, E.¹ Chen, M.¹ Doudell, K.³ Day, Y.¹ Alipio Jocson, V.¹ Lazzeroni, L.⁴ Noda, A.⁴ Hernandez, B.⁴ Holty, J.¹ Kuschner, W.¹ Kushida, C.⁴ Yaffe, K.⁵ Cheng, J.¹ Yesavage, J. A.¹

¹VA Palo Alto Health Care System, Palo Alto, CA, ²VA Palo Alto Health Care System, Palo Alto, CA, ³University of Alabama, Tuscaloosa, AL, ⁴Stanford University, Palo Alto, CA, ⁵San Francisco VA Medical Center, San Francisco, CA.

Introduction: The occurrence of obstructive sleep apnea (OSA) is high in veterans with posttraumatic stress disorder (PTSD). Our previous research on OSA in Vietnam-era veterans found that 69% had an AHI≥10 (Yesavage, 2012). Efficacious treatments are available for OSA, PAP therapy; however, veterans with OSA frequently fail to use them (Yesavage, 2012; Kuna, 2011). Of the veterans diagnosed with OSA, 63% were not using their prescribed PAP device. The reasons for low PAP adherence include discomfort using PAP and psychological barriers. We developed a novel cognitive-behavioral therapy (CBT) intervention to increase PAP adherence in veterans with PTSD and OSA, called CBT-OSA.

Methods: Participants included 37 veterans age 18+ from clinics at VA Palo Alto. Participants were randomly assigned to CBT-OSA or an education arm. All participants received treatment as usual in VA Pulmonary Service or a community-based Sleep Medicine Center. Participants in CBT-OSA received therapy from a Clinical Psychologist. The other veterans received education sessions. All participants received weekly, individual sessions during the first four weeks of PAP treatment. Average mask on time was calculated for each participant during week 1-4 of PAP use.

Results: An independent samples t-test was conducted to compare average mask on time in the CBT-OSA and education conditions. There was a significant difference in the average mask on time for CBT-OSA (M=235.33, SD=139.22) and education (M=136.68; SD=149.19); t(35)=-2.08, p=0.045. These results suggest that veterans who received the CBT treatment increased their PAP use compared to the veterans in the education condition.

Conclusion: CBT-OSA has shown early efficacy. CBT-OSA increased PAP adherence in veterans with PTSD compared to veterans in the education condition. Veterans receiving CBT-OSA demonstrated a longer average mask on time compared to veterans in the education condition. We are following the participants for one year to examine if CBT-OSA fosters long-term PAP adherence.

Support: This research is supported by the Research Service of the Department of Veterans Affairs (Grant Number 1101RX001799-01A2).

0653

POSITIVE EFFECTS OF LONG TERM CONTINUOUS POSITIVE AIRWAY PRESSURE THERAPY ON BLOOD PRESSURE IN OBSTRUCTIVE SLEEP APNEA PATIENTS

Shirahama, R.¹ Tanigawa, T.¹ Tomooka, K.¹ Fan Yun, L.³ Ikeda, A.¹ Wada, H.¹ Kales, S. N.³

¹Department of Public Hearth. Graduate School of Medicine, Juntendo university, Tokyo, JAPAN, ²Department of Public Hearth. Graduate School of Medicine, Juntendo university, Tokyo, JAPAN, ³Occupational medicine Residency, Harverd TH Chan School of Public Health, BOSTON, MA. **Introduction:** Obstructive sleep apnea (OSA) is one of the common causes of hypertension. Therefore, we examine the longitudinal effect of continuous positive airway pressure (CPAP) therapy and its adherence on blood pressure among OSA patients.

Methods: One thousand two hundred ninety-three (male 1,130, female 163) patients, who were diagnosed with OSA and underwent CPAP therapy were investigated for longitudinal changes (24 months observation period) in the levels of blood pressure and body weight. The longitudinal analyses were performed by mixed effect model. Multiple Imputation with Chained Equations was also used to impute missing data. Good CPAP adherence is defined as more than 70% of the time using CPAP more than 4hours at all the measuring. Poor CPAP adherence is defined as less than 70% of the time using CPAP more than 4hours at all the measuring time points.

Results: The patient group with good CPAP adherences), compared to poor CPAP adherence, showed significant diastolic blood pressure reduction in 24 months follow-up period (β =-0.13, p=0.03) despite a lack of significant weight loss (β =-0.02, p=0.59). However, no significant associations were found between systolic blood pressure and CPAP adherence (β =-0.14, p=0.11).

Conclusion: CPAP therapy was found to have a longitudinal effect on diastolic blood pressure despite a lack of significant weight loss. **Support:**

0654

OBSTRUCTIVE SLEEP APNEA IN OLDER ADULTS: GEOGRAPHIC VARIATION IN CPAP TREATMENT

Yu, Y.¹ Levine, R. S.¹ Braley, T. J.¹ Burke, J. F.¹ Chervin, R. D.¹ Dunietz, G. L.¹

¹University of Michigan, Ann Arbor, MI, ²University of Michigan, Ann Arbor, MI, ³University of Michigan, Ann Arbor, MI.

Introduction: Obstructive sleep apnea (OSA) is prevalent and consequential among older adults. Positive airway pressure (PAP) is likely to reduce associated morbidity, but adherence is inconsistent. Regional treatment variations that may reflect addressable differences in care are not sufficiently studied. We examined geographic variations in PAP treatment among older US adults.

Methods: A representative 5% sample of all Medicare beneficiaries, age 65+, enrolled in fee-for-service program in 2013 was analyzed. OSA diagnosis was defined by ICD-9 codes. PAP treatment was identified by HCPCS codes. Treatment adherence was defined as \geq 2 HCPCS claims for PAP supplies on separate months. We examined state-specific proportions of Medicare beneficiaries with OSA who obtained PAP and showed adherence. Maps were created to represent state-specific proportions of beneficiaries who were treated and adherent, by quantiles. To examine more granular regional variations, we created maps representing hospital referral region (HRR)-specific proportions of treated among diagnosed, and adherent among treated. Scatterplots were used to identify the relationship between proportions of PAP treatment and adherence, by state.

Results: For the state-level data, PAP treatment and adherence rates were between 54%-87% and 59%-81%, respectively. Midwest states had higher CPAP treatment proportions (>80%), while Northeast, Southwest and Southern states had CPAP treatment rates <73%. State-level CPAP adherence showed similar patterns, with lowest rates in southern states and California (<70%). Within-state variability of treatment patterns were observed, especially along the east and the west coasts. A scatterplot revealed that state-level

CPAP treatment and adherence rates were linearly correlated, with Washington D.C., NY and NJ ranked lowest. In contrast, MT, ND and VT had the highest treatment and adherence rates.

Conclusion: These data show substantial state-level and regional variability of CPAP treatment and adherence among Medicare beneficiaries. Some geographic areas may merit prioritization in efforts to improve OSA treatment and adherence.

Support: This study was supported by The American Sleep Medicine Foundation Strategic Research Award 115-SR-15

0655

CBT-I AND CPAP IN COMORBID INSOMNIA AND SLEEP APNEA: EFFECTS ON DAYTIME FUNCTIONING

Tu, A. Y.¹ Crawford, M. R.² Dawson, S. C.¹ Fogg, L. F.³ *Turner, A. D.*⁴ Wyatt, J. K.⁵ Crisostomo, M. I.⁶ Chhangani, B. S.⁷ *Kushida, C. A.*⁸ Edinger, J. D.⁹ Abbott, S. M.¹ Malkani, R. G.¹ *Attarian, H. P.*¹ Zee, P. C.¹ Ong, J. C.¹

¹Center for Circadian and Sleep Medicine, Department of Neurology, Northwestern University Feinberg School of Medicine, Chicago, IL, ²School of Psychological Sciences and Health, University of Strathclyde, Glasgow, UNITED KINGDOM, ³College of Nursing, Rush University Medical Center, Chicago, IL, ⁴Center for Sleep and Brain Health, Department of Psychiatry, New York University, New York, NY, ⁵Department of Psychiatry and Behavioral Sciences, Rush University Medical Center, Chicago, IL, ⁶None,., IL, ⁷Department of Medicine, Rush University Medical Center, Chicago, IL, ⁸Division of Sleep Medicine, Department of Psychiatry and Behavioral Sciences, Stanford University, Stanford, CA, ⁹Division of Pulmonary, Critical Care, and Sleep Medicine, Department of Medicine, National Jewish Health, Denver, CO.

Introduction: This study examines the effects of treatment sequences using cognitive-behavioral therapy for insomnia (CBT-I) and continuous positive airway pressure (CPAP) therapy on daytime functioning in people with comorbid insomnia and sleep apnea (COMISA).

Methods: 118 participants with COMISA (Age=49.99 \pm 13.12; 53.4% female) were randomized to one of the three study arms: Arm A- CBT-I followed by CPAP, Arm B- CBT-I concurrent with CPAP, and Arm C- CPAP only. Participants were assessed at four time points [baseline/ start of phase 1 (A1), CPAP titration/ start of phase 2 (A2), 30 days (A3) and 90 days (A4) after CPAP initiation]. This study examined secondary outcome measures of daytime functioning, including the Functional Outcomes of Sleep Questionnaire (FOSQ), Epworth Sleepiness Scale, and Flinders Fatigue Scale (FFS).

Results: Linear mixed model analyses showed a main effect of time on improving functional outcomes in all measurements, with all p< 0.001. There were also arm by time interactions on FOSQ [F(6, 105.36)=4.21, p=0.001] and FFS scores [F(6, 106.95)=3.10, p=0.008]. Pairwise comparisons with Bonferroni adjustment showed improved FOSQ scores in Arm A from A1 to A2 (p=0.011) and A2 to A3 (p=0.005), Arm B from A2 to A3 (p< 0.001), and Arm C from A2 to A3 (p=0.006). For FFS scores, improvements were shown in Arm A from A1 to A2 (p=0.003), and Arm B from A2 to A3 (p< 0.001).

Conclusion: The results show daytime functioning improvements in patients with COMISA following CPAP and CBT-I. In addition,

CBT-I appears to facilitate improvement in sleepiness-related functional status and daytime fatigue. The findings suggest that the combination of CBT-I and CPAP may have a beneficial effect on daytime functioning in patients with COMISA.

Support: This study was supported by the National Institutes of Health (R01HL114529).

0656

MORTALITY AND HOSPITALIZATION IN PATIENTS WITH HEART FAILURE AND SLEEP APNEA: A RETROSPECTIVE STUDY OF POSITIVE AIRWAY PRESSURE THERAPY IN MEDICARE BENEFICIARIES

Huang, F.¹ Patel, S. L.² Combs, D.³ Parthasarathy, S.² ¹Philips Healthcare, Cambridge, MA, ²2Division of Pulmonary, Allergy, Critical Care, and Sleep Medicine, Department of Medicine, University of Arizona, Tucson, AZ, ³3 University of Arizona Health Sciences, Center for Sleep and Circadian Sciences, Tucson, AZ.

Introduction: Sleep apnea is common in patients with heart failure (HF) and can be treated with positive airway pressure (PAP) therapy. In patients with both HF and sleep apnea, whether treatment of PAP therapy is associated with reduction in hospitalization or mortality is unclear.

Methods: We used 5% Medicare limited-dataset (LDS) from 2013-2015 to perform a retrospective study of hospitalizations and mortality in HF patients with sleep apnea who received or did not receive PAP therapy over an 18-month time period. All-cause mortality during post-treatment period, any and HF-related hospitalizations in baseline, pre-treatment and post-treatment periods were measured and compared. Propensity score matching, generalized estimating equations (GEE) model for repeated measures analysis and COX-survival analysis adjusted by multiple covariates were used for longitudinal comparisons and mortality.

Results: We have identified 281,161 patients with at least two distinct HF onsets and 62,800 of them had sleep apnea diagnosis (22%). Of these patients, 5,540 of them had initiated their PAP therapy while 12,129 of them only had their sleep apnea diagnosis during the selection time frame from Jan 1st, 2014 to June 30th, 2015 without PAP treatment (control group). After adjusting for various confounders and propensity score matching, bilevel PAP was strongly associated with lower hospitalization and HF-associated hospitalization (Bilevel-PAP vs. Control: Any hospitalization, OR=0.62, 95%CI=0.53-0.74, p<0.0001; HF-associated hospitalization, OR=0.65, 95%CI=0.55-0.78, p<0.0001). Cox proportional hazards survival analysis revealed that all of the PAP-treated groups had a better 6-month survival after treatment initiation when compared to controls (any PAP therapy vs. Control: HR=0.32, 95%CI=0.28-0.37, p<0.0001).

Conclusion: In a retrospective analysis, PAP therapy was associated with lower 6-month all-cause mortality among Medicare beneficiaries with HF and sleep apnea. Bilevel-PAP therapy was consistently associated with significant reduction in hospitalization among these patients. Our observational findings warrant confirmation by future prospective intervention trials.

Support: NIH (HL126140; MD011600; HL138377; HL140144; IPA-014264-00001), PCORI (PPRND-1507-31666; PCS-1504-30430), American Academy of Sleep Medicine Foundation (169-SR-17), and Philips Healthcare/ Philips-Respironics Inc.

0657

IMPACT OF TREATMENT WITH MANDIBULAR ADVANCEMENT ORAL APPLIANCE ON RESPIRATORY PARAMETERS, SLEEP AND CARDIOMETABOLIC RISK FACTORS OF CPAP NON-ADHERENT PATIENTS WITH SEVERE OBSTRUCTIVE SLEEP APNEA

Giannasi, L.¹ Gomes, M.² Oliveira, L.³ Nacif, S.⁴ Oliveira, E.⁴ Rezende, T.¹ Dutra, M.² Bacigalupo, E.² Soviero, L.² Nazário, L.² Oliveira, W.¹ Rode, S.¹ Amorim, J.² Salgado, M.² Meira e Cruz, M.⁵ ¹COAT - Institute of Science and Technology, São José dos Campos, BRAZIL, ²CEBAPE - Institute of Science and Technology, São José dos Campos, BRAZIL, ³University Center of Anápolis-UniEnvangélica, Goiás, BRAZIL, ⁴Hospital do Servidor Público Estadual de São Paulo - IAMSPE, São Paulo, BRAZIL, ⁵Sleep Unit, Cardiovascular Center of University of Lisbon, Lisbon School of Medicine, Lisbon, PORTUGAL.

Introduction: Obstructive sleep apnea (OSA) may trigger systemic changes linked to important cardiometabolic risk factors such as hypertension, stroke and diabetes II. As a life-threatening, multifactorial disorder, OSA demands a multiprofessional approach. The most common worldwide treatments are Continuous Positive Airway Pressure (CPAP) and Mandibular Advancement Oral Appliance (OAm). The aim of this study was to evaluate the impact of OAm treatment on CPAP non-adherent patients with severe OSA, comparing objective and subjective data between baseline and follow up.

Methods: A prospective study was carried out including nonadherent severe OSA patients, which were referred to OAm therapy evaluation. Patients presenting with snoring, gasping/ choking during sleep, fatigue and daily sleepiness were evaluated by a sleep medicine specialist and the diagnosis of severe OSA with a basal polysomnography (PSG). All the patients were treated with a standard OAm (PMPositioner®). Baseline and Follow up (6 months) sleep parameters (PSG and Epworth Sleepiness Scale -ESS) were compared to assess treatment efficacy.

Results: Seventeen patients (9 with hypertension and 8 with hypertension + diabetes) met the inclusion criteria and 13 finished the protocol. After treatment with OAm the following parameters improved significantly: OSA severity (44.5 \pm 13.5 to 9.0 \pm 4.3, p≤0.001), ODI (46.8 \pm 11.6 to 12.1 \pm 9.1(p<0.05)), REM (18.4 \pm 4.8 to 21.5 \pm 2.9 (p<0.05)) and SaO2nadir (75.7 \pm 9.4 to 87.0 \pm 3.6, p<0.001), ESS (p<0.005). Ten patients (58%) reported a reduction either in systolic and diastolic blood pressure with 3 of them (30%) reduced the hypertensive drug dose.

Conclusion: Our findings show that OAm is a safe and effective treatment option to CPAP non-adherent severe OSA patients. Furthermore OAm therapy had also a positive impact on cardiometabolic risk factors which are particularly relevant outcomes in OSA patients.

Support: State of Sao Paulo Research Support Foundation (FAPESP).

0658

THE EFFECT OF TEXT MESSAGE REMINDERS ON POSITIVE AIRWAY PRESSURE ADHERENCE IN VETERANS WITH OBSTRUCTIVE SLEEP APNEA

Al Saleh, Q. Kim, D. Jordan, K. Balish, M. Kataria, L. Veterans Affairs Medical Center, Washington, D.C., Washington, D.C.

Introduction: Adherence to positive airway pressure (PAP) therapy continues to be a challenge. The main objective of this study was to

determine whether mobile text-message reminders increased PAP adherence in veterans with obstructive sleep apnea.

Methods: This is a retrospective study of 25 patients with poor adherence to PAP therapy who used the Veterans Affairs ANNIE app to receive nightly mobile text message reminders. PAP adherence was measured at baseline, 1,and 3 months. A one-way repeated measures ANOVA was used to compare the effect of time on PAP adherence, specifically the percentage of overall days used and percentage use >4 hours. A two-way repeated measures ANOVA without replication was used to determine the effects of AHI severity and time on PAP adherence.

Results: Our sample demographics included a mean age of 59 \pm 12 years, mean BMI of 33.5% \pm 5.4, and mean AHI of 26.3 \pm 25.4. The mean PAP overall percentage use and mean use >4 hours respectively were 32% \pm 27 and 18% \pm 19 at baseline, 66% \pm 27 and 45% \pm 30 at 1 month, and 54% \pm 32 and 37% \pm 30 at 3 months. After three months of receiving nightly text-message reminders, there was a statistically significant effect of time on percentage overall PAP use, F (2, 48)=21.54, p=2.10E-07, as well as in the percentage PAP use >4 hours, F (2, 48)=22.05, p=1.61E-07. A two-way repeated measures ANOVA without replication yielded a main effect of the interaction of time with PAP adherence, F (2, 48)=22, p=<0.0001. AHI severity was not a significant factor.

Conclusion: Nightly text message reminders significantly improved PAP adherence from baseline to 3 months. Though PAP adherence was not optimal, it doubled initially in our sample. Further studies are warranted to determine how text-messages can be used long term to improve adherence.

Support: Nil

0659

A SAFE AND PRECISE TONGUE BASE SURGERY FOR OBSTRUCTIVE SLEEP APNEA: REAL-TIME INTRAOPERATIVE ULTRASOUND-ASSISTED TRANSORAL ROBOTIC SURGERY

Lin, C.^{1,2} Chang, C.¹ Hsiao, J.¹ Wu, J.¹ Tsai, H.³ ¹ENT Department, National Cheng Kung University Hospital, Tainan, TAIWAN, ²Sleep Medicine Center, National Cheng Kung University Hospital, Tainan, TAIWAN, ³Spring-Sun Psychiatric Clinic, Tainan, TAIWAN.

Introduction: Lingual artery (LA) injury is a devastating complication of tongue base surgery. Compared with the anatomic findings of computed tomography angiography (CTA), intraoperative blade of mouth gag might change the thickness of base of tongue (BOT) and anatomy of LA. We aimed to investigate the position of LA in the BOT with intraoperative ultrasound (IOU) imaging during transoral robotic surgery (TORS), and evaluate the bleeding complications when assisted with / without IOU.

Methods: Adult obstructive sleep apnea (OSA) patients who received TORS in BOT resection were recruited since 2016. Assessment tools were pre-op over-night hospital polysomnography (PSG) and anatomy-based Friedman Staging System. Ultrasound imaging was utilized to identify anatomic parameters of LA in BOT, including distance to midline, arterial depth and diameter.

Results: Ninety-three OSA patients (82 male, 88.2%) were analyzed. The mean age was 42.2 ± 10.0 years old and body mass index was 29.2 ± 4.5 kg/m². The average apnea hypopnea index (AHI) was 58.1 ± 21.4 events/hour. There were 66 (71.0%), 24 (25.8%) and 3 (3.2%) patients in Friedman stages II, III and IV, respectively. Seventy patients underwent TORS with IOU had shorter operation time (191.7 ±3.8 minutes) than 23 patients without IOU (220.1 ±6.6

minutes), less total blood loss $(11.3\pm10.8$ versus 19.6 ± 26.7 ml), and more BOT tissue reduction volume $(7.1\pm2.5$ versus 3.9 ± 1.6 ml). Significant predictors of arterial depth were higher AHI level during rapid-eye-movement (REM) sleep stage (p=0.038), bigger tonsil size (p=0.034) and more elevated Friedman tongue position (p=0.012). Postoperative complication associated with LA injury was not found in the patients with use of IOU.

Conclusion: When tongue retracted with blade, the distance to midline and depth of LA were altered in BOT. With IOU assisted, surgeon could identify LA position confidently. It is expectable to maximize efficiency and minimize catastrophic bleeding complications when OSA patients received TORS in BOT resection. **Support:** nil

0660

THE ASSOCIATION BETWEEN SLEEP-RELATED BREATHING DISORDERS AND FREE FLAP RECONSTRUCTION SURGERY IN PATIENTS WITH ORAL AND OROPHARYNGEAL CANCERS DURING 6-MONTH FOLLOW-UP

Fu-Hsin, L.¹ Chan-Chi, C.¹ Yu-Cheng, L.¹ Wei-Shu, L.¹ Cheng-Yu, L.¹

¹National Cheng Kung University Hospital, Tainan, TAIWAN, ²National Cheng Kung University Hospital, Tainan, TAIWAN.

Introduction: Little was known about the association between sleep-related breathing disorders (SRBDs) and oral and oropharynx cancers (OOCs). To clarify the impact of free flap reconstruction on SRBDs, we performed a pilot study to investigate the change of SRBDs severity in patients with OOC before and after flap reconstruction surgery.

Methods: This study recruited 15 patients who were newly diagnosed with OOCs and expected to receive free flap reconstruction surgery. For each participant, polysomnography tests were performed repeatedly at the time of pre-operative, post-operative 1-week, and post-operative 6-month periods.

Results: All the subjects were male. Median age was 56 years (range 37-68). Mean of body mass index (BMI) was 24.5 (SD 5.8). Pre-operative apnea-hypopnea index (AHI) was 21.1/hour (SD 20.1). During post-operative 1-week period, BMI was 24.1(SD 5.8) and AHI was 40.2/hour (SD 27.9). During post-operative 6-month period, BMI was 23.4 (SD 3.3) and AHI was 33.3/hour (SD 21.6). Comparison between pre-operative and post-operative 6-month periods, there was no significant difference in BMI, but AHI increased significantly (21.1/hour v.s. 33.3/hour, P = 0.01).

Conclusion: Our study showed that OOCs patients with free flap reconstruction surgery had significantly increased AHI level during post-operative 1-week period. The SRBDs severity became partial remission after 6 months. We recommend that the head and neck cancer team should pay attention to the SRBDs issues in OOCs patients with free flap reconstruction surgery. **Support:**

0661

COMPARISON BETWEEN VENTILATOR DETECTED APNEA HYPOPNEA INDEX AND MANUAL SCORED RESULTS

Ni, Y.¹ Dunhsm, K.¹ Cunningham, L.¹ Thomas, R.¹

¹Beth Israel Deaconess Medical Center, Brookline, MA, ²Beth Israel Deaconess Medical Center, Brookline, MA.

Introduction: The apnea hypopnea index and percentage of periodic breathing detected by the ventilator machine are often used by sleep doctors to evaluate whether sleep apnea has been adequately treated or need further interventions. There are concerns about the accuracy of this autodetection.

Methods: Patients with sleep apnea who were treated with positive airway pressure at the Beth Israel Deaconess Medical Center (Boston) and tracked by the EncoreAnywhere system were included. The machine detected AHI(AHIm) and PB(PBm) were extracted from the first week data in every month from the start of use. The manual scored AHI(AHIs) and PB(PBs) were calculated from the last waveform graph during every month. The apnea hypopnea index as well as periodic breathing in 1st, 2nd, 3rd,6th month AHIm, AHIs, PBm and PBs were compared respectively.

Results: A total of 128 patients were included. The mean age was 56.5 and 66% of them were male. In the first month, the mean AHIs was significantly higher than AHIm, 16.27 vs. 5.36, p<0.001. There was also a large difference between percentage of PBs and PBm, 15.55% vs. 1.96, p<0.001. 78% patients whose AHIm <5 were actually has AHIs >5. The Kappa value for the AHIm and AHIs were 0.074, p=0.069; the value of PBm and PBs was 0.216, p=0.015. In the 2nd, 3rd and 6th months, the mean difference between AHIs and AHIm was 10.58, 10.68, 10.12, respectively. The mean difference between PBs and PBm was 12.32%,11.53%,and 9.18%.

Conclusion: Autodetection of respiratory events consistently under-estimates the severity of residual events. Mean differences remained stable over 6 months. Caution is recommended when attributing non-apnea reasons for residual symptoms in patients with apparently low machine estimated AHI and PB.

Support: This study is supported by American Academy of Sleep Medicine Foundation, category-I award to RJT

0662

HYBRID THERAPIES TO IMPROVE SLEEP APNEA MANAGEMENT

Pogach, M.¹ Cohn, V.² Thomas, R. J.¹

¹Beth Israel Deaconess Medical Center, Boston, MA, ²Sleep Apnea Dentists of New England, Boston, MA.

Introduction: A one-size fits all approach to sleep apnea management, as is promoted by insurance requirements, pervades the field of sleep medicine but does not address individual differences in disease phenotype or treatment tolerance or attempt to achieve meaningful targets for adherence or disease optimization. Continuous positive airway pressure (CPAP) is considered to be the gold standard treatment for sleep apnea, yet CPAP nonadherence rates remain high (estimates at > 30%) while usage goals (at least 4 hours/night) and therapeutic success targets (machine detected AHI < 5) allow for substantial residual disease to persist. Hybrid therapy, combining mandibular advancement device (MAD) and positive airway pressure (PAP), has demonstrated additive effects on lowering the AHI, ODI, and therapeutic PAP pressure in severe OSA patients with pressure intolerance. This analysis explores the impact of hybrid therapy on treatment adherence and optimization, and identifies patient and data characteristics suggestive of benefit from combined therapies.

Methods: In a retrospective analysis, we reviewed the demographic data, medical histories, home sleep test, diagnostic and therapeutic attended polysomnography results, and PAP device settings and data (including usage, leak, residual event index, and waveforms) pre-and post-hybrid approach, in patients treated with hybrid

therapy in our multidisciplinary academic sleep disorders clinic from 2014-2019.

Results: Hybrid therapies utilized include simultaneous (MAD worn together with PAP), alternating (MAD and PAP separately over parts of or on alternating nights), and anchoring (MAD to maintain mouth closure and jaw stability to minimize leak). Preliminary analysis (N=30) shows that hybrid therapy compared to PAP alone improves PAP adherence, lowers residual AHI, minimizes periodic breathing, reduces aerophagia, and lowers therapeutic PAP pressure in these patients. Patient and data characteristics suggestive of benefit include high loop gain sleep apnea, complex apnea, mouth breathing, and position dominance.

Conclusion: Individualizing treatment by combining therapies can result in improved PAP tolerance, usage, and disease control. **Support:**

0663

THE LONGTERM EFFECT OF CPAP COMPARED TO MANDIBULAR ADVANCEMENT DEVICE ON METABOLIC PROFILE IN MILD OBSTRUCTIVE SLEEP APNEA

Togeiro, S. Oliveira, L. S. Guimaraes, T. M. Luz, G. P. Coelho, G. Badke, L. N. Tufik, S. Bittencourt, L.

Universidade Federal de Sao Paulo, Sao Paulo, BRAZIL.

Introduction: Moderate and severe Obstructive Sleep Apnea (OSA) have been independently associated to dyslipidemia with controversial results of improvement with CPAP. Less evidence exists regarding this issue in mild OSA. A current treatment for mild OSA is Mandibular Advancement Device (MAD), however its effectiveness on metabolic profile needs to be compared to CPAP.Our aim was to compare MAD with CPAP and no treatment on metabolic profile during one year in mild OSA.

Methods: Cross sectional analyses included 79 mild OSA patients randomized in CPAP group (n: 31), MAD group (n. 25) and Control group (n: 23). Metabolic profile was investigated before and after 6 and months.

Results: Mean age: 47 ± 9 years, BMI: 28 ± 3.7 kg/m AHI: 9.5 ± 2.9 /h. There were no differences in anthropometric data, total cholesterol (TCT) HDL-C, LDL-C, Triglycerides (TC) and glycated Hemoglobin (Hb1c) among groups. MAD and CPAP reduced AHI at 6 and 12 months (9.3 ± 5.2 to 4.2 ± 9.1 to $3.8\pm 12.6/10.0\pm 4.6$ to 1.2 ± 9.9 to 1.7 ± 14.2 , p: 0.01 respectively). BMI did not change in groups at 6 and 12 months. MAD adherence was higher than CPAP at 6 months (5.8 ± 2.8 hs/day vs 3.8 ± 3.0 hs/day; p: 0.01) and 12 months 5.7 ± 2.7 hs/day vs 3.8 ± 3.4 hs/day; p: 0.01). Despite of lower adherence than MAD, CPAP was effective in reduce TCT and LDL- CT at 6 and 12 months (Intention to treat analyses TCT: 189.3 ± 51.4 mg/dl to 186.1 ± 51.4 mg/dl to 174.6 ± 51 mg/dl; p: 0.03 respectively), however HDL-C, TG and Hbc didn't change.

Conclusion: Long term CPAP treatment was effective in reducing cholesterol in mild OSA.

Support: Associação Fundo Incentivo à Psicobiologia (AFIP) CAPES

0664

A LOW AROUSAL THRESHOLD CAUSES BAD SHIFT OF POSITIVE AIRWAY PRESSURE COMPLIANCE OVER TIME IN OBSTRUCTIVE SLEEP APNEA PATIENTS

Wu, H. Wei, Y. Fang, F.

Beijing An Zhen Hospital, Capital Medical University, Beijing, CHINA.

Introduction: To determine the predictive factors of initial and long-term adherence to positive airway pressure (PAP) therapy and which factors leading a shift of good initial compliance to noncompliance.

Methods: In this follow-up study, A cohort of 166 adult patients who underwent polysomnography (PSG) between January 2017 and April 2019, newly diagnosed with obstructive sleep apnea (OSA) and were amenable to PAP therapy were selected. Information on basic demographics, comorbidities and sleep-related symptoms was collected. PAP adherence data were collected at the end of the first week and the third month. After 3 months of follow-up, 142 participants were included for final data analysis.

Results: Pressure levels were stable during 3 months of PAP treatment. Overall average daily usage time and percentage of PAP used days \geq 4 hrs were lower for 3 months than that in the first week. After adjustment for age and gender, multinomial logistic regression analysis showed that less number of sleep-related symptoms (OR, 0.69; 95% CI, 0.52-0.91) and low arousal threshold (ArTH) (OR, 4.44; 95% CI, 1.52-12.98) were associated with a higher odds of consistent noncompliance of PAP; Low ArTH (OR, 2.87; 95% CI, 1.09-7.57) and less BIM (OR, 0.88; 95% CI, 0.78-0.99) increased the risk of compliance-to-noncompliance shift.

Conclusion: Different from the predictors of consistent PAP noncompliance of OSA patients, only less BMI and low ArTH would cause a good PAP compliance shift to noncompliance over time.

Support: Science and technology Beijing 100 leading talents (Z171100001117168)

0665

BILATERAL HYPOGLOSSAL NERVE STIMULATION FOR TREATMENT OF OBSTRUCTIVE SLEEP APNEA

Eastwood, P. R.¹ Barnes, M.² MacKay, S. G.³ Wheatley, J. R.⁴ Lewis, R.⁵ Campbell, M. C.² Jones, A. C.⁶ Palme, C. E.⁷ Petelle, B.⁸ Meslier, N.⁹ Bertolus, C.¹⁰ Denoncin, K.¹¹ Attali, V.¹² Gagnadoux, F.¹³ Launois, S. H.¹⁴

¹University of Western Australia, Centre for Sleep Science, Perth, AUSTRALIA, ²Institute for Breathing and Sleep, Austin Hospital, Melbourne, AUSTRALIA, ³Illawara ENT Head & Neck Clinic, Woollongong, AUSTRALIA, ⁴Depatment of Respiratory and Sleep Medicine, Westmead Hospital, Sydney, AUSTRALIA, ⁵Department of Otolaryngology, Head & Neck Surgery, Royal Perth Hosptital, Perth, AUSTRALIA, ⁶Illawarra ENG Head & Neck Clinic, Woollongong, AUSTRALIA, ⁷University of Sydney at Westmead Hospital, Sydney, AUSTRALIA, 8Service ORL Chirurgie de la Face et du Cou, Hopital Tenon, Paris, FRANCE, ⁹Department of Respiratory and Sleep Medicine, University Hospital of Angers, Angers, FRANCE, ¹⁰AP-HP, Groupe Hospitalier Pitié-Salpêtrière Charles Foix, Paris, FRANCE, ¹¹Nyxoah, S.A., Mont-Saint-Guibert, BELGIUM, ¹²Sorbonne Universite, INSERM, UMRS ¹¹⁵⁸ Neurophysiologie Respiratoire Experimentale et Clinique, Paris, FRANCE, ¹³Department of Respiratory and Sleep Medicine, University of Angers, Angers, FRANCE, ¹⁴Unite de Somnologie et Fonction Respiratoire, Hospital St Antoine, Paris, FRANCE.

Introduction: Hypoglossal Nerve Stimulation (HGNS) decreases obstructive sleep apnea (OSA) severity by contracting the tongue and decreasing upper airway collapsibility. This study assessed the safety and effectiveness of a new implantable device that delivers bilateral HGNS: the GenioTM system.

Methods: The BLAST OSA study (BiLAteral Hypoglossal Nerve Stimulation for Treatment of Obstructive Sleep Apnea), was a prospective, open-label, non-randomized, single arm treatment study conducted at eight centres in three countries (Australia, France, UK). Primary outcomes were the incidence of devicerelated Serious Adverse Events (SAEs) and change in the Apnea-Hypopnea Index (AHI). The secondary outcome was change in the 4% Oxygen Desaturation Index (ODI). Additional outcomes included measures of sleepiness, quality of life, snoring, and device use. Participants were eligible if: 21-75 years old; BMI \leq 32 kg/m²; obstructive AHI 20-60 events/hr and combined central and mixed AHI < 10 events/hr; no positional OSA; no Complete Concentric Collapse of the soft palate during Drug Induced Sleep Endoscopy; and failed to tolerate or accept Positive Airway Pressure treatments. Results: 27 participants were implanted (63% male, aged 55.9±12.0 years, BMI 27.4±3.0 kg/m²). 22 completed the protocol. At 6 months, AHI decreased from 23.7±12.2 to 12.9±10.1 events/hr [p<0.001]; and ODI decreased from 19.1 ± 11.2 to 9.8 ± 6.9 events/hr [p<0.001]. Daytime sleepiness (ESS, p=0.011) and sleep-related quality of life (FOSO-10, p=0.016) both significantly improved. 91% of participants reported using their device >5 days per week, and 77% used it >5 hours per night. The number of bed partners reporting disruptive snoring decreased from 96% to 35%. No device-related SAE occurred. Conclusion: In a targeted population of individuals with moderate-to-severe OSA, the Genio system reduced OSA severity and sleepiness, improved quality of life, and was associated with high adherence and an acceptable safety profile.

Support: This study trial was funded by Nyxoah S.A. This trial was registered with ClinicalTrials.gov, number NCT03048604.

0666

IMPLEMENTING A SLEEP TECHNICIAN SUPERVISED MASK/INTERFACE FITTING SESSION CAN IMPROVE ADHERENCE WITH HOME AUTOMATIC POSITIVE AIRWAY PRESSURE THERAPY

Syed, Z.^{1,2} Mehta, I.³ Khorfan, F.^{1,2}

¹Ascension Genesys Hospital, Grand Blanc, MI, ²Michigan State University College of Osteopathic Medicine, East Lansing, MI, ³Windsor University School of Medicine, Cayon, SAINT KITTS AND NEVIS.

Introduction: Obstructive sleep apnea (OSA) is a common disorder associated with increased risk of motor vehicle accidents in addition to cardiovascular and neurocognitive comorbidities. Home Automatic Positive Airway Pressure (APAP) therapy is becoming a mainstay treatment of OSA in the outpatient setting. It is typically prescribed without any prior supervised titration. Discomfort related to the APAP interface is thought to contribute to poor adherence with home APAP use. We examined whether implementing a dedicated and personalized interface fitting session improves APAP adherence.

Methods: After obtaining IRB approval, 132 adult patients newly diagnosed with OSA were prospectively randomized into two groups (Groups A and B). Group A received a personalized interface/mask fitting session supervised by a sleep technician prior to starting home APAP therapy. During this 30-minute session, patients were educated on APAP use and sampled different masks to address any issues with comfort, leaks, etc. Group B received the usual care where they obtained an interface through Durable Medical Equipment. APAP adherence and interface air leaks during the initial 30 days of home APAP use were compared between the two groups.

Results: Mean APAP adherence during the initial 30 days of APAP therapy was 12.9% higher in Group A compared to Group B; p=0.05. Fewer APAP interface air leaks were present in Group A (14.8 l/min) compared to Group B (21.2 l/min); p=0.04.

Conclusion: Discomfort related to the APAP interface can quickly lead to non-adherence with APAP therapy. Initial experience of APAP treatment is important in determining subsequent use. Our findings demonstrate that implementing a personalized interface fitting session supervised by a sleep technician can improve APAP adherence. Potential benefits of increased APAP adherence include improvement in quality of sleep and in turn improvement in quality of life.

Support: Prior studies have shown that the initial experience of APAP treatment was highly important in determining subsequent use. Patients establish a pattern of APAP adherence during the early days of treatment and maintain this pattern long-term. Early intervention with solutions may help improve APAP adherence.

0667

POSITIVE AIRWAY PRESSURE NON-ADHERENCE INTERACTS WITH POST-TRAUMATIC STRESS DISORDER TO INCREASE RISK OF BACK PAIN

Taylor, K. A.^{1,2} Schwartz, S. W.¹ Sebastião, Y. V.³ Anderson, W. M.^{1,4} Foulis, P. R.^{4,1}

¹University of South Florida, Tampa, FL, ²Gannon University, Ruskin, FL, ³Nationwide Children's Hospital, Columbus, OH, ⁴James A. Haley Veterans' Hospital, Tampa, FL.

Introduction: Altered sleep, as is associated with obstructive sleep apnea (OSA), may increase the risk of back pain (BP). However, little to no evidence regarding the effect of OSA on musculoskeletal pain is currently available, let alone the effect of positive airway pressure (PAP) treatment non-adherence. The purpose of this analysis is to investigate the effect of PAP non-adherence on future BP diagnosis.

Methods: A sample of 1,662 veterans who had a sleep study between January 2003 and October 2006 and receiving PAP treatment for OSA were used for analysis. Measures at baseline included demographic and OSA symptom severity data. Up to 3 weeks of PAP adherence data were collected and patient chart data was collected through May 2010 to determine outcomes. Time was calculated from PAP treatment start to BP diagnosis or censoring, which occurred at date of death or last recorded encounter. Survival analysis was conducted to obtain the hazard ratios (HR) for the effect of PAP non-adherence on BP diagnosis and to investigate whether post-traumatic stress disorder (PTSD) is an effect modifier of this relationship.

Results: PAP treatment non-adherence significantly increased the risk of BP diagnosis (HR 1.88 [95% CI: 1.08, 3.27]) among veterans with PTSD, while non-adherence among veterans without PTSD was not a statistically significant risk factor. Relative excess risk due to interaction (RERI) was 0.97 (95% CI: -0.07, 2.02; p-value=0.068). These estimates are independent of age, sex, race, body mass index, apnea severity (based on Apnea-Hypopnea Index), PTSD diagnosis, income level, and marital status.

Conclusion: PAP treatment non-adherence among veterans with PTSD appears to result in a significant increase in risk of future BP diagnosis. The interaction between PAP non-adherence and PTSD appears to be borderline synergistic. Therefore, improving PAP adherence among veterans with PTSD may decrease risk of future BP diagnosis.

Support: This material is the result of work supported with resources and the use of facilities at the James A. Haley Veterans' Hospital.

0668

IDENTIFYING RISK OF POSITIVE AIRWAY PRESSURE THERAPY NON-ADHERENCE BEFORE BEGINNING THERAPY: THE IMPORTANCE OF FIRST IMPRESSIONS *Shaughnessy, G. F.¹ Morgenthaler, T. I.¹*

¹Mayo Clinic, Rochester, MN, ²Mayo Clinic, Rochester, MN.

Introduction: Prior studies established that compliance during the early days of positive airway pressure (PAP) therapy predicts long-term adherence, which in turn relates to outcomes. However, patient impressions gathered immediately after PAP titration may be the earliest indication of adherence and could help focus efforts at improving compliance. The aim of this study was to examine whether a survey on PAP experience during polysomnography predicts adherence.

Methods: This prospective cohort study included PAP naïve adults with obstructive sleep apnea who had PAP titration during polysomnography and had 30 day adherence data available. Patients answered a pre-existing 5-question survey with dichotomous answers (yes/no) regarding their PAP experience (improved sleep, general discomfort, PAP related discomfort, mask discomfort, willingness to use PAP). Primary outcome was 30 day adherence (average time used per night, percent of nights used, percent of nights with >4 hours use).

Results: We enrolled 47 patients (68% male) with average age of 62.4 years, body mass index 32.6 kg/m², and apnea-hypopnea index 27.8/hr. Patients answered an average of 3.4 ± 1.3 survey questions favorably. The number of favorable answers correlated with percent of nights used and percent of nights with >4 hours use (P<0.05). Patients with improved sleep during PAP titration had a greater percent of nights with >4 hours use (82.4% vs 60.3%; P<0.05). In addition, successful titration during polysomnography (less than 5 events per hour) correlated with improved adherence and less leak after 30 days.

Conclusion: Initial PAP titration experience influences future PAP adherence. Although there was a correlation between answers on our pre-existing questionnaire and outcomes, some questions did not contribute much for this use. We believe a next step would be to develop questions that help predict future usage and better guide therapy. **Support:** None

0669

THE EFFECTS OF CONTINUOUS POSITIVE AIRWAY PRESSURE THERAPY IN MODERATE TO SEVERE OBSTRUCTIVE SLEEP APNEA: A HIGH-DENSITY EEG STUDY

D'Rozario, A. L.¹ Kao, C.² Mullins, A. E.³ Memarian, N.² Yee, B.² Duffy, S.⁵ Banerjee, D.² Cho, G.² Wong, K. K.² Kremerskothen, K.² Chapman, J.² Haroutonian, C.¹ Bartlett, D. J.² Naismith, S. L.¹ Grunstein, R. R.²

¹School of Psychology, Faculty of Science, Brain and Mind Centre and Charles Perkins Centre, The University of Sydney, NSW, Australia., Sydney, AUSTRALIA, ²CIRUS, Centre for Sleep and Chronobiology, Woolcock Institute of Medical Research, University of Sydney, Sydney, Australia., Sydney, AUSTRALIA, ³Icahn School of Medicine, Mount Sinai, New York City, New York, USA., New York, NY, ⁴CIRUS, Centre for Sleep and Chronobiology, Woolcock Institute of Medical Research, University of Sydney, Sydney, Australia., Sydney, AUSTRALIA, ⁵Faculty of Health Sciences, The University of Sydney, NSW, Australia., Sydney, AUSTRALIA, ⁶School of Psychology, Faculty of Science, Brain and Mind Centre and Charles Perkins Centre, The University of Sydney, NSW, Australia., Sydney, AUSTRALIA. **Introduction:** A previous high-density electroencephalography (EEG) investigation in asymptomatic OSA showed regional deficits in sleep EEG power particularly slow wave activity (SWA) during NREM sleep in the parietal region. It is unclear whether treatment with CPAP can reverse local sleep EEG abnormalities in OSA, and whether any recovery is related to improvement in sleep-dependent memory consolidation.

Methods: Fifteen male participants (age 50.4 ± 6.5 yrs, AHI 51.7 ± 23.5 /h) with moderate-severe OSA (AHI>15/h) underwent overnight polysomnography with 256-channel high-density EEG at baseline and following 3 months of CPAP therapy. A word paired associates declarative memory task was administered before and after sleep. After artefact removal, spectral analysis was performed for all channels. Topographical power maps were calculated for standard frequency ranges for NREM sleep (164 channels within a 0.57 radius from the vertex). Maps were compared using both absolute and normalized power (z-scores computed for each subject) and differences between baseline and treatment were determined by statistical nonparametric mapping.

Results: In 11 CPAP compliant patients (intolerant of CPAP [n=3]/ high-density EEG [n=1]), analysis of polysomnographic variables showed that total sleep time did not differ but N1 (baseline vs. treatment: 66.9 vs. 39.5 mins,p=0.008) and N2 (195.0 vs. 150.6 mins,p=0.002) sleep was lower and N3 (89.8 vs. 128.7 mins,p=0.003) was higher after CPAP. Topographic analysis of high-density EEG data revealed a regional increase in SWA (1-4.5Hz) EEG power during N3 sleep in a cluster of electrodes overlying the centro-parietal cortex (cluster mean t-value=2.87,p=0.02). The change in overnight declarative memory consolidation (% recognition) after CPAP was significantly correlated with the change in slow spindle frequency activity in frontal regions (cluster mean r=0.875,p=0.003).

Conclusion: CPAP treatment may enhance localised deficits in sleep EEG activity in OSA, and specific regional recovery may translate to memory improvements in the short-term. These data also highlight the potential for long-term therapeutic effects on cognitive outcomes in OSA.

Support: .

0670

THE OUTCOME OF CPAP TITRATION UNDER DRUG-INDUCED SLEEP ENDOSCOPY: A RANDOMIZED CONTROLLED CROSSOVER TRIAL

Wang, T.¹ Lin, T.¹ Ni, Y.² Lo, Y.¹

¹Chang Gung Memorial Hospital, Taipei, TAIWAN, ²Department of Chest Medicine, Buddhist Tzu Chi General Hospital, Taichung Branch, Taichung, Taiwan, Taichung, TAIWAN.

Introduction: The titration pressure of continuous positive airway pressure (CPAP) is an important issue for patients with obstructive sleep apnea (OSA). The aim of this study was to understand the impact of drug-induced sleep endoscopy (DISE)-guided CPAP pressure and doctor-guided CPAP pressure on patients with OSA. **Methods:** In this randomized controlled single-blinded crossover trial, we compared the effects of 1 month CPAP treatment on patients with OSA. Twenty-four patients with OSA were recruited and completed this study. They all underwent polysomnography, DISE-guided CPAP titration and accommodation. Doctor-guided CPAP pressure was determined before DISE. Patients were randomly assigned to receive DISE-guided or Doctor-guided CPAP pressure treatment for 1 month. Then, they switched to another

CPAP pressure for another 1 month. Epworth sleepiness scale (ESS) will be recorded at baseline, 1 month and 2 months.

Results: The DISE-guided CPAP pressure and Doctor-guided CPAP pressure had no significant difference $(13.9\pm0.7 \text{ cm H}_2\text{O} \text{ vs} 13.5\pm0.5 \text{ cm H}_2\text{O}; \text{P}=0.92$). In addition, residual AHI and compliance were also no significant difference between two groups. The ESS was significantly improved from baseline to 1 month CPAP treatment in both groups. Epiglottis (anterior-posterior collapse) was significant associated with AHI (P < 0.001, by Spearman correlation). Both Epiglottis (anterior-posterior collapse) and tongue base collapse were significant associated with 95% CPAP pressure (P = 0.031 and 0.038). After multivariate regression analyses, epiglottis (anterior-posterior collapse) is the independent factor for 95% CPAP pressure.

Conclusion: The DISE-guided CPAP pressure and Doctor-guided CPAP pressure had no significant difference on the improvement of ESS. Epiglottis (anterior-posterior collapse) is the independent factor for AHI and 95% CPAP pressure.

Support: Chang Gung Memorial Hospital and Chang Gung University

0671

COMBINATION OF TRANSORAL ROBOTIC SURGERY AND OROPHARYNGEAL MYOFUNCTIONAL THERAPEUTIC TRAINING FOR PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Lai, Y. J.¹ Hung, C. H.^{1,2} Lin, C. Y.^{3,4}

¹Institute of Allied Health Sciences, College of Medicine, National Cheng Kung University, Tainan, TAIWAN, ²Department of Physical Therapy, College of Medicine, National Cheng Kung University, Tainan, TAIWAN, ³Department of Otolaryngology, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University, Tainan, TAIWAN, ⁴Sleep Medicine Center, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University, Tainan, TAIWAN.

Introduction: Obstructive Sleep Apnea (OSA) is a type of sleep disorder characterized by intermittent, partial or complete upper airway collapse. Mostly, moderate to severe OSA cases were recommended to treat with continuous positive airway pressure, however, some of them were withdrawn. Transoral robotic surgery (TORS) was considered for OSA patient with tongue base hypertrophy, but the success rate was only 66.9% and the symptoms might relapse because of aging and gaining weights. Myofunctional therapeutic training (MFTT) was also an alternative treatment for patients with mild to moderate OSA. In our study, we investigated the effect of TORS surgery and oropharyngeal MFTT for OSA patients. Methods: Seven adult patients were recruited, who were newly diagnosed with moderate to severe OSA (Apnea-hypopnea Index, AHI, 49.8±27.7/h). Polysomnography, questionnaire (Pittsburgh sleep quality index, PSQI; Snore Outcomes Survey, SOS), and the muscle strengths over tongue and jaw-opening were assessed before TORS surgery, 6-week and 18-week after surgery. The components of MFTT program involved jaw opening, tongue protrusion, tongue left, tongue right, tongue up and tongue down. It began at 6th week after surgery and these patients underwent 12 weeks of the home-based oropharyngeal MFTT. During the training intervention period, subjects were interviewed every week for adjusting the treatment intensity.

Results: Mean age was 45.9 years old (SD 9.8) and body mass index (BMI) was 26.6 (SD 3.6). After combination treatment of

TORS surgery and MFTT, AHI-supine was significantly decreased from 66.3/h to 26.8/h (p<0.05). PSQI and SOS scores were significantly improved (-2.1, 28.8, respectively). Compared with different components of MFTT program, muscle strength of tongue protrusion was the only significant predictor of AHI-supine reduction. **Conclusion:** Our study presented that combination of TORS surgery and oropharyngeal MFTT could improve OSA severity and symptoms. **Support:** This work was supported by National Cheng-Kung University Hospital (grant number NCKUH-10802018).

0672

THE EFFECT OF ARMODAFINIL ON SLEEP SPINDLES IN OBSTRUCTIVE SLEEP APNEA: SECONDARY ANALYSIS OF A RANDOMIZED PLACEBO-CONTROLLED TRIAL

Emami, L.¹ Marshall, N. S.¹ Chapman, J. L.^{1,2} Cho, G.¹ Grunstein, R. R.^{1,4} Yee, B. J.^{1,4} D'Rozario, A. L.^{1,5} ¹CIRUS, Centre for Sleep and Chronobiology, Woolcock Institute of Medical Research, University of Sydney, Sydney, AUSTRALIA, ²Brain and Mind Sleep Team, University of Sydney, Sydney, AUSTRALIA, ³CIRUS, Centre for Sleep and Chronobiology, Woolcock Institute of Medical Research, University of Sydney, Sydney, AUSTRALIA, ⁴Royal Prince Alfred Hospital, Camperdown, Sydney, AUSTRALIA, ⁵School of Psychology, Faculty of Science, Brain and Mind Centre and Charles Perkins Centre, The University of Sydney, Sydney, AUSTRALIA.

Introduction: Armodafinil has been trialed in OSA patients to promote wakefulness and simulated driving performance. We have previously completed a 6-month trial of 150mg of armodafinil vs placebo in moderate-severe OSA patients not using CPAP (ACTRN# 12611000847910) observing that participants on armodafinil learned to perform better across a 90-minute driving simulator task faster than those on placebo. It is possible that this reduction in time-on-task decrement may have been due to increased learning on armodafinil.

Sleep spindles have previously been implicated in procedural learning and neurobehavioral performance. We hypothesized that armodafinil increases sleep spindle events during NREM sleep to enhance learning.

Methods: Sixty-three overweight severe OSA patients (mean BMI: 32.3kg/m2 (26.1-42.5); age 53.1 years (28-71), 52 males) underwent overnight in-lab polysomnography at baseline (0 months) and at a 6-month follow-up. All-night EEG signals were analyzed using a previously validated automated spindle detection algorithm. EEG recordings were visually inspected by an experienced sleep physician (LE), who was blinded to drug allocation. To minimize the likelihood of type 1 error we selected three key spindle variables detected at Cz for analysis of change between 0 and 6 months: 1) total number of spindle events (11-16 Hz) in NREM sleep 2) density of slow spindles (≥ 11 to ≤ 13 Hz) per minute of NREM sleep, and 3) fast spindle density in NREM (>13 to ≤ 16 Hz).

Results: The change in total spindle count in NREM sleep (armodafinil=11.6 vs Placebo =-17.1, p=0.57), fast spindle density (armodafinil=0.06 vs Placebo =-0.02, p=0.63) and slow spindle density (armodafinil=-0.00 vs. Placebo =-0.03, p=0.74) were not increased by armodafinil.

Conclusion: If armodafinil enhances simulated driving performance in a way that suppresses time-on-task effects it does not appear to be through a sleep spindle enhancing mechanism. Armodafinil is probably not a pharmacological method of enhancing sleep spindles. **Support:** World Sleep Society (International Sleep Research Training Program)CIRUS, Centre for Sleep and Chronobiology, Woolcock Institute of Medical Research, University of Sydney

0673

EFFECTS OF SOLRIAMFETOL ON DRIVING PERFORMANCE IN PARTICIPANTS WITH EXCESSIVE DAYTIME SLEEPINESS ASSOCIATED WITH OBSTRUCTIVE SLEEP APNEA

Vinckenbosch, F.¹ Asin, J.² De Vries, N.³ Vonk, P.³ Donjacour, C.⁴ Lammers, G.⁴ Overeem, S.⁵ Janssen, H.⁵ Wang, G.⁶ Chen, D.⁶ Carter, L.⁶ Zhou, K.⁶ Vermeeren, A.¹ Ramaekers, J.¹ ¹Maastricht University, Maastricht, NETHERLANDS, ²Amphia Ziekenhuis, Breda, NETHERLANDS, ³Onze Lieve Vrouwe Gasthuis, Amsterdam, NETHERLANDS, ⁴Sleep-Wake Centre SEIN, Zwolle, NETHERLANDS, ⁵Kempenhaeghe, Heeze, NETHERLANDS, ⁶Jazz Pharmaceuticals, Palo Alto, CA.

Introduction: Excessive daytime sleepiness (EDS) in patients with obstructive sleep apnea (OSA) is associated with an increased risk of driving accidents. Solriamfetol, a dopamine/norepinephrine reuptake inhibitor, is approved in the US (Sunosi[®]) for EDS associated with OSA (37.5-150 mg/day). This study evaluated solriamfetol's effects on on-road driving performance in participants with EDS associated with OSA.

Methods: In each period of this randomized, double-blind, placebo-controlled, crossover study (NCT 02806895; EudraCT 2015-003930-28), driving performance during an on-road driving test was assessed at 2 hours and 6 hours postdose following 7 days of treatment with solriamfetol (150mg/day \times 3, then 300mg/day \times 4) or placebo. The primary endpoint—standard deviation of lateral position (SDLP), a measure of "weaving," at 2 hours postdose—was compared between solriamfetol and placebo per time point using a repeated mixed-effects analysis of variance model.

Results: The study included 34 participants. Baseline characteristics reflected the broader OSA population (88% male; mean age=52 years; mean Epworth Sleepiness Scale score=14.4). SDLP at 2 hours postdose was statistically significantly lower following solriamfetol (least squares [LS] mean [standard error; SE], 18.83cm [0.63]) compared with placebo (19.92cm [0.63]): LS mean difference, -1.08cm; 95% confidence interval (CI), -1.85, -0.32; *P*=0.0062 (incomplete driving tests: solriamfetol, n=1; placebo, n=4), indicating better performance with solriamfetol. At 6 hours postdose, SDLP following solriamfetol (LS mean[SE], 19.24cm [0.63]) was statistically significantly lower compared with placebo (20.04cm [0.63]): LS mean difference, -0.80cm; 95% CI, -1.58, -0.03; *P*=0.0432 (incomplete driving tests: solriamfetol, n=3; placebo, n=7). Common adverse events (\geq 5%) with solriamfetol were head-ache, nausea, insomnia, dizziness, and agitation.

Conclusion: Solriamfetol (300mg/day) improved SDLP, an important measure of driving performance, at 2 and 6 hours in participants with EDS associated with OSA.

Support: Jazz Pharmaceuticals

0674

ASSOCIATION BETWEEN LATERAL WALL COLLAPSE ON DRUG-INDUCED SLEEP ENDOSCOPY AND MRI FINDINGS IN HYPOGLOSSAL NERVE STIMULATOR PATIENTS

Liu, Y.¹ Wiemken, A.¹ Steffen, A.² Schwab, R.¹ Dedhia, R.¹ ¹University of Pennsylvania, Philadelphia, PA, ²University of Lubeck, Lubeck, GERMANY. **Introduction:** Hypoglossal nerve stimulator (HNS) is an effective and safe alternative therapy for obstructive sleep apnea (OSA) in selected patients. Emerging evidence demonstrates that the outcome of HNS is variable, especially for patients with lateral wall collapse on drug-induced sleep endoscopy (DISE). Awake magnetic resonance imaging (MRI) offers detailed visualization of soft tissue. The aim of this study was to determine whether lateral wall collapse on DISE is associated with awake MRI findings in prospective HNS patients.

Methods: Patients from the ADHERE Registry, an international outcomes study for UAS were used for this study. At baseline, awake, supine MRI scans of each subject's head and neck region were collected. The distance between the lateral walls was measured at the level of the hard palate, located by the appearance of the posterior nasal spine, using axial T2 turbo spin echo MRI. DISE assessments of the upper airway were recorded using the VOTE classification. All statistical analyses were performed using SPSS IBM 19.0 software program. Kendall's Tau-b was performed to compare the association between VOTE scoring and MRI findings. **Results:** Twenty-seven patients (N = 3 female, AHI = 28.8 ± 10.5 , BMI = $28.8 \pm 3.8 \text{ kg/m}^2$, age = $53 \pm 9.9 \text{ years}$) were included in this study. The mean overall VOTE score and lateral wall score was 5.6 ± 1.1 and 0.5 ± 0.5 , respectively. The mean lateral wall distance was 18.8±3.2 mm. A significant, inverse association was found between MRI lateral wall measurement and oropharyngeal lateral wall scoring on DISE (T=-.332,p=0.042) but not other anatomic subsites on DISE.

Conclusion: In our study, greater lateral wall collapse on DISE corresponded to narrower lateral airway distance on MRI. The utility of static imaging modalities such as MRI as patient selection tools for HNS warrants further study.

Support: Drs. Dedhia and Schwab receive related support for this project from the National Institutes of Health (NHBLI R01HL144859)

0675

NOCTURNAL OXYGEN SUPPLEMENTATION WITH POSITIVE AIRWAY PRESSURE THERAPY FOR OBESITY HYPOVENTILATION SYNDROME: CLINICAL PREDICTORS AND LIBERATION FROM OXYGEN

Quintos, A. Grewal, R. Lee, A.

Thomas Jefferson University Hospital, Philadelphia, PA.

Introduction: Obesity hypoventilation syndrome (OHS) is associated with a high morbidity and mortality. Many patients require nocturnal supplemental oxygen on top of positive airway pressure (PAP) therapy for hypoxemia independent of apneic events. We need to clinically identify patients likely to require nocturnal oxygen supplementation. Follow up is essential as with adequate control of sleep apnea, hypoxia improves and liberation from nocturnal oxygen supplementation may be achievable.

Methods: Researchers obtained a list of patients with coding diagnosis of OHS, seen at the Jefferson Sleep Center between November 2016 and September 2019. Patients with BMI of \geq 30 and evidence of hypoventilation were included. Hypoventilation was defined as an elevated CO2 level of \geq 45 mmHg on blood gas analysis, elevated serum bicarbonate level of \geq 27 mmol/L or by evidence of nocturnal hypoventilation by AASM criteria on polysomnography. Patients with pulmonary and neuromuscular disorders were excluded

Results: Out of 189 patients reviewed, 36 met the inclusion and exclusion criteria. Nineteen patients (53%) required nocturnal

oxygen supplementation. A higher serum bicarbonate level of 33 mmol/L against 30 mmol/L (p=0.0078) and a lower resting awake SaO2 of 89% versus 95% (p <0.01) were observed in the oxygen supplementation group. In polysomnographic data, the oxygen supplementation group had lower SaO2 nadir of 67% versus 73% (p=0.026) and had a longer time with SaO2 <88% at 238.2 minutes versus 65.5 minutes (p <0.01). Nine out of the 19 patients (47%) underwent nocturnal oximetry on PAP and room air. Of these, 4 patients (44%) were liberated from oxygen.

Conclusion: Fifty three percent of patients with OHS required nocturnal oxygen supplementation on top of PAP therapy. Higher serum bicarbonate level and lower resting awake SaO2 are potential clinical predictors of nocturnal oxygen supplementation. After nocturnal oximetry on PAP, 44% were successfully liberated from supplemental oxygen.

Support:

0676

LONG-TERM EFFECTS OF UPPER AIRWAY STIMULATION FOR TREATMENT OF OBSTRUCTIVE SLEEP APNEA ON MEASUREMENTS OF CENTRAL AND MIXED APNEAS

*Myers, S. A.¹ Sundar, K. M.¹ Strollo, P. J.*² ¹University of Utah, Salt Lake City, UT, ²University of Pittsburgh Medical Center, Pittsburgh, PA.

Introduction: Upper airway stimulation (UAS) of the hypoglossal nerve for obstructive sleep apnea (OSA) is well-tolerated and results in sustained reduction in the apnea-hypopnea index (AHI). Treatment-emergent CSA is reported to occur in 3.5-19.8% of OSA patients treated with CPAP. We aimed to examine the occurrence or emergence of central and mixed apneas in a cohort of participants that received UAS and were followed for 5 years post implantation.

Methods: The Stimulation Trial for Apnea Reduction (STAR) was a Phase III trial evaluating the safety and efficacy of UAS for CPAP-intolerant OSA. Major inclusion criteria were CPAP intolerance, AHI between 20-50, less than 25% central and mixed apneas and BMI <= 32. Polysomnography was performed at baseline, 12, 18, 36 and 60-month follow-up. Data were scored by a core lab and was then retrospectively analyzed via the STAR PSG database to measure the evolution of central and mixed apneas on UAS therapy.

Results: Baseline age was 54.5 \pm 10.2 years, BMI was 28.4 \pm 2.6 kg/m² and 83% male (n=126). AHI data were non-normally distributed. Median AHI was 29.3/hr at baseline, that was reduced to 9/hr at 12-months and 6/hr at 60-months. Median central apnea index (CAI) was 0.8/hr at baseline, 0.4/hr at 12-months, and 0.2/hr at 60-months. Median mixed apnea index (MAI) was 0.2/hr at baseline, 0.7/hr at 12-months and 0.4/hr at 60-months. The 12- and 60-month CAI was significantly lower than baseline (p<0.05), but MAI was not. The percentage of central and mixed events remained stable throughout follow-up, approximately at 5% of the total AHI.

Conclusion: UAS reduced the overall AHI and results in a small but significant decrease in CAI. Given that OSA and CSA frequently co-exist, the role of UAS on reducing CSA in patients with combined OSA and CSA deserves further investigation.

Support: STAR study was sponsored by Inspire Medical Systems

0677

PATIENT PREFERENCES ON INITIATING TREATMENT WITH CONTINUOUS POSITIVE AIRWAY PRESSURE

Borker, P. V.¹ Wyland, C.² Patel, S. R.¹

¹Division of Pulmonary, Allergy, and Critical Care, University of Pittsburgh, Pittsburgh, PA, ²Lake Erie College of Osteopathic Medicine, Greensburg, PA.

Introduction: Clinical guidelines recommend continuous positive airway pressure (CPAP) therapy be initiated in patients with obstructive sleep apnea (OSA) either at home using an auto-titrating device or following manual titration in the sleep laboratory. Patient preference between these two options is unknown.

Methods: Patients newly prescribed CPAP therapy for the treatment of OSA at an academic sleep medicine clinic were surveyed by telephone within one month of initiating treatment. Data on demographics, disease severity and CPAP adherence were obtained from the medical record.

Results: A total of 75 participants (56% male, mean age 52±15 yrs, 48% moderate to severe OSA) were surveyed. Physicians prescribed home initiation of CPAP in 51%, lab initiation in 23%, and allowed the patient to choose in 27% of cases. Overall, 67% of participants (95%) CI [56%-77%]) reported preference for home initiation. Preference for home initiation did not vary by age, sex, AHI, degree of sleepiness, or type of diagnostic study (home vs. lab sleep study) performed (p>0.10 for all). Convenience (44%) and starting treatment faster (44%) were the most common reasons provided for those favoring home initiation, while sleep technician availability (40%), optimization of pressure settings (32%), and ability to try multiple masks (28%) were cited by those favoring lab initiation. The prevalence of CPAP adherence at 90 days tended to be higher in those whose treatment was initiated aligned to preference (79% in those whose treatment initiation was concordant with preference vs. 64% in those whose treatment initiation was discordant, p=0.16).

Conclusion: Overall, two-thirds of patients with OSA prefer initiation of CPAP be done at home. This preference does not vary by demographics, OSA severity or diagnostic modality. Initiating treatment concordant with patient preference may lead to greater adherence.

Support: American Thoracic Society Academic Sleep Pulmonary Integrated Research/Clinical (ASPIRE) Fellowship, K24 HL127307

0678

OBJECTIVE EFFICACY OF ORAL APPLIANCE THERAPY IN AN INTEGRATED CARE MODEL

Stothard, E. R. Deol, L. I. Hickey, M. G. McCarty, D. E. Wertz, A. T.

Colorado Sleep Institute, Boulder, CO.

Introduction: Custom-fabricated Oral Appliance (OA) therapy is a recommended alternative to positive airway pressure for the treatment of obstructive sleep apnea (OSA). However, objective efficacy outcomes are limited as dental sleep medicine professionals often work independently of sleep clinics. The integrated care model (ICM) of sleep medicine includes a full-time dentist (diplomate of the American Academy of Dental Sleep Medicine), making it uniquely positioned to examine the efficacy of OA therapy in reducing apnea-hypopnea index (AHI) and creating individualized, patient-centered treatment plans.

Methods: All patients who completed initial consult for OA therapy with the ICM from 2014-2018 were considered. Patients who declined OA were excluded. Diagnostic testing for sleep apnea was completed prior to OA therapy initiation and efficacy testing was prescribed post-OA therapy.

Results: 795 patients met inclusion criteria. Efficacy testing was completed in 482 patients (60.6%; 33.9% of patients with treatment follow-up but no efficacy testing reported clinical improvement). 99 were excluded due to insufficient data. 383 patients (55% male; 48.4 \pm 0.6 years (\pm SEM), BMI 28.4 \pm 0.3) were included in final analyses. AHI improved in 82.5% of patients (12.5 \pm 0.5 to 7.0 \pm 0.4, p<0.0001). AHI was reduced <5 in 47.5% of patients (9.9 \pm 0.4 to 2.6 \pm 0.1, p<0.0001); AHI was reduced by >50% in 51.4% of patients (13.9 \pm 0.7 to 3.5 \pm 0.2, p<0.0001). 32.1% of patients completed more than one efficacy test to reevaluate adjustment of OA therapy, which significantly reduced final treatment AHI (13.7 \pm 0.9 to 7.6 \pm 0.8, p<0.0001). Among patients with treatment AHI <5 or >50% reduction in AHI, 28.6% and 32.0% of patients, respectively, required multiple adjustments and efficacy tests to achieve optimal results.

Conclusion: Custom-fabricated OA therapy through the ICM significantly reduced AHI and approximately half of patients achieved AHI <5 and/or >50% reduction in AHI as demonstrated by objective efficacy testing. To optimize therapy response, multiple adjustments were required to significantly improve AHI in a subset of patients. This demonstrates the variability of objective efficacy of OA therapy in the management of OSA, and the necessity of repeated objective testing for treatment optimization. **Support:** N/A

0679

EFFECTS OF CANNABIMIMETIC ENHANCEMENT ON SUBJECTIVE SLEEP QUALITY AND FUNCTION IN OBSTRUCTIVE SLEEP APNEA

Yin, G.-.¹ Reid, K.² Carley, D.³ Prasad, B.⁴ Zee, P.²
¹Beijing Tsinghua Changgung Hospital, School of Clinical Medicine, Tsinghua University, Beijing, CHINA, ²Northwestern University Department of Neurology, Division of Sleep Medicine, Chicago, IL, ³Department of Biobehavioral Health Science, University of Illinois at Chicago, Chicago, IL, ⁴Department of Medicine, University of Illinois at Chicago, Chicago, IL.

Introduction: We previously reported that dronabinol a tetrahydrocannobinol, reduced the apnea hypopnea index (AHI) in patients with obstructive sleep apnea (OSA) in a dose-dependent manner. The aim of this report is to assess the effects of dronabinol on subjective sleep quality and daytime function in the Pharmacotherapy of Apnea by Cannabimimetic Enhancement (PACE) II trial.

Methods: By random assignment, 73 adults with moderate or severe OSA received either placebo (N = 25), 2.5 mg dronabinol (N = 21), or 10 mg dronabinol (N = 27) daily, 1 hour before bedtime for 6 weeks. Participants completed the Pittsburgh sleep quality index (PSQI) and Functional Outcomes of Sleep Questionnaire (FOSQ-10) at baseline and at the end of intervention.

Results: Between group comparisons were performed using a one-way ANOVA. At baseline, there were no significant difference between groups in the PSQI or FOSQ-10. When compared to placebo, the 10 mg group had a significant reduction in the global PSQI score (p=0.039). Paired t-test analysis showed, in comparison to the baseline, the subscale and total score of the FOSQ-10 were significantly increased (P=0.005); the global PSQI score, subjective

sleep quality score, habitual sleep efficiency score and daytime dysfunction score were significantly reduced (p<0.001, p<0.001, p=0.024, p=0.007 respectively) in the 10 mg group, while no improvement was found in the 2.5 mg or placebo groups. Bivariate correlational analysis was used to identify the relationship between the changes of variables. Both Δ global PSQI and Δ total FOSQ were correlated with Δ ESS, but not with change in AHI or sleep parameters such as sleep stage percentage, sleep efficiency, arousal index, minutes of wake after sleep onset, time and duration of oxygen saturation below 90% percent.

Conclusion: These findings indicate that in addition to its ability to reduce the AHI, dronabinol can improve subjective sleep quality and daytime function in patients with moderate to severe OSA.

Support: This study was funded by National Institutes of Health, National Heart Lung and Blood Institute Grant Number UM1-HL112856 and National Center for Advancing Translational Sciences, Grant Numbers UL1TR001422 and UL1TR002003.

0680

THE EFFECT OF CONTINUOUS POSITIVE AIRWAY PRESSURE ON THE LEVELS OF THE PROINFLAMMATORY MARKERS IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Wali, S. O.¹ Al-Mughales, J.² Alhejaili, F.³ Manzar, M.⁴ Alsallum, F.³ Almojaddidi, H.³ Gozal, D.⁵

¹King Abdulaziz University Hospital, Jeddah, SAUDI ARABIA, ²Department of medical microbiology and immunology, Jeddah, SAUDI ARABIA, ³Sleep Medicine and Research Center, King Abdulaziz University Hospital, Jeddah, KSA, Jeddah, SAUDI ARABIA, ⁴Physiology Department, College of Applied Medical Sciences, Majmaah University, Saudi Arabia, Majmaah, SAUDI ARABIA, ⁵Pediatric Department, Pritzker School of Medicine, Chicago, USA, Chicago, IL.

Introduction: Obstructive sleep apnea (OSA) develops systemic inflammation, with increased levels of mediators. In general, significant elevations in serum levels of C-reactive proteins (CRP), and interleukin 6 (IL-6) are seen in patients with OSA. The literature is inconsistent regarding the effect of Continuous Positive Airway Pressure (CPAP) on the level of CRP and IL-6. The aim of this study is to evaluate the effect of CPAP on the levels of IL-6 and CRP in patients with OSA.

Methods: All patients newly diagnosed with moderate-severe OSA at the King Abdulaziz university sleep center were targeted. Patients with conditions that may affect the levels of inflammatory markers were excluded. All included patients had their fasting blood taken post the initial diagnostic polysomnography for pro-inflammatory markers. Patients were then classified into two groups. The first group included those that accepted treatment with CPAP (CPAPG). The second group included patients with OSA but refused treatment (No-CPAPG). Each group had been followed for one month, and then another blood samples were drawn for the levels of the pro-inflammatory biomarkers.

Results: Twenty patients in each group were recruited. At baseline IL-6 was significantly higher in the No-CPAPG compared to CPAPG (median 6.7 pg/ml [interquartile range 1.6-7.6] vs. 1.6 pg/ ml [1.3-1.9], respectively; p = 0.006) despite similar OSA severity. At 1-month follow-up, IL-6 remained significantly higher in the No-CPAPG; p = 0.003 and there was no effect of CPAP on IL-6 in the CPAPG. There was no significant difference in CRP level at baseline or at 1-month follow-up among the two groups. However, there was neither an effect of time (F (1, 38) = 0.08, p = 0.78), nor

an interaction effect between CPAP and time (F(1, 38) = 0.006, p = 0.94) on IL-6 level. Similarly, there was neither an effect of time (F(1, 38) = 1.68, p = 0.20), nor an interaction effect between CPAP and time (F(1, 38) = 0.17, p = 0.68) on CRP level.

Conclusion: IL-6 and CRP levels did not change significantly with CPAP over a one month period in OSA patients.

Support: By the DSR, KAU, Jeddah, KSA. No: KEP-2-140-39

0681

COMPARISON OF UPPER AIRWAY STIMULATION OUTCOMES BETWEEN REGIONS AND BMI GROUPS FROM THE ADHERE REGISTRY

Walia, H. K.¹ Mehra, R.¹ Kominsky, A.¹ Kent, D.² Pham, H.² Upender, R.² Manchanda, S.³

¹Cleveland Clinic, Cleveland, OH, ²Vanderbilt University Medical Center, Nashville, TN, ³Indiana University, Indianapolis, IN.

Introduction: As factors influencing Upper Airway Stimulation (UAS) effectiveness in obstructive sleep apnea (OSA) patients are of interest, we compared changes in apnea hypopnea index (AHI) and Epworth Sleepiness Scale (ESS) based on region and baseline body mass index (BMI).

Methods: Patients (15 \geq AHI \leq 65) of the ADHERE registry with AHI at one-year were grouped by region (Europe (EU) vs United States (US)), and BMI (\leq 32kg/m² vs 32-35kg/m²). T-tests and equivalence testing (if the former non-significant) was performed using two-one-sided t-tests. Equivalence margin for AHI was set between -5 and 5 and -2 and 2 for ESS.

Results: By December 2019, 553 of 1600 patients completed 1-year follow-up. Average age was 60±11, 75% male, BMI 29±4 kg/m², ESS=11±6. Median AHI decreased from 33 to 10, median ESS decreased from 11 to 6. Response defined by 50% AHI reduction and <20 was 70%. Both regions had similar improvements in median AHI (EU: 33 to 10, US: 34 to 10, p < 0.001 vs baseline), median ESS (EU: 12 to 7; US: 11 to 6, p<0.001 vs baseline), and treatment response (EU: 71%, US: 68%). The mean AHI and ESS difference between regions met the equivalence margin. (AHI: mean difference: 0.34, CI:-1.78, 2.46, ESS: mean difference: 0.57, CI:-0.04, 1.19). Mean change in AHI at 1-year was equivalent in BMI groups (≤32 kg/m² vs 32-35 kg/m² respectively) median difference: -19.6 vs. -18.8; mean difference: -0.48, (CI:-3.95, 2.97) However, treatment response ratio was different; 73% vs. 60%, p=0.02, i.e. higher BMI patients were less likely to achieve AHI < 20. ESS scores were equivalent; median: 6 vs. 7; mean difference: -0.33, CI: [-1.16, 0.47]. Conclusion: UAS influence on OSA severity defined by AHI and sleepiness was similar irrespective of region and BMI category, however, treatment response defined by 50% AHI reduction and <20 was greater in those with lower BMI.

Support: The statistical support was provided by Inspire Medical System.

0682

EVALUATION OF AN INCENTIVE-BASED INTERVENTION TO IMPROVE 90-DAY ADHERENCE IN PAP-NAIVE PATIENTS

Merchant, G.¹ Valentine, K.¹ Hevener, W.² Willes, L.³ Ta, D.¹ Hernandez, R.¹ Gagnon, R.⁴ Chen, K.⁴ Blase, A.¹ ¹ResMed Science Center, San Diego, CA, ²Sleep Data LLC, San Diego, CA, ³Willes Consulting Group, Inc, Encinitas, CA, ⁴Wellth, Los Angeles, CA. **Introduction:** Although PAP therapy is the gold standard treatment for obstructive sleep apnea, adherence to treatment is suboptimal. Without sustained therapy adherence, patients are at risk of serious negative health outcomes. The objective of this study was to test whether a digitally delivered monetary and social reward program helped patients new to PAP therapy. Financial incentive schemes are effective in helping patients adhere to difficult medication or therapy plans. Additionally, there is an abundance of evidence that social support is a critical component to long-term health behavior change.

Methods: This prospective, randomized, single site pilot is evaluating the effectiveness of an app-based intervention in helping patients adhere to PAP therapy. The financial incentive design leverages loss aversion, and the social incentive design leverages the strength of close ties and variable reinforcement. The primary endpoint is mean PAP usage at 3 months. Secondary endpoints include Medicare compliance, change in functional status, and baseline scores of perceived disease severity, claustrophobia, coping skills, and health literacy as moderators of the intervention's effectiveness. Study recruitment is ongoing, with an expected sample size of 150 subjects.

Results: Of the 132 subjects enrolled, 56% are male, 61% are Caucasian, and 65% are married. The mean age is 49.6 ± 12.0 years and mean BMI is 32.4 ± 8.4 kg/m². Additional demographics such as income level, education level, and number of children along with the primary and secondary endpoints will be presented. A subgroup analysis of the primary endpoint will be generated for subjects identified as strugglers within the first 3 days of usage.

Conclusion: The results of this study will provide insight into methods such as financial and social incentives delivered via a smartphone on initial compliance with PAP therapy, as well as provide more information on the behavioral change associated with beginning PAP therapy.

Support: ResMed

0683

CPAP ADHERENCE RELATIVE TO SLEEP DURATION AND SLEEP PERIOD IN DIFFERENT STUDY POPULATIONS

Deering, S.¹ Shumard, T.² Zamora, T.¹ Martinez, S.³ Stepnowsky, C. J.^{1,5}

¹VA San Diego Healthcare System, San Diego, CA, ²American Sleep Apnea Association, Washington, DC, ³COPD Foundation, Miami, FL, ⁴VA San Diego Healthcare System, San Diego, CA, ⁵University of California, San Diego, La Jolla, CA.

Introduction: CPAP is intended for use during sleep to alleviate disordered breathing. Most patients who use CPAP do so for only a portion of their sleep period, although anecdotally it is known that some also use CPAP while awake. We compared the unusually high levels of CPAP adherence found in a recent study of patients with Overlap Syndrome to a VA clinical population and to participants from the APPLES study.

Methods: CPAP adherence levels were taken from three sources: (1) The O2VERLAP Study, a large comparative effectiveness trial that used two different methods of providing information and support to current CPAP users diagnosed with both OSA and COPD. (2) Combined data from the four most recent clinical CPAP trials conducted at VA San Diego Healthcare System. (3) The APPLES study. Total sample sizes were 332, 957, and 405, respectively. Total sleep time (TST) and total sleep period (TSP) were assessed via

the Pittsburgh Sleep Quality Index (PSQI) for (1) and (2) and via polysomnography for (3).

Results: Mean CPAP use, TST, and TSP for each source were: (1) 6.7, 6.8, & 8.1; (2) 4.0, 6.1, & 7.5; (3) 4.5, 6.6, & 8.0. We examined the ratios of adherence over either TST or TSP, and the ratios for each source were: (1) 98% & 83%; (2) 66% & 55%; (3) 68% & 56%. **Conclusion:** This comparison demonstrates that unlike many CPAP users who tend to use therapy for only a fraction of time spent asleep, patients with COPD and OSA exhibit higher levels of adherence which often exceed sleep time and may be obtaining additional benefits from CPAP use during non-sleep periods. More research is needed both to improve CPAP delivery and support for patients who are using CPAP sub optimally and to understand the factors that account for the heightened levels of CPAP adherence in COPD.

Support: PPRND #1507-31666; IIR 02-275; IIR 07-163; IIR 12-069; PULM-028-12F.

0684

OVERNIGHT FLUID SHIFT IN APNEA AND STROKE: DOES BEING PHYSICALLY ACTIVE MATTER

Frange, C.¹ Coelho, F. M.¹

¹Universidade Federal de São Paulo, São Paulo - SP, BRAZIL, ²Universidade Federal de São Paulo, São Paulo - SP, BRAZIL.

Introduction: Obstructive sleep apnea (OSA) and physical inactivity are frequent in patients with stroke. Overnight fluid shift may increase the propensity to pharyngeal obstruction and thus predispose to OSA. Also, physical inactivity can lead to consequent edema in the hemiparetic side.

Methods: In 7 patients at 3 months following post-first-ever ischemic stroke, we measured change in AHI (PSG), neck circumference, and arms and legs fluid volume (bioelectric impedance), before and after PSG in two conditions during the day before PSG: (1) inactive (sitting/lying), and (2) physically active (standing, walking, climbing stairs and mild exercise for 30 mins/hour).

Results: Being physically active for one day decreased mean AHI (inactive: 17.9/h; active: 12.6/h). Mean neck circumference increased overnight in both conditions (active and inactive) but increased more when the participants were physically active (inactive: 38 to 38.5cm; active: 38.43 to 39.5cm). Mean paretic arm fluid volume increased after inactive condition and did not change after physically active condition (inactive: 1.33L to 1.45L; active: 1.82L). Mean non-paretic arm fluid volume increased after active and inactive conditions (inactive: 1.34L to 1.45L; active: 1.74 to 1.78L). In both conditions and for paretic and non-paretic arms, being physically active for one day increased arm fluid volume between pre- and post-sleep comparisons. Mean paretic leg fluid volume increased after both conditions but increased more when the participants were inactive (inactive: 6.08L to 6.52L; active: 6.12 to 6.24L). Mean non-paretic leg fluid volume increased after inactive condition and decreased after being physically active (inactive: 6.01L to 6.45L; active: 6.15 to 6.06L).

Conclusion: Being physically active for one day decreased AHI, despite the increased fluid accumulation in the neck, and contributed to leaving no edema in the hemiparetic side. Breaking up inactivity and increasing physical activity in patients following stroke may be a promising intervention to reduce edema and OSA.

Support: São Paulo Research Foundation (FAPESP, grant #2018/18952-1 to CF).

0685

SAFETY EVALUATION OF AN ALGORITHM DETERMINING NEEDS FOR TREATMENT IN OBESE SLEEP APNEA PATIENT AWAITING BARIATRIC SURGERY: RESULTS OF A PROSPECTIVE 1103 PATIENTS COHORT STUDY

Sériès, F. genest, C. Boutin, I. Marceau, S. Bussieres, J. Minville, C. IUCPQ, Quebec, QC, CANADA.

Introduction: Screening for obstructive sleep apnea (OSA) is recommended before bariatric surgery. We developed an algorithm based on results of nocturnal oximetry and capillary gas. According to this algorithm, CPAP/BiPAP is prescribed only in severe OSA (ODI \geq 25/h) and/or with features of hypoventilation. The objective of this prospective cohort study was to determine the safety of our algorithm comparing peri and post-operative outcomes in patients not receiving pre-operative treatment (without/mild OSA (ODI < 10 /h: controls or ODI 10-24 /h: OSA untreated) and in those receiving CPAP/BiPAP (severe OSA: OSA treated or hypoventilation).

Methods: We collected data from 1103 subjects undergoing bariatric surgery (447 controls; 358 OSA untreated, 289 OSA treated and 9 hypoventilation). For treated patients, a good CPAP/ BiPAP compliance was mandatory for surgery with treatment installed immediately after extubation and continued after. Peri and post-operative outcomes were compared according to apnea status with adjustment for the type of surgery when applicable.

Results: Patients with severe OSA/hypoventilation were significantly older and heavier than other patients. Hypertension and diabetes were less prevalent in controls than in the other groups. No difference was found between the 3 groups regarding occurrence of 10 days reoperation and 30 days rehospitalisation. No difference was found regarding occurrence of cardiopulmonary complications except that admission to ICU was more frequent in patients with hypoventilation and occurrence of cardiac arrhythmia was higher in the OSA/hypoventilation treated patients than in the other groups (2.4%, 11.1 %, 0.4% and 0.6%, p = 0.01). OSA/hypoventilation patients had a longer length of stay in the recovery room (1.7 ± 0.5, 1.9 ± 0.6, 1.5 ± 0.5 and 1.5 ± 0.5 hours p<0.0001) and longer length of hospital stay (2.8 ± 1.8, 3.2 ± 1.1, 2.6 ± 2.1 and 2.6 ± 1.8 days, p<0.0001) than controls and OSA untreated patients.

Conclusion: Our algorithm safely selects patients who don't need treatment before surgery, with no increase in risk of complications following bariatric surgery not treating OSA patients with mild/ moderate sleep apnea without hypoventilation. Patients with severe OSA/hypoventilation, even when correctly treated remain at higher risk of complications.

Support: Fondation IUCPQ-UL

0686

DIABETES SLEEP TREATMENT TRIAL: THE EFFECT OF TREATMENT OF OSA WITH CPAP ON GLYCEMIC CONTROL IN TYPE 2 DIABETES

*Chasens, E. R.*¹ *Sereika, S. M.*¹ *Kortykowski, M.*² *Stansbury, R.*³ *Burke, L.*¹ *Strollo, P. J.*⁴ *Bizhanova, Z.*¹ *Atwood, C. W.*⁴ ¹University of Pittsburgh, Pittsburgh, PA, ²University of Pittsburgh, korytkowski@dom.pitt.edu, PA, ³West Virginia University, Morgantown, WV, ⁴Veterans Administration Pittsburgh Healthcare System, Pittsburgh, PA.

Introduction: Evidence remains unclear whether treatment of OSA with CPAP results in improved glycemic control. This study evaluated if CPAP improved glucose control compared to sham-CPAP and the effect of adherence to active CPAP on glucose control after 6 and 12 weeks of treatment.

Methods: This was a multi-center, double-blind clinical trial. Participants were adults with type 2 diabetes (T2D), A1C \geq 6.5%, apnea + hypopnea index (AHI) \geq 10, and naïve to CPAP. All participants received diabetes education. Glucose control was evaluated with frucostamine and A1C levels; CPAP adherence with a wireless modem system. Statistical analysis followed an "intent-to-treat" approach with linear mixed modeling. The dose of active CPAP was calculated as the percentage of days with active CPAP use \geq 4 hours and the average adherence of active CPAP with sham coded as "0" dose".

Results: Randomized participants (N=98, CPAP=50; sham-CPAP=48) were primarily middle-aged (age=58.7±9.8 years), White (75%), males (57%) obese (BMI=36.2±6.6), suboptimal glucose control (A1C=7.9%±0.9) and OSA (AHI=23.9±14.4). There were no significant baseline differences except in A1C (Active CPAP=7.7%±0.8; sham-CPAP=8.1%±1.0). There was no significant difference in use of their devices at 6 or 12 weeks. Based on linear mixed modeling, participants on active CPAP had improved A1C (b (SE): -.76 (.24), P<.01) and frucostamine (-21.8 (10.5), P=.04) at 6 weeks with A1C trending to significance at 12 weeks (p=0.10). Both the % of cumulative days of active CPAP usage (≥4 hours/day) (.002 (.003), P=.09) and cumulative hours of active CPAP use (.03 (.03), P=.08) showed a trend being associated with greater change in A1C but not in frucostamine (P=.61, P=.51). The rate of change in A1C varied by time, increasing the % of cumulative days of CPAP use (≥4 hours/day) at week 6 predicted greater change in A1C (.006 (.002), P=.01) than week 12 (.002 (.003), P=.38). Higher average hours of CPAP usage were associated with greater change in A1C (.08 (.03), P=.01) at week 6 compared to week 12 (.03 (.03), P=.47).

Conclusion: In our study, individuals with T2D and OSA, adherence to active CPAP use improved glycemic control over 6 weeks. **Support:** NIDDK grant R01DK096028; CTRI grant UL1TR001857 and UL1TR000005.

0687

COMPARATIVE CHANGES OF PATIENT REPORTED OUTCOMES IN POSITIVE AIRWAY PRESSURE AND UPPER AIRWAY STIMULATION FOR OBSTRUCTIVE SLEEP APNEA

Bhambra, R.¹ Pascoe, M.¹ Kominsky, A.² Mehra, R.¹ Aylor, J.¹ Wang, L.³ Phillips, K.¹ Waters, T.¹ Walia, H. K.¹

¹Sleep Disorders Center, Cleveland, OH, ²Department of Otolaryngology, Cleveland, OH, ³Department of Quantitative Health Sciences, Cleveland, OH.

Introduction: Upper Airway Stimulation (UAS) is increasingly being used for obstructive sleep apnea (OSA) treatment, however, data comparing changes in patient reported outcomes (PROs) in response to positive airway pressure (PAP) versus UAS are limited. We hypothesize that there will be no difference in PROs between the two groups after treatment.

Methods: UAS and PAP groups were 1:3 matched on age, sex, Body Mass Index (BMI) and Apnea Hypopnea Index (AHI, category 15-30, >30). Linear mixed models assessed the difference of change in Epworth Sleepiness Scale (ESS), Functional Outcomes of Sleep Questionnaire (FOSQ), Patient Health Questionnaire (PHQ9) and Insomnia Severity Index (ISI) measures on matched strata of UAS versus PAP groups with adjustment of baseline and matching factors. All analysis was performed in SAS software (version 9.4, Cary, NC).

Results: The analytic sample comprised 193 PAP patients and 69 UAS patients, with mean age=62.9+/-9.4 years, 27.5% female, mean BMI=29.1+/-3.2kg/m², and median AHI 42.7, IQR: 31.5, 57.2. ESS in PAP (n=190) reduced by -2.63 (-3.38,-1.88) and in UAS (n=56) reduced by -2.22 (-3.34, -1.10), with a mean difference of 0.41 (-0.70, 1.52, p=.46). FOSQ in PAP (n=188) showed a change of 1.38 (0.99, 1.78) and in UAS (n=49) a change of 1.82 (1.17, 2.46), with a mean difference of 0.43 (-0.23, 1.09, p=.19). PHQ9 in PAP (n=185) showed a significant change of -2.24(-3.00, 1.47) and in UAS (n=45) a change of -3.75(-5.07,-2.42), with a mean difference of -1.51(-2.93,-0.088, p=.038). ISI in PAP (n=193) showed a significant change of -3.20(-4.39,-2.02) and in UAS (n=47) a change of -4.83(-6.77,-2.90), with a mean difference of -1.63(-3.62, 0.37, p=.11).

Conclusion: Similar improvements in PROs were observed in both UAS and PAP patient groups, however UAS appeared to confer greater benefit in depressive symptoms relative to PAP. Randomized clinical trials should be designed to confirm these findings. **Support:** N/A

0688

EVALUATION OF A NOVEL NASAL AIRWAY STENT FOR SNORING AND OSA TREATMENT BY PROSPECTIVE JAPANESE PATIENTS

Wang, W.¹ Ding, F.² Satoh, M.³ Kushida, C.¹

¹Stanford University, Stanford, CA, ²Cleveland Clinic, Cleveland, OH, ³University of Tsukuba, Tsukuba, JAPAN.

Introduction: This cross-sectional study evaluated a novel nasal airway stent (NAS) in the form of a single-use, disposable device (naśtent® classic, Seven Dreamers Laboratories, Inc., Tokyo, Japan), which represents a significant advancement in NAS technology for the treatment of OSA.

Methods: 1353 individuals whom were interested in NAS treatment for their snoring and/or OSA were enrolled in this study conducted in different districts in Tokyo, Japan from 8/21/2015 to 3/7/2016. A total of 1335 participants with complete data were included in the final dataset. Collected data included demographic features, self-reported sleep and OSA characteristics, anatomic traits, Epworth Sleepiness Scale (ESS) scores, and NAS tolerance. Results: The majority of the participants were middle-aged men (91.5% male, 45±10.8 years) with normal BMI (24.5±5.7). Selfreported sleep was 9-11 hours (0.1%), 7-9 hours (8.2%), 5-7 hours (66.7%), 3-5 hours (22%), 1-3 hours (1.2%), and unreported (1.9%). Their mean Epworth Sleepiness Scale score was 9 ± 5.56 , and their peak Mallampati and tonsillar grading scores were 2/4 and 3/5, respectively. Of the participants, 87.4% were aware of possible OSA either by snoring or by prior diagnosis of OSA, and their reasons for desiring use of NAS therapy presented in a similar ratio. Over 80% of the participants had never been treated for their diagnosis of OSA; for those with current or prior treatment for their OSA, 62.9% reported PAP as their primary therapy, followed by oral appliances (25.7%), surgery (0.5%), and other therapies (10.8%). On a scale of 1 to 5 where 5 indicated good tolerance to the NAS, the mean scores were 4±1.3 for easy of insertion and 4±1.2 for convenience of use.

Conclusion: This large, cross-sectional study indicated that the majority of individuals seeking a novel NAS treatment for their

snoring and/or OSA in Tokyo, Japan were middle-aged men with 5-7 hours of self-reported sleep whom had some daytime sleepiness, displayed signs of mild anatomic upper airway narrowing, had not been treated for their OSA, and whom felt that the NAS was easy and convenient to use.

Support: Seven Dreamers Laboratories, Inc. and WSS ISRTP

0689

USE OF AN AUTOMATED SCANNING SYSTEM TO SELECT A PATIENT INTERFACE

Hardy, W.¹ Jasko, J.¹ Bogan, R.³

¹Philips Respironics, Monroeville, PA, ²Philips Respironics, Monroeville, PA, ³SleepMed of South Carolina, Columbia, SC.

Introduction: There is no universal process for selecting mask style, size, and fit, and there is considerable variance in clinician and patient mask preference and patient anatomy. Poor mask fit may negatively affect adherence. A three-dimensional (3D) facial scanner and proprietary analytical software were developed to bring efficiencies to mask selection. This study explored the impact of that system on initial mask success compared to standard practice.

Methods: This was an open-label, randomized-controlled study. Participants provided written informed consent. **3D Scanner Arm** (**3DA**): Participants answered questions about sleeping habits then had 3D facial images taken. Proprietary software recommended a hierarchy of up to four Philips Respironics masks and sizes. **Traditional Fitting Arm (TFA)**: A designated clinician selected and fit masks using their standard methods. Mask selection was assessed by applying therapy and soliciting patient and clinician feedback. Mask refits and adherence were tracked through 90 days. Five sleep centers recruited 115 participants into the 3DA (61 males, 51.1 ± 13.4 years, BMI 35.2 ± 7.0 , diagnostic AHI 26.2 ± 21.9) and 123 into the TFA (79 males, 51.1 ± 11.9 years, BMI 35 ± 7.9 , diagnostic AHI 26.9 ± 22.6).

Results: A significantly higher percentage of 3DA patients required only one mask fitting (with no refits) compared to TFA during the initial setup (89.6% vs. 54.5%, p<0.001) and through 90 days (62.6% vs 37.4%, p<0.001). 3DA subjectively rated confidence in and satisfaction with the scanner-selected mask significantly higher than TFA. Mask leak was lower in the 3DA compared to TFA (29.4 \pm 10.6 vs 32.3 \pm 11.4 L/M, p= 0.043). The CMS adherence rate tended to favor 3DA vs. TFA (66.7% vs. 55.3, p=0.083). There were no significant differences in AHI or other adherence metrics.

Conclusion: The 3D scanner system was successful in mask selection with lower mask leak and greater patient satisfaction and confidence. This tool may bring about operational efficiencies to the mask selection process.

Support: This study was sponsored by Philips Respironics

0690

AN EVALUATION OF GENIOGLOSSUS STRENGTHENING ON OBSTRUCTIVE SLEEP APNEA TREATMENT OUTCOMES

Maghsoudipour, M. Bosompra, N. Jen, R. Li, Y. Moore, S. De Young, P. Fine, J. Edwards, B. Gilbertson, D. Owens, R. Morgan, T. Malhotra, A. University of California San Diego, La Jolla, CA.

Introduction: Obstructive sleep apnea (OSA) is characterized by repetitive episodes of pharyngeal collapse. The genioglossus is a major upper airway dilator muscle thought to be important in

OSA pathogenesis. Upper airway (UA) muscle training has reported benefits in some OSA patients. Our goal was to assess the effect of upper airway muscle training on OSA outcomes.

Methods: Sixty five patients with OSA (AHI>10/h) were divided in three subgroups: 1) Treated with auto-CPAP (n=21), 2) Previously failed or refused CPAP therapy (no treatment), (n=24), 3) Currently treated with an oral appliance who still have residual OSA (AHI>10/h), (n=20). All subjects were given a custom-made tongue strengthening device. Within each group we conducted a prospective, randomized, controlled study examining the effect of upper airway muscle training. In each subgroup, subjects were randomized to UA muscle training (volitional protrusion against resistance) or sham group (negligible resistance), with 1:1 ratio over 6 weeks of treatment (twice daily for 20 min/session). In the baseline and the final visit, subjects completed home sleep testing, questionnaires (ESS, PSQI), acoustic pharynogometry, Iowa Oral Performance Instrument (IOPI), and Psychomotor Vigilance Test (PVT).

Results: Results remain blinded; 33 patients received treatment Y and 32 patients received treatment Z. To date, we have not observed a main effect of treatment group on several measures of OSA severity. Some changes in subjective measures over time were observed but difficult to interpret until unblinding occurs.

Conclusion: Treatment of OSA using upper airway muscle training exercises requires further study. Whether muscle training is a viable approach for a definable subset of OSA patients remains unclear. **Support:** R01HL085188-05A1 (U.S. NIH Grant/Contract)

0691

COMPARISON OF ODDS RATIO PRODUCT AND OTHER POLYSOMNOGRAPHIC METRICS AMONG RESPONDERS AND NON-RESPONDERS TO UPPER AIRWAY STIMULATION TREATMENT OF OBSTRUCTIVE SLEEP APNEA

Yu, J. L.¹ Keenan, B. T.¹ Kuna, S. T.² Younes, M.³ ¹University of Pennyslvania - Department of Medicine-Division of Sleep Medicine, Philadelphia, PA, ²Crescenz Veterans Affairs Medical Center, Philadelphia, PA, ³University of Manitoba -Sleep Disorders Centre, Winnipeg, MB, CANADA.

Introduction: Upper airway stimulation (UAS) is a surgical method of treating obstructive sleep apnea (OSA). UAS involves an implantable neuro-stimulator that stimulates the hypoglossal nerve to protrude the tongue during sleep. OSA fails to improve in 22% of patients who receive UAS as defined by a > 50% reduction in Apnea-Hypopnea Index (AHI) and an AHI <20 events/ hour. Light sleep may predict UAS failure in that it may limit the stimulus strength that can be applied. The odds ratio product (ORP) is a novel polysomnographic (PSG) metric of sleep depth. We hypothesized that ORP values prior to surgery will be higher (lighter sleep) in non-responders. Having markers that predict surgical success can help reduce unnecessary surgeries.

Methods: This is a retrospective cohort study of 126 patients (83 responders vs. 43 non-responders) who received UAS implantation for the treatment of OSA. PSG data was obtained from the Stimulation for Apnea Reduction (STAR) trial. Raw baseline PSG data were analyzed and ORP values calculated using Michele Sleep Scoring Software (Cerebra Medical, Winnipeg, CA). In addition, 13 PSG metrics that were considered possibly relevant to surgical outcome were calculated as an exploratory analysis. The measurements included: spindle density, spindle power, spindle frequency, alpha intrusion, Right/Left sleep

depth correlation coefficient, respiratory duty cycle, respiratory flow limitation, and arousal intensity. **Statistical Analysis:** Comparisons between responders and non-responders used parametric t-tests for continuous data and chi-squared or Fisher's exact tests for categorical data. Statistical significance was based on a Bonferroni-corrected p<0.00357.

Results: Differences in ORP values and other PSG metrics between responders and non-responders were not statistically significant. Of all PSG metrics only differences in spindle density approached statistical significance (Responders = 2.33 spindles/minute vs Non-Responders = 1.39 spindles/minute, p=0.00360).

Conclusion: The findings suggest that differences in sleep depth and several other sleep characteristics do not play a significant role in determining response to UAS therapy.

Support: This project was supported by a Sleep Research Society Career Development Award #023-JP-19

0692

DIFFERENCE IN IMPROVEMENT OF ESS SCORE AFTER CPAP USE IN PATIENTS FROM DIFFERENT WORKFORCES.

Mehta, T. R.¹ Gurung, P.² Digala, L.¹ Nene, Y.¹ Bollu, P. C.¹ ¹University of Missouri, Columbia, Columbia, MO, ²University of Missouri-Columbia, Columbia, MO.

Introduction: Obstructive sleep apnea (OSA) is characterized by recurrent occurrences of apnea and hypopnea throughout the night during sleep. Reported to prevail in 23.4% women and 49.7% men aged 40 years or older, OSA is considered to be the most preventable cause of excessive daytime sleepiness.

Methods: After obtaining approval from the institutional review board (IRB) for this retrospective study, a total of 825 patient records from a prospective registry of obstructive sleep apnea from our sleep lab affiliated with the University of Missouri Hospital were searched for variables including but not limited to age, race, gender, occupation, medications any sleep-related comorbidities, psychiatric comorbidities, cardio-vascular comorbidities, pre CPAP ESS score and post CPAP ESS score. The mean improvement score of ESS in both these populations was compared and possible causes for the difference in these groups were analyzed.

Results: Initial analysis from 22 patients belonging to the white-collar and 22 patients belonging to the blue-collar workforce with a mean age of 49.27 (\pm 14.28) years and a mean BMI of 37.60 (\pm 9.41) showed a mean improvement of -1.27 and 0.63 respectively with no significance statistically. Statistical analysis will be performed after gathering data from a larger sample size.

Conclusion: Although insignificant, the blue-collar workforce showed more improvement than the white-collar workforce in the initial analysis.

Support: No support, financial or otherwise was used for this study.

0693

EFFECTS OF SOLRIAMFETOL ON 24-HOUR BLOOD PRESSURE PATTERNS IN PARTICIPANTS WITH EXCESSIVE DAYTIME SLEEPINESS ASSOCIATED WITH OBSTRUCTIVE SLEEP APNEA

Strollo, P. J.¹ Malhotra, A.² Strohl, K.³ Pepin, J.⁴ Schweitzer, P.⁵ Lammers, G.⁶ Hedner, J.⁷ Baladi, M.⁸ Carter, L.⁸ Bujanover, S.⁸ Menno, D.⁹ Dauvilliers, Y.¹⁰ ¹University of Pittsburgh/Veterans Administration Pittsburgh Health System, Pittsburgh, PA, ²Division of Pulmonary, Critical Care and Sleep Medicine, University of California San Diego, La Jolla, CA, ³Case Western Reserve University, Cleveland, OH, ⁴Grenoble Alpes University Hospital, Grenoble, FRANCE, ⁵Sleep Medicine and Research Center St. Luke's Hospital, Chesterfield, MO, ⁶Sleep-Wake Centre SEIN, Zwolle, NETHERLANDS, ⁷Sahlgrenska University Hospital, Gothenburg, SWEDEN, ⁸Jazz Pharmaceuticals, Palo Alto, CA, ⁹Jazz Pharmaceuticals, Philadelphia, PA, ¹⁰Gui-de-Chauliac Hospital, Montpellier, FRANCE.

Introduction: Solriamfetol is a dopamine and norepinephrine reuptake inhibitor indicated to improve wakefulness in adult patients with excessive daytime sleepiness associated with obstructive sleep apnea (OSA; 37.5-150 mg/d) or narcolepsy (75-150 mg/d). Previous studies reported small mean increases in blood pressure (BP); however, the time course of these effects has not been evaluated. In addition, effects on BP dipping, which has been shown to be a risk factor for adverse cardiovascular outcomes have not been evaluated. These analyses evaluated the effects of solriamfetol treatment on BP using 24-hour ambulatory blood pressure monitoring (ABPM) and on the percentage of OSA patients with a nondipping BP profile.

Methods: Twenty-four-hour ABPM was conducted at baseline and week 8 in a 12-week randomized controlled trial in participants with OSA (n=474).

Results: At week 8, increases in BP were apparent in the 75 and 300 mg dose groups from ~6 AM until 8 PM. At baseline, 58% (placebo) and 55% (combined solriamfetol) of participants were non-dippers (defined as <10% decrease in mean arterial pressure [MAP] during sleep). There was no increase in the percentage of non-dippers at week 8 relative to baseline (placebo, 56%; combined solriamfetol, 53%). Results were similar when dipping was defined by changes in systolic BP and diastolic BP.

Conclusion: The effects of solriamfetol on BP at the highest approved dose of 150 mg/d are transient across the day. Solriamfetol was not observed to have an increase in non-dipping classification in participants with OSA at any dose studied.

Support: Jazz Pharmaceuticals

0694

POSITIVE AIRWAY PRESSURE USE IN THE CONTEXT OF TOTAL SLEEP TIME

Saconi, B.¹ Watach, A. J.² Sawyer, A. M.¹ ¹University of Pennsylvania, School of Nursing, Philadelphia, PA, ²University of Pennsyvlania Perelman School of Medicine, Center for Sleep & Circadian Neurobiology, Philadelphia, PA, ³University of Pennsylvania, School of Nursing, Philadelphia, PA.

Introduction: Positive airway pressure (PAP) adherence definitions are independent of total sleep time (TST). When PAP efficacy is examined by biophysiologic outcomes in adults with obstructive sleep apnea (OSA), sensitivity and specificity of the measurement of PAP adherence/non-adherence is important.

Methods: A post-hoc analysis of experimental data: 1) investigated agreement among concurrently-measured objective PAP use, objective and subjective TST; 2) assessed untreated TST among adherers and non-adherers defined by CMS PAP adherence criterion and separately by proportion of TST on PAP criteria (≥65%,

 \geq 75%, \geq 85%). Objective TST was measured with wrist actigraphy and concurrent sleep diary on the first five days of PAP use. Objective five day PAP use (hours/night) was abstracted from full record. Analyses included descriptive and exploratory correlations. **Results:** PAP-naïve adults (n=36; 76% male, 84% white) were middle-aged (51±10 years), with severe OSA (median AHI 31.1 events/hr), mean PAP use (371.5±85.4 min), mean objective and subjective TST (417.5±50.6 min, 439.6±58.1 min, respectively) from a single sleep center. Objective TST was correlated with concurrent objective PAP use (r=0.46) and with subjective TST (r=0.78). Eight percent (39mins) of objective TST was untreated among CMS-defined adherers, and 54.1% (216.8mins) of TST was untreated among CMS-defined non-adherers. When TST-ontreatment criteria were imposed ($\geq 65\%$, $\geq 75\%$, and $\geq 85\%$, respectively), the percentage of untreated TST decreased among adherers, 5.2% (23 min), 4.3% (19 min), 0% (0 min) and among non-adherers, 42.8% (168 min), 41.2% (162 min), and 28.7% (118 min). From CMS to ≥85% of TST-on-treatment criteria, categorization as an adherer decreased from 89% (n=34) to 58% (n=22); non-adherer categorization increased from 5% (n=2) to 37% (n=14).

Conclusion: In the context of understanding biophysiological responses to PAP treatment, more sensitive and specific criteria for adherence and non-adherence is necessary. PAP use based on untreated TST is an opportunity to address this gap.

Support: The parent clinical trial was supported by NIH/NINR (R00NR011173; Sawyer PI)

0695

SLEEPWELL24, A SMARTPHONE APPLICATION TO PROMOTE PAP THERAPY ADHERENCE: FEASIBILITY AND ACCEPTABILITY

Petrov, M. E.¹ Hasanaj, K.¹ Hoffmann, C. M.¹ Epstein, D. R.¹ Krahn, L.² Park, J. G.³ Hollingshead, K.¹ Yu, T.¹ St. Louis, E. K.³ Morgenthaler, T. I.³ Buman, M. P.¹

¹Arizona State University, Phoenix, AZ, ²Mayo Clinic, Scottsdale, AZ, ³Mayo Clinic, Rochester, MN.

Introduction: We aimed to test the feasibility and acceptability of *SleepWell24*, a multicomponent, smartphone-delivered intervention to increase positive airway pressure (PAP) adherence among newly diagnosed OSA patients.

Methods: Sleep Well24 targets PAP adherence along with other health behaviors through education, trouble-shooting, goalsetting, and near real-time biofeedback of PAP machine use, and sleep and physical activity levels (via Fitbit integration), and other chronic disease self-management components. Patients with a first-time diagnosis of OSA (AHI≥5) and prescribed PAP therapy were enrolled from the Centers for Sleep Medicine at Mayo Clinic in Rochester, MN and Phoenix, AZ. Patients were randomized to SleepWell24 or usual care (UC) and assessed for PAP use over 60 consecutive nights. UC patients received a Fitbit monitor to control for non-specific intervention effects related to the introduction of a new personal technology. Feasibility was assessed with recruitment and retention rates and acceptability was assessed post-intervention with the validated, 8-item Treatment Evaluation Questionnaire (TEQ; range:0-4). ANCOVA models, adjusting for age, sex, and AHI severity, compared intervention arms on acceptability ratings.

Results: OSA patients were consented and randomized (N=111). Before the intervention began 4 participants withdrew, 12 were lost to follow-up, and 5 could not start the trial due to durable medical equipment (DME) vendor barriers. Ninety OSA patients (n=41 *Sleep Well24*, n=49 UC; age $M\pm SD=57.2\pm 12.2$; 44.4% female, 61.1% AHI≥15) started the intervention, with 2 participants withdrawing, 1 becoming deceased (unrelated to treatment) and 7 with missing PAP data due to DME vendor barriers. There was no significant between-groups differences on post-treatment acceptability (*Sleep Well24* $M\pm SD=2.7\pm 1.1$ vs. UC $M\pm SD=3.1\pm 0.9$, *F*[1,73]=2.3, *p*=0.11), and 77% of *Sleep Well24* participants found the app to be moderately to totally acceptable.

Conclusion: Overall, *SleepWell24* was found to be feasible for delivery in two large clinical sleep medicine centers, and patients found the app to be acceptable. A number of challenges in trial delivery were encountered that have implications for scaled-up efficacy testing: (a) partnerships with DME vendors for near real-time PAP data integration; (b) alignment with clinical practice (i.e., referral, medical record integration); and (c) patient engagement. **Support:** National Institute of Nursing Research / National Institutes of Health: R21NR016046

0696

THE O₂VERLAP STUDY: HIGH CPAP USE LEVELS FOUND IN OVERLAP SYNDROME (OSA AND COPD) PATIENTS

Martinez, S.¹ Deering, S.² Sullivan, J.¹ Pasquale, C.¹ Shumard, T.³ Clark, B.¹ Amdur, A.³ Malanga, V.¹ Malanga, E.¹ Yawn, B.¹ Stepnowsky, C.^{2,4}

¹COPD Foundation, Miami, FL, ²VA San Diego Healthcare System, San Diego, CA, ³American Sleep Apnea Association, Washington, DC, ⁴University of California at San Diego, La Jolla, CA.

Introduction: CPAP therapy is prescribed to help manage disordered breathing during sleep time periods. Most users, especially those with non-severe obstructive sleep apnea (OSA), use it only for some portion of their sleep period. Patients with Overlap Syndrome have both OSA and chronic obstructive pulmonary disease (COPD). While there has been some research on CPAP use levels in this patient population, there has been little indication that they use CPAP any differently than those with OSA only.

Methods: The O₂VERLAP Study was a large comparative effectiveness trial enrolling people with COPD and OSA and using two different methods of providing information and support to current users of CPAP therapy. The study utilized an electronic national recruitment strategy and 332 participants were enrolled. CPAP data from the 12-week study period was analyzed. The Pittsburgh Sleep Quality Index was used to determine both estimated total sleep period (TSP) and total sleep time (TST). Because participants were all current users of CPAP, data from the total sample was combined and used. The percentage of TST and TSP that CPAP was used was calculated as CPAP use divided by either TST or TSP. Results: The mean TST was 6.8 hours, TSP was 8.1 hours, and CPAP use was 6.7 hours. CPAP was used during 98.5% of the TST and during 82.7% of the TSP. Over 35% of the sample used CPAP at a level that was equal to or greater than their total sleep period. Conclusion: Most OSA study populations use CPAP for some fraction of their night's sleep. This COPD/OSA study population used CPAP to a markedly high level, including over one-third of the sample (n=~100) who used CPAP more than their selfreported sleep period. Further research on the extent and reasons for non-sleep period (i.e., daytime) CPAP use in COPD patients is warranted.

Support: PPRND #1507-31666.

0697

EARLY SLEEP APNEA TERMINATION BY BONE-CONDUCTED SOUND STIMULATION REDUCES OXYGEN DESATURATION MAGNITUDE AND DURATION

Waeber, A.¹ Arnal, P.² Mignot, E.³ Heinzer, R.¹

¹Center for Investigation and Research in Sleep, Lausanne University, SWITZERLAND, ²Science Team, Dreem, New York, NY, ³Stanford University, Palo Alto, CA, ⁴Center for Investigation and Research in Sleep, Lausanne University, SWITZERLAND.

Introduction: Obstructive sleep apneas (OSA) usually end with an oxygen desaturation and/or an arousal. In most epidemiological studies, OSA-associated oxygen desaturations are stronger predictors of cardiovascular morbidity than OSA-associated arousals. The aim of this study was to determine if induction of a premature arousal by a bone-conducted sound stimulation shortly after the onset of an event can reduce the magnitude of OSA-associated oxygen desaturation.

Methods: Eight severe OSA patients (2 women, 45 [20-68]y.o.) underwent polysomnography at the Lausanne University Sleep Center (CIRS). Short acoustic stimulations were administered every second sleep apnea by remote control using a Dreem® head-band worn by the patients. Acoustic stimulations were administered by bone conduction. The magnitude(%) and the duration(s) of the oxygen desaturations following these prematurely-terminated apneas were compared with previous and following non-acoustic stimulated sleep apnea events.

Results: Analysis of 549 paired (stimulated-unstimulated) respiratory events in N1(14.2%), N2(69.9%), N3(4.2%), and REM sleep (9.6%) showed a 30.3% reduction in oxygen desaturation amplitude (mean difference \pm SD: -1.9 \pm 2.8%, p<0.0001), a 39.6% decrease in desaturation duration (-5.7 \pm 9.2 seconds, p<0.0001), and a 21.4% decrease in apnea duration (-3.4 \pm 7.2 seconds, p<0.0001) in stimulated apneas compared to the previous and subsequent non-stimulated apnea events. When analyzed individually, each patient showed a significant improvement following acoustic stimulation of events. Sound-associated discomfort was rated 1.14 \pm 1.53 on an 8 points scale (8=worst). Of the 68.6 \pm 38 administered sound stimuli per patient, only 6.8% of were perceived by the patients. A reduction in the desaturation amplitude occurred in each sleep stage but was milder in N3.

Conclusion: Bone-conducted sound stimuli applied trough a headband during the apneas decreased duration and magnitude of OSA-associated oxygen desaturation. These were well tolerated and often not perceived by the patient. This new treatment approach should be further investigated, with monitoring of its effect on cardiovascular parameters and daytime sleepiness.

Support: Customized Dreem headbands were provided by Dreem company

0698

A COMPARISON OF TWO VISUAL HYPOPNEA CLASSIFICATION METHODS

Dupuy-McCauley, K. Mudrakola, H. V. Morgenthaler, T. I. Mayo Clinic, Rochester, MN.

Introduction: The rules for classifying apneas as either obstructive or central using usual polysomnography (PSG) channels are well established, but classification of hypopneas is less straightforward without special sensors. Visual scoring methods have been proposed by the American Academy of Sleep Medicine (AASM) and

by Randerath, et al. These two scoring methods have never been compared. We evaluated these two scoring methods for clinical use. Methods: We selected 50 hypopnea segments from patient's PSGs with very clear obstructive physiology (average total AHI 48.6, central AI 0), assumed to be obstructive hypopneas, and from patient's PSGs with very clear central physiologies (average total AHI 34.3, obstructive AI 0.3), assumed to be central hypopneas. These 100 hypopnea-containing PSG segments (HCPS) were deidentified, placed in randomized order, and sent to two groups of 6 PSG scorers (2 RPSGTs, 2 sleep medicine fellows, 2 sleep medicine specialists). One group scored using the AASM criteria and the other used the Randerath algorithm. After a washout period, re-randomized HCPS were sent to be scored using the alternative method. We used Fleiss' kappa to determine inter-rater reliability—i.e., how consistently multiple scorers came to the same conclusion about a given hypopnea segment using each method, and accuracy—how often the scorers rated the HCPS in a manner consistent with the assumed physiology. We also recorded the time it took to score.

Results: Overall accuracy of both methods was 68%. Among 12 scorers, Fleiss' Kappa coefficient was 0.32 and 0.27 for the AASM and Randerath scoring methods, respectively. Average scoring time (24.3 minutes for AASM and 26.2 minutes for Randerath) was similar (p=0.79).

Conclusion: Inter-rater agreement was only fair using these methods, and accuracy was only 68%. More work is needed to discover a convenient, non-invasive way to reproducibly and accurately characterize hypopneas.

Support: This project does not have any funding support.

0699

EFFECT OF KIDNEY TRANSPLANTATION ON SLEEP APNEA SEVERITY: A PROSPECTIVE CONTROLLED POLYSOMNOGRAPHIC STUDY

Forni Ogna, V.¹ Ogna, A.² Haba-Rubio, J.³ Heinzer, R.⁴ ¹Nephology Department, Locarno Hospital, Locarno, SWITZERLAND, ²Pulmonary Department, Locarno Hospital, Locarno, SWAZILAND, ³Center for investigation in Lseep (CIRS), Lausanne University Hospital, Lausanne, SWITZERLAND, ⁴Lausanne University center for investigation and research in sleep, Lausanne, SWITZERLAND.

Introduction: Renal failure-associated fluid overload has been associated with a high prevalence of sleep apnea (SA) in patients with end-stage kidney disease (ESKD). Kidney transplantation has been shown to restore a normal renal function but its effect on SA remains unclear. In this prospective study, we hypothesized that improvement of kidney function and hydration status after kidney transplantation (Tx) may result in an improvement of SA severity. **Methods:** A total of 196 patients on kidney transplant waiting list were screened for SA using home nocturnal polysomnography (PSG) to measure the Apnea-Hypopnea Index (AHI) and underwent bioimpedance to assess body composition. Polysomnography and bioimpedance were repeated 6 months after kidney transplant. Patients still on the waiting list after 6 months underwent same investigations as a control group.

Results: Of 88 participants (44.9%) with SA (AHI \ge 15/h) at baseline, 42 patients were reassessed 6 months post-Tx and were compared to 27 control patients remaining on the waiting list after 6 months. There was a significant, although partial, post-Tx improvement in SA severity as measured by the AHI (from 44.2±24.3/h to 34.7±20.9/h, p=0.02), with a concomitant reduction

in body water (from 54.9% to 51.6%, p=0.003), suggesting a causal implication of fluid overload. A post-Tx increase in body fat mass (from 26% to 30%, p=0.003) may have blunted the beneficial impact of kidney Tx on SA. These parameters remained unchanged in the control group.

Conclusion: SA is a frequent condition in ESKD patients. Kidney transplantation is associated with a reduction of fluid overload but an increase in fat mass, yielding only a partial improvement in SA severity. These results suggest that SA should be systematically assessed before and after kidney Tx

Support:

Swiss Kidney Foundation, the Pulmonary League and the Organ Transplant Foundation of Lausanne

0700

PREDICTION OF SURGICAL OUTCOME USING RESPIRATORY PATTERN CLAFIFICATION

CHIBA, S.

Ota Memorial Sleep Center, Kawasaki, JAPAN.

Introduction: Introduction: A diagnosis of the surgical indication is important in Sleep surgery. In otolaryngology regions, it is usually considered that a site of occlusion diagnosis by DISE. However, for OSA patients with aging, heart failure and neurologic disease, the effect with the surgical treatment is still low. In these OSA patients, it is a reason that a functional factor is associated as well as an anatomical factor. Similarly, without clear complications including Aging etc, there are a lot of the OSA patients whom a functional factor is associated with. OSA is multi-factorial disease according to Wellman model, OSA is associated with the functional factor including loop gain and the ability of the upper airway to dilate associated with ventilatory drive, arousal threshold only other than the anatomical factor of the upper airway. By normal PSG, the respiratory event is classified as obstructive, mixed and central. However, there are a lot of events that a functional factor is associated with, even if it is a respiratory event to be diagnosed as an obstructive respiratory event. An effect of sleep surgery is expected only for an anatomical factor. Therefore, in the case of sleep surgery, we must diagnose a pure obstructive respiratory event precisely. Purpose of this study: To clarify a respiratory pattern to influence surgical success.

Methods: Methods: 26 surgical patients with Pharyngeal surgeries diagnosed as OSA by PSG + Pes measurements were enrolled. We distinguish respiratory event as Pure obstructive event or not, using Pes signal pattern and EEG arousal timing.

Results: Results: Rate of pure obstructive event of all respiratory event varied by an individual patient. Mean rate of pure obstructive event is 57.3%. We find significant difference of AHI improvement rate between two groups which Rate of pure obstructive event shows more than 55% or less than 55%.

Conclusion: Conclusion: Breathing pattern is variety and pure obstructive event is few than ever considered. Respiratory pattern influence surgical success.

Support: Non

0701

POSITIVE AIRWAY PRESSURE THERAPY TO TREAT SLEEP DISORDERED BREATHING IMPACTS NUMBER OF HOSPITALIZATIONS IN PATIENTS WITH HEART FAILURE

Patel, S. I.¹ Vasquez, M.² Huang, F.³ Combs, D.¹ Parthasarathy, S.¹

¹UAHS Center for Sleep and Circadian Sciences, University of Arizona, Tucson, AZ, ²Asthma & Airway Disease Research Center, University of Arizona, Tucson, AZ, ³Philips Respironics, Cambridge, MA.

Introduction: Some studies have shown a benefit while others have shown possible harm in patient outcomes when using positive airway pressure therapy (PAP) for treating sleep disordered breathing (SDB) in patients with heart failure (HF). The goal of this study was to evaluate the number of HF-related and all-cause related hospitalizations in patients with HF and SDB on various forms of PAP therapy versus those on no PAP therapy.

Methods: Administrative claims data from the Truven Health MarketScan Database from 1/1/2005- 10/31/2015 were analyzed. Those included were at least 21 years old, were continuously enrolled for 12 months before and 6 months after their index date (date of PAP prescription), had at least two distinct HF-related claims and were prescribed PAP therapy (n=1,324,414). To model the relationship between each device and hospitalization risk, and to account for the longitudinal and correlated nature of these binary outcome data, generalized estimating equations with binomial family, logit link, and unstructured correlation structure were used.

Results: There were a total of 12,538 patients on Bilevel-PAP, 2,700 patients on bilevel-PAP with backup rate, and 57,405 patients on CPAP, and 73,353 patients with HF and comorbid sleep apnea who were not on any treatment. The reduction in HF-related hospitalization for patients with HF and comorbid SDB treated with bilevel-PAP therapy (0.28; 95% CI 0.26, 0.31) was greater than that in patients receiving CPAP (OR 0.46 95% CI 0.43, 0.49), bilevel PAP with back-up rate (0.39; 95% CI 0.32, 0.49), or no PAP treatment (OR 0.54; 95%CI 0.50, 0.57)(P<0.01). Similar trend was observed for all-cause related hospitalizations. All results were adjusted for propensity score and other relevant confounders.

Conclusion: In claims-based analysis of patients with HF and comorbid SDB, bilevel PAP treatment was associated with reduced hospitalizations when compared to CPAP therapy or no PAP treatment.

Support: Phillips Respironics

0702

PHRENIC NERVE STIMULATION IMPROVES OXYGENATION AND QUALITY OF LIFE IN PATIENTS WITH CENTRAL SLEEP APNEA AND HISTORY OF CEREBROVASCULAR ACCIDENTS

Schwartz, A. R.¹ Khayat, R.² Sundar, K. M.³ Germany, R.⁴ McKane, S.⁴ Costanzo, M.⁵

¹Perelman School of Medicine at the University of Pennsylvania, Philadelphia, PA, ²University of California-Irvine, Orange, CA, ³University of Utah, Salt Lake City, UT, ⁴Respicardia, Minnetonka, MN, ⁵Advocate Heart Institute, Naperville, IL.

Introduction: Central sleep apnea (CSA) is a recognized complication of cerebrovascular accidents (CVA), although CSA treatment in this setting is of uncertain benefit. A recent randomized clinical trial showed that transvenous phrenic nerve stimulation (TPNS, **rem**edē system, Respicardia Inc.) treats CSA. The clinical impact of TPNS on CSA outcomes in the subgroup of patients with prior CVA was studied.

Methods: Six subjects with predominantly CSA and history of CVA >6 months prior to enrollment in the **rem**edē System Pivotal Trial were analyzed. Patients underwent attended full-night

polysomnography prior to TPNS implantation and 6, 12 and 18 months on TPNS therapy. Apnea-hypopnea index (AHI) and sleep metrics were evaluated. The Epworth Sleepiness Scale (ESS) and Patient Global Assessment (PGA) questionnaire were completed at 12 months. Treated patients and former controls (therapy was turned on after 6 months) with 18 months of TPNS therapy were pooled for analysis based on months of therapy.

Results: Apnea-hypopnea index decreased from a median of 47 events/hour [interquartile range: 23, 71] at baseline to 15 [4,24], 17 [6,48], and 12 [8,27] at 6, 12, and 18 months. Comparable improvements were also seen in oxygen desaturation index (4%) and arousal index. Central apnea index decreased from 30 [21,61] to \leq 3/ hour at each follow up. Compared to baseline, ESS decreased by 5 [-10,-3] and 4 [-8,-4] points at 6 and 12 months, while moderate or markedly improved overall health per the PGA was reported by 4/6 and 3/6 patients, respectively. No patient reported recurrent CVA or transient ischemic attack.

Conclusion: Transvenous phrenic nerve stimulation improved sleep, daytime somnolence and quality of life in patients with CSA and prior CVA. Transvenous phrenic nerve stimulation is a novel therapy that may be an option for treating patients with CSA and prior CVA.

Support: Respicardia and NIH R01 HL 144859

0703

TRANSVENOUS PHRENIC NERVE STIMULATION PROVIDES SAFE AND EFFECTIVE THERAPY FOR CHEYNE STOKES RESPIRATION

Schwartz, A. R.¹ Sundar, K.² McKane, S.³ Germany, R.³ Khayat, R.⁴

¹Perelman School of Medicine, Philadelphia, PA, ²University of Utah, Salt Lake City, UT, ³Respicardia, Minnetonka, MN, ⁴University of California-Irvine, Orange, CA.

Introduction: Cheyne-Stokes respiration (CSR), a specific type of central sleep apnea (CSA) is characterized by a waxing and waning pattern of breathing with absent air flow at the ventilatory nadir followed by oxyhemoglobin desaturation. CSR is most common in patients with heart failure (HF) and predicts morbidity/mortality. Therapeutic options remain limited, especially for patients with reduced left ventricular ejection fraction (LVEF).

Methods: Patients (n=151) with predominantly CSA were implanted with a transvenous phrenic nerve stimulation device (TPNS, **reme**dē system, Respicardia Inc.) and randomized to 6-months of active vs. deferred (control) therapy. Patients were divided into subgroups based on percentage of sleep in CSR (<20%, 20-50%, >50%) on their baseline polysomnogram. Response to TPNS, defined by \geq 50% reduction in apnea-hypopnea index, and Epworth Sleepiness Scale were assessed. TPNS efficacy and safety was analyzed in each subgroup.

Results: As percentage of CSR during sleep increased, more patients had a history of HF and lower LVEF. The proportion of TPNS responders was similar among CSR subgroups at 6 months (63% [17/27] CSR<20%, 52% [11/21] CSR 20-50%, 54% [7/13] CSR>50%); the corresponding control response rates were $\leq 16\%$ in each subgroup. Central apnea index decreased from median 33, 17 and 30 events/hour in these subgroups to ≤ 2 in TPNS-treated subgroups; control subgroups had median ≥ 17 events/hour at 6 months. Daytime sleepiness improved more in TPNS patients with <20% CSR (4/24 points vs. 2 in $\geq 20\%$ CSR subgroups). In the CSR>50% subgroup, cardiovascular death (pump failure) was observed in 2/25 control and 0/14 treatment subjects through

6 months. One cardiovascular death (sudden death) occurred in the TPNS 20-50% CSR subgroup.

Conclusion: TPNS effectively treats CSA regardless of CSR. Risk of cardiovascular death did not differ by CSR severity with TPNS, but may increase in CSR without treatment. TPNS therapy appears safe and efficacious for CSA with and without CSR. **Support:** Respicardia and NIH R01 HL 144859

0704

TREATMENT-EMERGENT CENTRAL SLEEP APNEA PREDICTS RESIDUAL RESPIRATORY INSTABILITY DURING CPAP USE AT 6 MONTHS

Thomas, R. J.

Beth Israel Deaconess Medical Center, Boston, MA.

Introduction: The prevalence, severity, significance, and predictors of residual sleep apnea during use of continuous positive airway pressure (CPAP) remain uncertain. High loop gain is associated with or induces periodic breathing and central sleep apnea (CSA). Treatment-emergent CSA (TE-CSA) is often considered a transient phenomenon of no long-term clinical significance. Standard polysomnographic features were assessed as risk factors for high residual apnea during compliant CPAP use.

Methods: Patients with sleep apnea (mean AHI 53.6, SD:33/hour of sleep) who underwent split night studies were prospectively entered in a database. They were all treated with positive airway pressure at the Beth Israel Deaconess Medical Center (Boston) and tracked by the EncoreAnywhere system. Machine detected AHI (AHIm) was extracted for a week average at month 6. The manual scored AHI(AHIs) was calculated from the last waveform graph during every month. Logistic regression assessed predictors of elevated automated (5 or greater) or manual (10 or greater) residual events//hour of use.

Results: A total of 69 CPAP compliant (average of at least 4 hours) subjects were analyzed. Age: 59.5 (range 17-81), gender: 47/69 male. 44/69 had an elevated manual AHI, while 20/69 had an elevated autodetected AHI. The only predictors of high residual apnea were TE-CSA (5 or more central apneas and hypopneas/hour of sleep): Odds Ratio 3.6 (CI: 1.07-12-3), p: 0.39. and the treatment component arousal index: Odds Ratio 1.06 (CI: 1.01-1.11), p: 0.018. Machine estimated AHI, which under-detected events by a factor of 3 or more, was not associated with any measure.

Conclusion: Residual apnea is common after 6 months of compliant CPAP use, and the only predictors identified were TE-CSA and treatment component arousal index.

Support: This study is supported by American Academy of Sleep Medicine Foundation, Category-I award to RJT

0705

HIGH INCIDENCE OF INADEQUATE HEALTH LITERACY IN ADULTS WITH OBSTRUCTIVE SLEEP APNEA IS NOT ASSOCIATED WITH POSITIVE AIRWAY PRESSURE USE

Watach, A. J.¹ Saconi, B.² Sawyer, A. M.²

¹University of Pennsylvania, Center for Sleep and Circadian Neurobiology, Philadelphia, PA, ²University of Pennsylvania, School of Nursing, Philadelphia, PA, ³University of Pennsylvania, School of Nursing, Philadelphia, PA.

Introduction: Inadequate health literacy (HL) is associated with 1.5 - 3 times increased risk for poor health outcomes, nonadherence and lack of skills needed to manage one's own health. Inadequate HL prevalence in adults with obstructive sleep apnea (OSA) may

be as high as 30%. The relationship between HL and positive airway pressure (PAP) adherence has been rarely examined.

Methods: A secondary analysis of prospective observational data was conducted to: 1) examine the prevalence of inadequate HL in adults with newly-diagnosed OSA and 2) determine if inadequate HL is associated with 1-wk and 1-mo PAP use. HL was measured using a 3-item Health Literacy Screening Questionnaire. Descriptive statistics, multiple linear regression, and logistic regression were used.

Results: Participants (n=67) were white (85%), males (52%) and females (48%), middle-aged (50±12 yrs), 64.2% had a middle to high school education and severe OSA (mean AHI 38.2±21 events/hr). Mean PAP use was 4.62±2.43 hrs/night at 1-wk and 4.33±2.27hrs/ night at 1-mo. Using a threshold of ≥4 hrs/night, 64% were adherent at 1-wk and 60% at 1-mo. Sixty two percent (n=42) screened positive for inadequate HL. A positive screen for inadequate HL (by individual screening items or by cumulative number of items screened positive) was not associated with PAP use (mean hr/night) at 1-wk or 1-mo (not retained in the final model). HL was also not associated with PAP non-adherence (<4hrs/night) or PAP failure (<2hrs/night) by logistic regression.

Conclusion: Inadequate HL may be prevalent in adults with OSA. OSA and PAP patient education content and design should align with HL abilities and skills. Disease and treatment education are influential on PAP adherence. Future research should consider adequacy of three generalized items to assess HL and disease-specific HL as more robust measures are available. Larger, heterogeneous sample sizes are needed to precisely estimate the relationship between HL and PAP adherence.

Support: Lead author receives support from NIH/NHLBI Award T32 HL07953.

0706

COMPARING THE DOSE-RESPONSE CURVES OF UPPER AIRWAY STIMULATION AND CPAP USAGE ON CHANGES IN EPWORTH SLEEPINESS SCALE

Kazaglis, L.¹ Hsia, J.¹ Green, K.² Iber, C.¹

¹M Health Fairview, Minneapolis, MN, ²University of Colorado, Denver, CO.

Introduction: Upper Airway Stimulation (UAS) and Continuous Positive Airway Pressure (CPAP) are trackable therapies for obstructive sleep apnea. We used recent big-data cohorts to compare changes in sleepiness versus usage.

Methods: ADHERE is an international registry of real-world UAS outcomes from 2016 to date. General UAS criteria are CPAP intolerance, AHI 15-65 (<25% central+mixed), and suggested BMI≤35. Baseline ESS is collected from the medical record, and follow-up ESS and usage is collected 2-4 months after therapy activation. M Health Fairview maintains a database of cross-linked CPAP and EHR data. All new adult sleep patients from 2015 onward were included paralleling ADHERE: BMI≤35, AHI 15-65, and daily CPAP-EHR data starting at least 60 days prior to 2nd ESS measurement. Baseline ESS was collected at consult, and follow-up ESS was collected approximately 6 months later. Device-reported usage hours were compared with the changes in ESS from baseline. **Results:** UAS (n=690) and CPAP (n=514) groups were similar: age 59.7±10.8 versus 59.7±13.6, 78% versus 75% male, and AHI 35.3±14.4 versus 33.8±14.0. UAS group was slightly less obese, BMI 29.3 \pm 3.9 versus 30.0 \pm 3.4 (p=0.001), with higher baseline ESS, 11.4±5.6 versus 8.6±5.3 (p<0.001). UAS usage was higher at 6.4±2.0 hours/night versus 5.2±2.0 hours/night with CPAP

(p<0.001). UAS group average ESS decreased 2.5 points for patients with 0-4 hours of use (n=81), decreasing to 3.8 points with at 4 or more hours of use (n=609). CPAP group average ESS decreased 2.5 points for patients with 0-4 hours of use (n=125), decreasing to 3.3 points with at 4 or more hours of use (n=389).

Conclusion: Compared to prior works and the UAS cohort, this CPAP cohort was more likely to have normal ESS at baseline. UAS and CPAP both demonstrate a dose-response curve associating increasing hourly usage with larger ESS reductions.

Support: Kent Lee of Inspire Medical Systems provided background information and access to a de-identified ADHERE data set for analysis.

0707

"WHAT WE'VE GOT HERE IS FAILURE TO COMMUNICATE": APNEA PATIENTS AND THEIR DME SUPPLIERS

Thapa, S. Agrawal, S. Kryger, M. Yale University, New Haven, CT.

Introduction: Successful treatment of obstructive sleep apnea requires adherence to positive airway pressure (PAP) therapy. A key factor is the relationship between the DME provider and the patient so that treatment can be initiated and continued in a timely manner. Our quality improvement project aims to empower and enable patients towards active participation in their sleep apnea care. Our goal is to ultimately increase patients' knowledge of their Durable Medical Equipment (DME) supplies company, and thus improve their treatment. The first step was to determine patients' familiarity with their DME.

Methods: Forty-one patients with sleep apnea on PAP therapy volunteered to be questioned about their DME company during clinic visits at the Yale North Haven Sleep Center, Connecticut, starting November 2019. Patients were asked if they knew the name or the contact of their DME; whether they received adequate training on PAP therapy initiation; if they were receiving timely and correct PAP therapy supplies. They were asked to rate their satisfaction with the DME on a scale of 1 to 5; one being very dissatisfied and five being very satisfied.

Results: Only 12 out of 41 patients (29.3 percent) knew the names of their DME companies. The average satisfaction rating was 3 (neutral); 44% of patients were dissatisfied, or very dissatisfied with the performance of their DME. Detailed comments were mostly related to poor contact and communication with the DME.

Conclusion: Most apnea patients had difficulty identifying and contacting their DME. As the next step of this quality improvement project we plan to intervene to ensure that the patients have the name and contact information of their DME available and attached to their PAP machine equipment. We plan to repeat this questionnaire after this intervention to study the impact of this quality improvement project.

Support: None

0708

A STUDY TO DETERMINE EFFICACY OF A DEVICE IN ANTICIPATING OBSTRUCTIVE SLEEP APNEA EVENTS

Rechul, D.¹ Rechul, K.²

¹Yale University, New Haven, CT, ²DWI, Colorado Springs, CO.

Introduction: Current treatment options for obstructive sleep apnea pose multiple challenges ranging from issues with therapy adherence (i.e. PAP) to partial effectiveness (i.e. MAD) or invasiveness

(i.e. implantable nerve stimulation devices). SleepMethods, Inc. designed a device that proposed to solve these issues. It consisted of two integrated mechanisms; one to anticipate the development of an obstructive event and the other to deliver therapeutic intervention to abort the process of airway collapse before it ensued. A clinical trial was conducted to test the efficacy of the system designed to anticipate obstructive sleep apnea events.

Methods: Ten adults (7M;3F) aged 18-80y/o (avg. 54.7y/o) with a known AHI \geq 15/hr (avg. AHI = 42.6/hr) underwent 1 overnight PSG recording while wearing the device. Patients were required to forego their usual CPAP therapy on the night of study in efforts to expose the device to an adequate number of total obstructive events (defined as apneas and hypopneas; RERAs and snores were excluded). Standard PSG analysis was performed. Scoring rules were applied to determine whether signals were true/false positives and/or true/false negatives based on pre-clinical data showing anticipation accuracy for up to 45 seconds prior to an obstructive airway event.

Results: Preliminary results suggest the device functions with 96.9% sensitivity and about 67.1% PPV. Data analysis is ongoing to determine specificity, statistical significance, etc.

Conclusion: The device has shown promise in pre-clinical and clinical trials to accurately and consistently anticipate airway collapse; a possible breakthrough in designing more targeted therapies aimed at aborting obstructive sleep apnea events before they ensue to a clinically significant degree.

Support: N/A

0709

USING THE SLEEP ADJUSTED RESIDUAL APNEA HYPOPNEA INDEX IN THE REAL-WORLD MONITORING OF OBSTRUCTIVE SLEEP APNEA

Viteri, E.¹ McGhee, V.¹ Tackett, J. V.² Freire, A. X.^{1,2} ¹University of Tennessee Health Sciences Center, Memphis, TN,

²Veteran's Affairs Hospital, Memphis, TN.

Introduction: Treatment efficacy of obstructive sleep apnea (OSA) depends on controlling respiratory events for the majority of sleep time. Apnea-hypopnea index (AHI) and adherence are frequently used to determine efficacy of continuous positive airway (CPAP) therapy, but fail to capture the effect of residual events during untreated sleep-time. The Sleep Adjusted Residual AHI (SARAHI) consolidates treated and untreated AHI and CPAP use into a single number: SARAHI = [(Untreated AHI × Hours Untreated) + (Treated AHI × Hours Treated)] / (Total Sleep Hours). We attempted to determine the clinical applicability of this index as a determinant of OSA control and its relation with sleepiness improvement.

Methods: As part of a quality assessment project, a convenience sample was haphazardly collected from a database of patients initiated on CPAP in a Veteran's Affairs Hospital. Patients initiating treatment after OSA diagnosis by polysomnogram or portable sleep study were included. Information from a CPAP-download within a year of diagnosis and Epworth Sleepiness Scale (ESS) at diagnosis and follow-up were collected. SARAHI was calculated using two different measures of "total sleep hours": 8 hours (SARAHI-8hrs) or recorded sleep time during sleep study (SARAHI-PSG).

Results: Thirteen patients (12 male) with a mean age of 53.3 years were included. At diagnosis, mean AHI was 26.0 events/hour and ESS was 14.6. At follow-up, CPAP mean adherence was 338 min and average use was 61.8% of days; mean residual AHI was 4.4 events/hour and mean ESS 13.7. SARAHI-8hrs was 16.0 events/

hour and SARAHI-PSG was 13.8 events/hr. Simple linear regression did not show a significant correlation between ESS improvement and either of these indexes or with improvement in AHI.

Conclusion: SARAHI showed no correlation with ESS in this small sample. We recommend further research as SARAHI is simple to use and provides more information than currently used parameters.

Support: None

0710

HYPOGLOSSAL NEUROSTIMULATION IN REM- VS. NREM-PREDOMINANT OBSTRUCTIVE SLEEP APNEA

Cabrera, C. I.¹ Szelestey, B.¹ Strohl, K.² Schell, A.¹

¹UH Cleveland Medical Center Department of Otolaryngology-Head and Neck Surgery, Cleveland, OH, ²UH Cleveland Medical Center Department of Medicine, Division of Pulmonary, Critical Care, and Sleep Medicine, Cleveland, OH.

Introduction: Obstructive sleep apnea (OSA) is a chronic condition that requires appropriate treatment strategies to optimize outcomes while minimizing risk. In addition to anatomy, physiologic factors such as arousal threshold and loop gain (i.e. "endotypes") play a known role in the disease process. Because loop gain tends to be higher and arousal threshold lower in NREM sleep, we hypothesize that patients with NREM-predominance may achieve less success with anatomical therapy such as upper airway stimulation (UAS). Our study aims to evaluate baseline characteristics and objective results related to NREM-predominance in patients treated with UAS.

Methods: Using data from the STAR trial, we identified patients (n=103) with at least 20 minutes of REM on baseline testing and complete demographic and disease data at baseline and month 18. Baseline NREM-predominant disease (percent NREM events > 50) was defined as a binary variable. We created two cohorts: 1) patients with REM-predominant disease and 2) those with NREM-predominant disease. ODI and AHI were evaluated at month 18.

Results: Overall 62% (n=64) of patients had NREM-predominant disease at baseline. Other baseline characteristics were similar between both groups. In univariate analysis, age was significantly associated with lower AHI in the NREM-predominant population (p<0.05) but not in the REM-predominant group (p>0.05). Results were similar for ODI. For both groups, increasing age was correlated negatively with increasing AHI; this correlation was stronger in the NREM-predominant group

Conclusion: A majority of patients in the STAR trial had NREMpredominant OSA at baseline. There appears to be an interaction between NREM-predominance and age as predictors of UAS outcomes.

Support:

0711

SELECT SYMPTOMS FROM THE EPWORTH SLEEPINESS SCALE QUESTIONNAIRE AND RESPONSE TO THERAPY OF CENTRAL SLEEP APNEA WITH PHRENIC NERVE STIMULATION

Javaheri, S.¹ McKane, S.² Meyer, T. E.² Germany, R.² ¹Bethesda North Hospital, Cincinnati, OH, ²Respicardia, Minnetonka, MN.

Introduction: Some subjects with central sleep apnea (CSA) complain of subjective excessive daytime sleepiness (EDS), as assessed by the Epworth Sleepiness Score (ESS). However, there is

considerable variability in the level of chances of dozing for each of the 8 ESS questions, as each reflects a different situation. The aim of this analysis was to examine individual situations of the ESS and determine if transvenous phrenic nerve stimulation (TPNS) resulted in improvements of individual ESS situations which were scored moderate to high (2 and 3) at baseline. Patient Global Assessment (PGA) was also assessed.

Methods: All 151 subjects enrolled in the randomized (Treatment vs Control) **rem**edē System pivotal trial were included in the analysis. All subjects were implanted, but activation in Control arm was delayed 6 months.

Results: Greater than or equal to 50% of patients scored moderatehigh on several individual ESS situations: chance of dozing while sitting and reading (57%), watching television (62%), while lying down to rest in the afternoon when circumstances permit (76%). In the active arm, 68%, 44% and 29% of patients with moderate-high at baseline, respectively, for sitting and reading, watching television, and lying down to rest in the afternoon shifted to less than moderate at 6 months. Respective shifts for the control arm were 29%, 23% and 13%. Seventy-two percent of treated subjects with baseline ESS>10 shifted to \leq 10 at 6 months compared to 26% of control patients. Additionally, 72% of treated compared to 7% of control subjects with baseline ESS>10 reported markedly or moderately improved QoL.

Conclusion: Results of this randomized controlled trial shows that compared to the control arm, TPNS leads to improvements in various situations of the ESS as well as QoL. The most improved situations were less chance of dozing while reading and watching television.

Support: Respicardia

0712

CPAP COMPLIANCE: ROLES OF DEPRESSIVE SYMPTOMS, POLYSOMNOGRAPHIC AND SELF-REPORT SLEEP MEASURES

Dubrovsky, B. Inamac, A. Gikashvili, L. Weingarten, J. Cunningham, J.

New York-Presbyterian Brooklyn Methodist Hospital, Department of Medicine, Division of Pulmonary and Critical Care, Center for Sleep Disorders, Brooklyn, NY.

Introduction: OSA poses major health risks; however, CPAP compliance is often suboptimal. Comorbid insomnia and depression contributed to poor CPAP compliance in different studies. Presently, PSG variables, self-report measures of insomnia, sleep quality and depression were tested simultaneously as predictors of compliance in new CPAP users who at 180 days of therapy had any non-zero use of CPAP.

Methods: PSG-diagnosed 47 patients (18-79 y.o., 24 women) were titrated in the lab and initiated on CPAP. Compliance was assessed during days 1-30 and 151-180. For each period, percentage of days with >4hrs of CPAP use (%>4h-days30, %>4h-days180) and the average hours-per-day use (Tav30, Tav180) were measured. After regressing each compliance variable on age, sex and BMI, one at a time were tested: PSG variables from the diagnostic and titration studies, subjective reactions to CPAP after titration (sleep better vs. same/worse than usual; will vs. won't use CPAP at home), pre-treatment ISI, PSQI, ESS, and Center for Epidemiologic Studies Depression Scale-Revised (CESDR).

Results: AHI ranged 6/hr-101/hr (M=24.2±19.5); %>4h-days30, 0-100% (M=68%±32%); Tav30, 0.0-9.8 hours (M=5.2±2.5); %>4h-days180, 0-100% (M=64%±34%); Tav180, 0.2-10.5 hours

(M=4.7 \pm 2.8). PSG variables from both diagnostic and titration studies, reactions to CPAP, ESS and CESDR were not significant predictors of compliance (all p>0.1). ISI was marginally inversely related to %>4h-days30 (p=0.087) and Tav30 (p=0.075). Higher pre-treatment PSQI was related to lower %>4h-days30 (p=0.003, R²=19%) and Tav30 (p=0.011, R²=15%). Entered alone, PSQI was related to %>4h-days180 (p=0.042) and Tav180 (p=0.043); however, when the 1-30-day compliance was accounted for, PSQI no longer related to the 151-180-day compliance. The 1-30-day compliance strongly predicted respective 151-180-day measures (%>4h-days, p<0.001, R²=55%; Tav, p<0.001, R²=68%).

Conclusion: In this limited sample of naive CPAP users, higher pre-treatment sleep disturbance reported on PSQI was a useful predictor of lower 1-30-day CPAP compliance. Neither depressive symptoms nor PSG variables from diagnostic and titration studies predicted compliance. As the initial 30-day compliance is the best predictor of later CPAP use, early interventions are crucial. **Support:** none

0713

ASSOCIATION BETWEEN OBESITY INDICES AND OBSTRUCTIVE SLEEP APNEA IS MODIFIED BY AGE IN A SEX-SPECIFIC MANNER

Liu, Y. Meng, L. Guan, J. Yi, H. Yin, S.

Shanghai Jiao Tong University Affiliated Sixth People's Hospital, Shanghai, CHINA.

Introduction: The beneficial effects of weight loss on obstructive sleep apnea (OSA) are highly variable. Whether the variability is associated with the effects of age and sex remains unclear. This study examined this issue with large cross-sectional data.

Methods: A total of 4600 adult males and 1156 females with suspected OSA were included in the study. Anthropometric measurements, polysomnographic variables, biochemical indicators, and medical history were collected for each subject. Multivariable linear regression with interaction terms was used to estimate the modification effect of age on the associations between OSA severity (assessed by apnea-hypopnea index, AHI) with obesity indices (body mass index, BMI; neck circumference, NC; waist circumference, WC) in a sex-specific manner, and vice versa.

Results: BMI, NC, and WC were all positively correlated with AHI after adjusting for potential confounders in all populations. In males, these associations were much stronger and more significant in younger than older individuals (*P* for interaction < 0.001). For example, a 10% increase in BMI was independently associated with a 31.6% increase in AHI for males < 40 years old, whereas the corresponding increases were 20.8% and 16.7% for males 40-60 and >60 years old, respectively. By contrast, no modification effect of age was observed in females (*P* for interaction > 0.05). A 10% increase in BMI was associated with 25.6%, 26.8%, and 23.8% increases in AHI for females < 40, 40-60, and >60 years old, respectively.

Conclusion: Age modifies the associations between obesity indices and OSA severity in a sex-specific manner, and vice versa. These findings may broaden the understanding of age- and sex-related heterogeneities in the pathogenic role of obesity in OSA, and may be beneficial for individualized risk evaluation and treatment management for patients with OSA.

Support: ThisstudywasfundedbyShanghaiMunicipalCommission of Science and Technology (grant number.18DZ2260200); the National Key R&D Program of China (grant number: 2017YFC0112500); Multi-Center Clinical Research Project from the School of Medicine, Shanghai Jiao Tong University (grant number: DLY201502); and the Shanghai Shen-Kang Hospital Management Center Project (grant number: SHDC12015101 and 16CR3103B).

0714

EFFECT OF ONE-WEEK VELOPHARYNGEAL-TASK TRAINING ON GENIOGLOSSUS CORTICOMOTOR EXCITABILITY AND SLEEP APNEA SEVERITY

Li, W.¹ Wei, Z.¹ Wang, W.¹ Frédéric, S.²

¹China medical university, Shenyang, Liaoning, CHINA, ²Professeur titulaire département de Médecine Université Laval Pneumologue Institut Universitaire de Cardiologie et de Pneumologie de Québec, Quebec, QC, CANADA.

Introduction: The effectiveness of the contraction of the UA dilator muscles plays a crucial role in the maintenance of UA patency. This study aimed to assess the effects of one-week velopharyngeal-task training (VTT) on sleep apnea severity and its genioglossus (GG) corticomotor excitability.

Methods: Ten patients with sleep apnea underwent 1 h VTT on seven consecutive days. During the VTT protocol, subjects were asked to develop repetitive intra-oral positive pressure using cheekbulging maneuvers while wearing a mouth piece to keep the jaw opened and maintaining an exclusive nasal breathing. They were encouraged to generate approximately 4% of Max pressure by maintaining the pressure inside the corresponding pre-set pressure target window for 2 sec every 10 sec. PSG recording and GG transcranial magnetic stimulation (TMS) response was obtained before and after the one-week VTT.

Results: One-week VTT was associated with a global AHI decrease by 33.8% (pre-VTT: 34.5 ± 31.9 n/h; post-VTT: 25.5 ± 26.7 n/h; p < 0.05) and progressed from moderate/severe to mild/moderate in 40% of patients. Although the bulging pressure remained unchanged (pre-VTT: 17.1 ± 5.6 kPa, post-VTT: 19.4 ± 5.4 kPa, p > 0.05), the amplitude of GG motor evoked potential in response to TMS significantly increased after the one-week VTT (pre-VTT: 639.4 ± 380.9 mV; post-VTT: 1128.5 ± 623.9 mV; p < 0.05).

Conclusion: One-week VTT is sufficient to confer clinical benefits on patients with sleep apnea. VTT protocol is not oriented toward strength gain, but rather toward an enhancement in the upper airway muscle cortical excitability and improvement in the coordination of their contraction. The authors consider these results to be potentially clinically relevant and worthy of further investigation in a large randomized trial.

Support: National Natural Science Foundation of China, Grant/ Award Number: 81670085.

0715

LONG-TERM CPAP ADHERENCE IN A PUBLIC SLEEP CLINIC

Rezayat, T.¹ Vassar, S.² Hakopian, S.² Wallace, J.³

¹Sleep Medicine Fellowship Program, David Geffen School of Medicine at UCLA, Los Angeles, CA, ²Department of Medicine, Olive View-UCLA Medical Center, Sylmar, CA, ³Division of Sleep Medicine, Olive View-UCLA Medical Center, Sylmar, CA.

Introduction: CPAP adherence may drop substantially over a long time frame. Since minorities including Hispanics and low socioeconomic groups have lower short-term acceptance and adherence, long-term adherence may also be reduced. We adapted a brief motivational enhancement education program (BMEEP)

(Lai, CHEST 2014) in a Los Angeles County safety-net sleep clinic and found improved CPAP adherence at 3 months. We now report longitudinal long-term adherence over ≥ 2 years. Our hypothesis: Many patients would meet CMS adherence criteria over ≥ 2 years.

Methods: During 3/1/2016 - 4/1/17, 118 patients completed BMEEP during CPAP initiation and were scheduled for a reinforcement session and routine clinic visits at 1 and 3 months and every 6 months thereafter. Electronic adherence and efficacy data were accessed each visit. Multivariate regression analyses explored association of adherence variables with demographic, clinical and workflow features.

Results: Baseline characteristics (mean (SD) or percent): Age 57.2 (17.8); Women 44.9%; Hispanic 69.5%, Non-Hispanic-White 22.9%, Other 5%, Black 3%; BMI 37; Epworth score 10.4 (6.05). Home sleep testing (69 patients) respiratory event indices were $\geq=15/hr$ in 67% and $\geq=30/hr$ in 57%. Polysomnography (81 patients) apnea hypopnea indices were (AHI) $\geq=15$ in 78% and $\geq=30/hr$ in 43%. By 7/1/2019, 23 (19%) patients were lost with unknown CPAP use status, while 76 (70.5%) of remaining patients continued to use CPAP. At 2 years, CPAP adherence parameters included: Average nightly use, 75% (27.7); Average hours/night, 4.74 (2.5); Average nightly use $\geq=4$ hours, 65.2% (31.6); $\geq=70\%$ nightly use $\geq=4$ hours, 35 (51.5%). Average residual AHI was 2.05 (1.69). Adherence parameters in individual patients remained similar throughout 2 years. Long-term adherence was not associated with the demographic, clinical or workflow variables tested.

Conclusion: Programs that educate, motivate and provide regular follow-up for predominantly Hispanic low income populations can achieve acceptable long-term CPAP adherence rates. **Support:** None

0716

THE CLINICAL CHARACTERISTICS AND THERAPEUTIC EFFICACY OF CATATHRENIA

Wen, Y. Xu, L. Han, F.

Peking university People's hospital, Beijing, CHINA.

Introduction: Catathrenia/nocturnal groaning, is a rare sleep disorder characterized by repeated groaning in a protracted expiration preceded by a deep inspiration. The classification was moved from ICSD-2 as parasomnia to sleep-disordered breathing(SDB) in ICSD-3. The pathogenesis of catathrenia is unknown.

Methods: To investigate catathrenia's response to continuous positive airway pressure (CPAP) treatment, and its relationship with SDB. We analyzed patients diagnosed at the Sleep Center of Peking University People's Hospital from 2009 to 2016. All patients were confirmed with nocturnal groaning by polysomnography(PSG), and recording of the sounds. The patients were recommended for treatment of CPAP.

Results: A total of 49 patients were recruited into this study, including 31 female and 18 male, age ranging from 16 to 64 years. The average onset age was 19.81 ± 8.65 years old, the average BMI was 21.76 ± 2.62 kg/m², the average groaning index (GI) was 11.58 ± 13.32 times per hour, and the average apnea hypopnea index (AHI) was 3.59 ± 8.83 times per hour. The GI in REM and NREM sleep were 39.31 ± 44.39 and 6.74 ± 8.5 times per hour respectively (p <0.01). The GI of 26 patients who received CPAP treatment decreased from 10.57 ± 12.01 to 5.65 ± 7.10 times per hour (p <0.001).

Conclusion: Catathrenia occurs mainly in REM sleep, and treatment with CPAP is effective, although cannot be completely eliminated. Suggest that catathrenia might be a sleep breathing disorder **Support:** There is no support of this study

0717

PEER-INTERVENTION CAN REDUCE HEALTH DISPARITIES IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Patel, S. I.¹ Combs, D.¹ Provencio-Dean, N.¹ Mashaqi, S.¹ Bhattacharjee, S.¹ Quan, S. F.² Morton, C. J.¹ Wendel, C.¹ Parthasarathy, S.³

¹UAHS Center for Sleep & Circadian Sciences; Division of Pulmonary, Allergy, Critical Care & Sleep Medicine; University of Arizona, Tucson, AZ, ²Harvard Medical School and UAHS Center for Sleep & Circadian Sciences; Division of Pulmonary, Allergy, Critical Care & Sleep Medicine; University of Arizona, Tucson, AZ, ³University of Arizona Health Sciences Center for Sleep and Circadian Sciences, Tucson, AZ.

Introduction: In patients with obstructive sleep apnea (OSA), adherence to continuous positive airway pressure (CPAP) therapy is a major problem. Moreover, up to 20% of patients with suspected OSA who are referred to sleep study testing do not adhere to such diagnostic work-up. Although, peer-driven intervention with an interactive voice response system (PDI-IVR) can improve CPAP adherence, whether such an intervention can improve adherence to sleep study testing is unknown. Also, there remain health disparities with greater levels of CPAP nonadherence disproportionately affecting individuals of lower socioeconomic status. We aimed to determine whether PDI-IVR can improve adherence to sleep study testing and CPAP adherence in a lower income population.

Methods: We performed a prospective, randomized, parallel group, controlled trial wherein patients with suspected OSA were randomly assigned to receive PDI-IVR or provided with educational information regarding OSA and CPAP therapy (attention-control group) while both groups received usual care. The PDI-IVR interactions aimed at promoting adherence to sleep study testing and in patients diagnosed with OSA the peer-intervention was focused on improving CPAP adherence. In the PDI-IVR group, trained peers (peer-buddies) with OSA were paired with randomized patients over a 6-month period combined with an ability to meet in-person, email, text message, or phone an inter-disciplinary team of providers.

Results: In this pilot study, there were 63 patients (48.4 ± 12.5 years; 30 men) who were randomized to intervention (n=31) and attention-control (n=32) arms. There were 36 peer-buddies who mentored the patients in the intervention group. Intention to treat analysis revealed that failure to undergo sleep study testing was 15.6% of patients in the attention-control arm and 9.7% in the PDI-IVR arm (P=0.7). Per protocol analysis revealed that failure to undergo sleep study testing was 18.4% of patients in the attention-control arm (P=0.13). At 6 months, CPAP adherence was greater in PDI-IVR arm (290 ± 45 min [SE]) than attention-control arm (181 ± 43 min; P=0.01).

Conclusion: In a lower income population, PDI-IVR improved CPAP adherence with a tendency for better adherence to sleep-study testing. Peer-intervention can reduce sleep health disparities. **Support:** HL138377

0718

HYPOXEMIA AND PULMONARY HYPERTENSION IN PATIENTS WITH CONCOMITANT RESTRICTIVE VENTILATORY DEFECT AND SLEEP APNEA: THE OVERLAP SYNDROME

Xie, J. Fan, Z. Wang, J. Li, F.

Department of Respiratory and Critical Medicine of Beijing An Zhen Hospital, Capital Medical University, Beijing, CHINA.

Introduction: Patients with severe restrictive ventilatory defect (RVD) have hypoxemia and a high risk of pulmonary hypertension (PHTN). Sleep apnea (SA) aggravates the severity of nocturnal desaturation significantly. The aim of this study was to investigate the severity of hypoxemia and prevalence of PHTN in patient with the overlap syndrome (OS) of RVD and SA.

Methods: Patients referred for both sleep test and spirometry for suspected SA and RVD or obstructive ventilatory defect (OVD) were recruited prospectively from January-December, 2018. SA was determined by an apnea-hypopnea index \geq 5/h; mean nocturnal oxygen saturation (meanSaO₂), minimum oxygen saturation (minSaO₂), saturation lower than 90% (T90) were calculated automatically. RVD was diagnosed in the presence of forced expiratory volume in the first second/forced vital capacity (FVC) >0.7 and FVC<80% predicted value. PHTN was defined by systolic pulmonary arterial pressure (SPAP) \geq 50mmHg, documented by noninvasive transthoracic echocardiography. Patients with PHTN secondary to extrapulmonary factors were excluded.

Results: Of 65 patients who completed the investigation, 16 (24.6%) subjects were diagnosed with isolated SA (without RVD or OVD), and 28 (43.1%) subjects were verified to have RVD, in which 22 (78.6%) were diagnosed with OS and 6 (21.4%) presented as isolated RVD. Patients with OS vs. those with isolated RVD had lower minSaO₂ (78.3% vs. 88.7%, p=0.003) and meanSaO₂ (91.5% vs. 95.8%, p=0.007) but higher T90 (37.2% vs. 0.3%, p=0.009). Patients with OS vs. those with isolated RVD or with isolated SA had higher SPAP (62.6 mmHg vs. 45.3 mmHg or 35.9 mmHg, p=0.334 or p=0.016 respectively). Higher proportion of patients with OS were diagnosed with PHTN than those with isolated RVD or isolated SA (8 [36.4%] vs. 1 [16%] or 1 [6.25%], p=0.360 or p=0.031, respectively). T90 was the only polysomnographic data associated with the prevalence of PHTN after adjusting for age and sex (OR 4.90, 95% CI 1.23-25.56, p=0.023).

Conclusion: Patients with the OS of RVD and SA had high odds of PHTN, which is probably associated with severe hypoxemia. Further investigation is needed to discern whether therapeutic strategies toward OS might eliminate PHTN in this cohort. **Support:**

0719

THE ASSOCIATION OF SLEEP APNEA AND CARDIORESPIRATORY FITNESS WITH LONG-TERM MAJOR CARDIOVASCULAR EVENTS

Barillas-Lara, M. Medina-Inojosa, J. Kolla, B. Smith, J. R. Bonikowske, A. R. Allison, T. G. Olson, T. Lopez-Jimenez, F. Somers, V. K. Caples, S. M. Mansukhani, M. P. Mayo Clinic, Rochester, MN.

Introduction: Sleep disordered breathing (SDB) is associated with adverse cardiovascular outcomes and decreased cardiorespiratory fitness (CRF). The risk of long-term major adverse cardiovascular events (MACE) when SDB and decreased CRF co-occur has not been determined.

Methods: We included consecutive patients that underwent a symptom-limited cardiopulmonary exercise test followed by first-time diagnostic polysomnography within 6 months. Patients were stratified based on the presence of moderate-severe SDB (apnea/hypopnea index \geq 15/hour) and decreased CRF defined as <70% predicted peak oxygen consumption (VO₂). MACE was a

composite outcome of myocardial infarction (MI), coronary artery bypass graft (CABG), percutaneous coronary intervention (PCI), stroke/transient ischemic attack (TIA) and death. Coxproportional hazard models adjusting for factors known to influence CRF and MACE were constructed.

Results: Of 498 included patients (60 ± 13 years, 28.1% female), 175 (35%) had MACE (MI=17, PCI=14, CABG=13, stroke=20, TIA=12, deaths=99) at a median follow-up of 8.7 years (interquartile range=6.5-10.3 years). After adjusting for age, sex, beta-blockers, systemic hypertension, diabetes mellitus, coronary artery disease, cardiac arrhythmia, chronic obstructive pulmonary disease, smoking and positive airway pressure (PAP) usage, decreased CRF alone (HR=1.91, 95%CI=1.15-3.18, p=0.012), but not SDB alone (HR=1.26, 95%CI=0.75-2.13, p=0.389) was associated with increased risk of MACE. Those with SDB and decreased CRF had increased risk of MACE compared to patients with decreased CRF alone (HR=1.85, 95%CI=1.21-2.84, p<0.005) after accounting for these confounders; the risk was attenuated after additionally adjusting for adequate adherence to PAP (HR=1.85, 95%CI=0.99-3.05, p=0.05).

Conclusion: The incidence of MACE, including mortality, was high in this sample. Moderate-severe SDB with concurrent decreased CRF was associated with higher risk of MACE than decreased CRF alone. These results highlight the importance of including CRF in the risk assessment of patients with SDB, and conversely, that of screening for SDB in patients with low peak VO₂.

Support: None.

0720

STUDY OF SERUM ADIPONECTIN AND RESISTIN IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Gharraf, H. S.¹ Zidan, M. H.¹ Ayad, M. W.¹

¹Faculty of Medicine, Alexandria, EGYPT, ²Faculty of Medicine, Alexandria, EGYPT.

Introduction: The syndrome of OSA is a very common disorder with several important complications and is still under diagnosed. Obesity is considered to be a chronic disease rather than a life style b. Adipose tissue secretory function is disturbed by chronic intermitted hypoxia occurring in OSA. The aim of this study was to study the level of serum adiponectin and resistin in patients with OSA and we tried to assess their association to different parameters of SDB as well as concomitant co morbidities in each patient **Methods:** This study contained two groups. Patients (group1) included 30 newly diagnosed OSA patients; controls (group 2) included 15 healthy volunteers. All patients were subjected to overnight polysomnography, routine laboratory investigations, Spirometry and fasting serum adiponectin and resistin

Results: Patients had statically significant lower serum adiponectin level than controls while serum resistin levels were statistically significant higher in patients than controls also serum adiponectin levels were significantly decreased with increase severity of OSA while serum resistin levels were increased significantly with increase severity of OSA, moreover there was a statistical significant decrease in serum adiponectin levels in patients group with increasing number of comorbidities existing in every patient a. There was a statistical significant increase in serum resistin levels in patients group with increasing number of comorbidities existing in every patient

Conclusion: this highlights the possible relationship of these hormones to the metabolic complications seen in OSA patients **Support:** no conflict of interest

0721

CORONARY ATHEROSCLEROTIC PLAQUE BURDEN AND COMPOSITION IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA BY CORONARY CT ANGIOGRAPHY *Lu, M.¹ Wei, Y.² Wang, Z.³ Fang, F.² John, S. E.⁴ Xu, L.²* ¹Beijing Anzhen Hospital, Capital Medical University, Beijing 100029, China, Beijing, CHINA, ²Beijing Anzhen Hospital, Capital Medical University, Beijing, CHINA, ³Department of Radiology, Beijing Hospital of Traditional Chinese Medicine, Capital Medical University, Beijing, CHINA, ⁴Beijing Institute of Heart Lung and Bloos Vessel Disease, Beijing, CHINA.

Introduction: OSA is closely associated with increased risk of coronary artery disease. Although previous small studies have investigated coronary plaque in OSA patients, limited data are available regarding the association of OSA with plaque morphology and composition. Therefore, we aimed to quantitatively characterize and compare coronary plaque burden and composition between patients with no or mild obstructive sleep apnea (OSA) and moderate-severe OSA using coronary computed tomography angiography (CTA) in a large-scale study.

Methods: We retrospectively analyzed consecutive patients who underwent sleep monitoring and coronary CTA. Metrics reflecting coronary plaque characteristics were compared between patients with no or mild OSA with apnea hypoxic index (AHI) \leq 15 and moderate-severe OSA (AHI>15). The associations of OSA with coronary plaque components were determined by logistic and linear regression analysis.

Results: A total of 854 patients were enrolled in the study. Of these, 162 did not meet the inclusion criteria and of the remaining 692 patients 400 (57.8%) had moderate-severe OSA and 292 had no or mild OSA. Patients with moderate-severe OSA had a significantly higher total plaque volume, total non-calcified plaque (NCP) volume and total low density non-calcified plaque (LD-NCP) volume, and corresponding burden than those with no or mild OSA (all with p < 0.05). Multivariate logistic regression analysis revealed that moderate-severe OSA patients are more likely to have any plaque, NCP and LD-NCP than those without no or mild OSA (p < 0.05). In addition, stepwise multivariate linear regression analysis further revealed an independent relationship between moderate OSA (15<AHI≤30) and more so between severe OSA (AHI>30) and, NCP volume, LD-NCP volume, NCP composition, and LD-NCP composition, following adjustment for traditional cardiovascular risk factors, compared to no or mild OSA (AHI<15) (all with a p<0.05). Moderate-severe OSA conferred a similar odds ratio for LD-NCPs (a high-risk plaque) as the usual cardiovascular risk factors.

Conclusion: In this large cross-sectional study, OSA severity was associated with high-risk plaque features independent of traditional cardiovascular risk factors, suggesting an increased risk for cardiovascular events.

Support: This study was supported by NSFC (Project 81870335), International Science & Technology Cooperation Program of China (No.2015DFA30160), Beijing Municipal Science & Technology Commission (No. Z141100006014057)

0722

THE ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA AND NEUROBEHAVIOURAL FUNCTION IN MEN: A LARGE, POPULATION-BASED COHORT STUDY

Parker, J. L.¹ Adams, R. J.^{1,2,3} Appleton, S. L.^{1,2,3} Melaku, Y. A.¹ Vakulin, A.^{1,4}

II. Sleep-Related Breathing Disorders

¹Adelaide Institute for Sleep Health, College of Medicine and Public Health, Flinders University, Australia, Adelaide, AUSTRALIA, ²Respiratory and Sleep Service, Southern Adelaide Local Health Network, Bedford Park, South Australia, Australia., Adelaide, AUSTRALIA, ³School of Medicine, the University of Adelaide, Adelaide, South Australia, Adelaide, AUSTRALIA, ⁴Sleep and Circadian Research Group, Woolcock Institute of Medical Research, University of Sydney, New South Wales, Australia, Sydney, AUSTRALIA.

Introduction: Obstructive sleep apnea (OSA) is linked with impaired vigilance, attention, memory and executive function. However, this evidence largely comes from small experimental studies or larger studies in clinical samples and therefore the scope and magnitude of OSA driven neurobehavioural dysfunction in the general population remains unclear. This study aimed to examine the cross-sectional association between OSA and neurobehavioural function in a large community sample of men.

Methods: A total of 837 participants from the Men Androgen Inflammation Lifestyle Environment and Stress (MAILES) study, a longitudinal cohort of men 40+ years, underwent full overnight polysomnography. Participants completed the inspection time (IT) test, mini-mental state examination (MMSE), Fuld object memory evaluation (FOME), and trail-making test (TMT) part A (TMT-A) and part B (TMT-B). Using regression models adjusted for multiple important covariates, we examined the association between neurobehavioural function scores, clinical metrics of OSA severity (Apnea-Hypopnea Index (AHI); percentage total sleep time with oxygen saturation <90% (TST90), and measures of sleep disruption (duration of rapid eye movement (REM) and non-REM (NREM) sleep; and total sleep time (TST).

Results: In multivariable linear regressions, greater TST was associated with worse IT scores (B=13.688, 95% CI [0.134, 27.241], P=0.048) and TMT-B scores (B=19.255, 95% CI [0.931, 37.578], P=0.040). In logistic regressions, greater TST was associated with better MMSE scores (Odds ratio [OR]=0.440, 95% CI [0.194, 0.997], P=0.049); and higher AHI was strongly associated with worse FOME scores in fully adjusted models (OR=1.358, 95% CI [1.252, 1.472], P<0.001).

Conclusion: The AHI and TST were positively, significantly associated with neurobehavioural function across different domains. This cross-sectional data shows that neurobehavioural function deficits in OSA are directly related to sleep and breathing disruptions. Future large prospective studies are needed to determine if OSA and sleep disruption predict future onset of neurobehavioural dysfunction and cognitive decline.

Support: National Health and Medical Research Council and the Adelaide Institute for Sleep Health.

0723

ASSOCIATIONS BETWEEN OPIOIDS, NON-OPIOIDS AND CENTRAL SLEEP APNEA: A CASE-CONTROL STUDY

Gavidia, R.¹ Meng, A. L.² Emenike, A.³ Hershner, S.¹ Jansen, E.¹ Goldstein, C.¹ Dunietz, G. L.¹

¹University of Michigan, Ann Arbor, MI, ²Department of Statistics, Ann Arbor, MI, ³Tallahassee Memorial Healthcare, Tallahassee, FL, ⁴University of Michigan, Ann Arbor, MI.

Introduction: Opioids are known to contribute to central sleep apnea (CSA), as they depress responsiveness to carbon dioxide and hypoxia. However, the role of non-opioid medications (antihistamines, myorelaxants, neuroleptics, antidepressants, and hypotics)

in CSA remains unclear. Given the hypothesized impact of nonopioids on the central nervous system, we examined associations between opioid and non-opioid medications and CSA.

Methods: Among all adults who underwent polysomnography testing at the University of Michigan's Sleep Center between 2013-2018 (n=10,479), we identified 105 cases of CSA. Of these patients, we randomly selected 300 controls. Demographic and health characteristics, use of medications were obtained from medical charts. We classified study participants into three categories based on medication use: non-opioids only, opioids alone or in combination with non-opioids, and none. CSA was defined as a binary outcome using polysomnographic criteria as per the International Classification of Sleep Disorders-Third Edition. We used logistic regression to examine associations between medication use and CSA.

Results: Among participants, male:female ratio was 1:1 with a mean age of 49 (\pm 14.3 SD) years. Opioid use alone was rare (4%), but more common in combination with non-opioids (17%), while the exclusive use of non-opioids was found among 38%. In adjusted analyses for age and sex, those who used non-opioid alone were less likely to have a CSA diagnosis (OR=0.88, (95% CI 0.5-1.6); however, the use of opioids (alone or in combination with non-opioids) was associated with a 4-fold higher odds of CSA.

Conclusion: These data suggest that non-opioids have a protective influence on CSA. Conversely, opioids, alone, or in combination with non-opioids, were associated with increased CSA risk, that may be attributed to opioids alone, or to opioids and non-opioids interactions. However, as opioids were mostly co-prescribed with non-opioids, the sole effect of opioids from the synergistic effect with non-opioids are difficult to disentangle.

Support: Dr. Gavidia was supported by a T32 Post-Doctoral Fellowship in Neuroscience NIH/NINDS T32 NS 007222

0724

ALTERED BRAIN NETWORK ORGANIZATION IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA $Bruk_{-} U_{+}^{1} Cha_{-} U_{+}^{2} Kim_{-} U_{+}^{3} Lag = E_{+}^{4}$

Park, H.¹ Cha, J.² Kim, H.³ Joo, E.⁴

¹Department of Neurology, Inje University College of Medicine, Ilsan Paik Hospital, Goyang, KOREA, REPUBLIC OF, ²Nash Family Center for Advanced Circuit Therapeutics, Icahn School of Medicine at Mount Sinai, New York, NY, ³USC Stevens Neuroimaging and Informatics Institute, Keck School of Medicine of USC, University of Southern California, Los Angeles, CA, ⁴Department of Neurology, Neuroscience Center, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, KOREA, REPUBLIC OF.

Introduction: Previous functional MRI (fMRI) studies have reported altered brain networks in patients with obstructive sleep apnea (OSA), but the extent of such abnormal connectivity was inconsistent across studies. Moreover, despite the important role of the cerebellum in respiration and OSA, connections of the cerebellum to the cerebral cortex have been rarely assessed. Here, we investigated functional network changes in cerebral and cerebellar cortices of OSA patients.

Methods: Resting-state fMRI, polysomnography and neuropsychological (NP) tests data were acquired from 74 treatment naïve OSA patients (age: 45.8 ± 10.7 years, apnea-hypopnea index: 46.4 ± 18.5 /h) and 33 normal controls (39.6 ± 9.3 years). Connectivity matrices were extracted by computing correlation coefficients from various ROIs, and Fisher r-to-z transformations. ROIs consisted of

234 regions matched to 17 functional networks, including 200 parcels of the cortex, and 34 parcels of the cerebellum. Between-group connectivity with age as a covariate was analyzed, and threshold for FDR correction was set at q<0.05. In the functional connections that showed the significant group differences, linear regression was conducted to examine the association between connectivity and composite score of NP tests in OSA patients.

Results: OSA subjects showed decreased attention, executive function, verbal fluency and verbal memory compared to controls. Resting-state functional connectivity was increased between regions involved in the default mode network (DMN), including left medial prefrontal, ventrolateral prefrontal and lateral temporal cortices. In OSA, the connectivity changes between these DMN areas negatively correlated with attention/executive function and verbal fluency. Multiple cerebellar regions showed reduces in connectivity with cerebral cortical areas including frontal eye field, temporoparietal junction, temporo-occipital gyrus, and parietooccipital association cortex.

Conclusion: OSA affects mainly the DMN and cerebello-cerebral pathway. The disruption of function in these two networks are known to relate to sleep deprivation and respiratory abnormality. The abnormal DMN found in OSA patients further related to their cognitive impairment.

Support: This research was supported by Basic Science Research Program through the National Research Foundation of Korea funded by the Ministry of Science, ICT & Future Planning, Republic of Korea (2017R1A2B4003120) and by Samsung Biomedical Research Institute grant (OTC1190671)

0725

PRESHYON: THE PREVALENCE ON THE RISK OF OBSTRUCTIVE SLEEP APNEA AMONG PATIENTS WITH UNCONTROLLED HYPERTENSION SEEN AT THE OUT-PATIENT DEPARTMENT OF THE UNIVERSITY OF THE PHILIPPINES- PHILIPPINE GENERAL HOSPITAL

Tojino, A. G.¹ Jorge, M. C.¹ De Leon, L. F.¹ Chua, J. R.¹ Dela Cruz, A. V.¹

¹University of the Philippines-Philippine General Hospital, Manila, PHILIPPINES, ²University of the Philippines-Philippine General Hospital, Manila, PHILIPPINES.

Introduction: Obstructive Sleep Apnea (OSA) is a breathing disorder linked to increased morbidity and mortality, ¹ and associated with decreased quality of life and functional impairment of daily living.² OSA has been established to be associated with hypertension wherein the prevalence of OSA is 30%, ^{24,25} however, local prevalence has been lacking. The gold standard for OSA is polysomnography but this is expensive and has limited availability. Questionnaires provide risk stratification to determine if the patient needs further sleep evaluation. Recognition and treatment of OSA could lead to improving long term outcomes of patients with uncontrolled hypertension. ³¹⁻³³

Methods: This is a cross-sectional study that enrolled 325 adult Filipino HTN patients seen at the outpatient department (OPD) of UP-PGH from January 2019 to July 2019. Participants also answered Berlin Questionnaire (BQ), Epworth Sleepiness Scale (ESS) and the St. Lukes Medical Center-Obstructive Sleep Apnea Clinical Score (SLMC-OSACS) for their OSA risk. Descriptive statistics were used to summarize the clinical characteristics of the patients. Chi-square test was used to analyze categorical data univariately. Logistics regression analysis was used to determine the association of the different independent variable with the outcome (dependent) variable.

Results: The risk of OSA was significantly high among uncontrolled HTN patients: 106 (60.7%) based on BQ (p<0.0001) and 68 (69.4%) based on SLMC-OSACS (p<0.0001). OSA risk among uncontrolled HTN was 6x higher (OR=5.69; 95% CI=3.49-9.28;p<0.0001) using the BQ and 4x higher (OR=3.70; 95% CI=2.19 - 6.26;p=<0.0001) using the SLMC-OSACS than those with controlled HTN. Other variables significantly associated with high risk OSA were BMI and other comorbidities.

Conclusion: The risk of OSA was significantly high among uncontrolled HTN patients. In utilizing these sleep questionnaires and determining the risk for OSA among patients with uncontrolled hypertension will lead for early evaluation of OSA and complications. Locally this will aid in improving detection and provide a more strategic management of uncontrolled hypertension to decrease associated cardiovascular events. This will guide our clinicians to prioritize patients who would benefit referral to sleep specialists

Support:

0726

IMPACT OF ANESTHETIC AGENTS ON SLEEP APNEA SEVERITY

Albrecht, E.¹ Hirotsu, C.² Bayon, V.² Heinzer, R.² ¹Department of Anesthesia, Lausanne University Hospital, Lausanne, SWITZERLAND, ²Center for Investigation and Research in Sleep, Lausanne University Hospital, Lausanne, SWITZERLAND.

Introduction: Sleep apnea is associated with negative outcomes after general anesthesia. Current recommendations suggest using short-acting over standard anesthetic agents to reduce this risk, but there is currently no evidence to support this. This randomized controlled triple-blind trial tested the hypothesis that a combination of short-acting agents (desflurane-remifentanil) would reduce the postoperative impact of general anesthesia on sleep apnea severity compared with standard agents (sevoflurane-fentanyl).

Methods: Sixty patients undergoing hip arthroplasty under general anesthesia were randomized to anesthesia with desfluraneremifentanil or sevoflurane-fentanyl. Respiratory polygraphy was performed before surgery and on the first and third postoperative nights. The primary outcome was the supine apnea-hypopnea index on the first postoperative night. Secondary outcomes were the supine apnea-hypopnea index on the third postoperative night, and the oxygen desaturation index on the first and third postoperative nights. Additional outcomes included intravenous morphine equivalent consumption and pain scores on postoperative days 1, 2 and 3.

Results: Preoperative sleep study data were similar between groups. Mean (95% confidence interval) values for the supine apnea-hypopnea index on the first postoperative night were 18.9 (12.7-25.0) and 21.4 (14.2-28.7) events/h, respectively, in the short-acting and standard anesthesia groups (p=0.64). Corresponding values on the third postoperative night were 28.1 (15.8-40.3) and 38.0 (18.3-57.6) events/h (p=0.34). Secondary sleep- and pain-related outcomes were generally similar in the two groups.

Conclusion: Short-acting anesthetic agents did not reduce the impact of general anesthesia on sleep apnea severity compared with standard agents. These data should prompt an update of current recommendations.

Support: SNSF

0727

STUDY ONTHE EFFECT OF OBSTRUCTIVE SLEEP APNEA-HYPOPNEA SYNDROME ONPERIOPERATIVE MANAGEMENT INENDOSCOPIC SINUSSURGERYPATIENTS

Xiaojun, Z.¹ Chan, W.¹ Hao, W.¹ Fang, F.¹ Wei, X.¹ ¹Beijing Anzhen Hospital, Beijing, CHINA, ²Beijing Anzhen Hospital, Beijing, CHINA.

Introduction: To determine the frequency of undiagnosed OSA patients in patients received endoscopic sinus surgery (ESS) and to investigate the effect of OSA on the perioperative management in those patients.

Methods: 308 patients undergoing ESS from 2017-2019 were enrolled. The patients were divided into two groups according to whether OSA was combined. STOP-Bang questionnaire scoring system was used to classify patients into high risk and low risk for OSA. The differences between perioperative management and complications between the two groups were compared.

Results: 308 consecutive cases were included, 46 cases (14.9%) combined with OSA and 108 cases (35.0%) were at high risk of OSA. OSA patients have higher morbidity of hypertension (OR, 2.05; CI, 1.07-3.92; P=0.03), hyperlipidemia (OR, 2.19; CI, 1.06- 4.51; P=0.03), longer hospitalization time(7.0 ± 2.7 vs. 5.4 ± 3.6 , P≤0.01) and higher incidence of intubation difficulties (OR, 3.74; CI, 1.39-10.1; P=0.01). Patients at high risk of OSA also had increased rates of hypertension, hyperlipidemia, coronary heart disease and post-operative cardiovascular and respiratory complications.

Conclusion: OSA or high scores of STOP-Bang are associated with increased perioperative complications in ESS patients. Preoperative OSA screening should be strengthened to improve the safety and prognosis of ESS surgery.

Support: National Natural Science Foundation of China under Grant [number 81670903]; and Beijing Municipal Administration of Hospitals Ascent Plan under Grant [number DFL20150602]

0728

IMPULSIVITY IS RELATED TO OSA SEVERITY BUT NOT WITH EXCESSIVE DAYTIME SLEEPINESS

Marelli, S.^{1,2} Somma, A.² Castelnuovo, A.¹ Mombelli, S.¹ Gialdi, G.² Barranca, M.² Fossati, A.² Ferini Strambi, L.^{1,2} ¹Sleep Disorder Center, San Raffaele Hospital, Milan, ITALY, ²Faculty of Psychology, Vita-Salute San Raffaele University, Milan, ITALY.

Introduction: Obstructive sleep apnea (OSA) is associated with cognitive deficits in vigilance, attention, and executive functions (EFs). To date, the mechanisms that characterize the impairment cognition in OSA remain partially uncertain. The dominant theories have mostly concentrated on hypoxia/hypercarbia, and sleep disruption and consequent excessive daytime sleepiness. Moreover, hypoxemia affects global cognitive functioning, but it is not clear whether and how the severity of the disease influences the EFs.

Methods: The current study aimed at assessing the relationships among OSA severity, daytime sleepiness, and EFs and self-reported impulsivity in a sample of OSA subjects who were classified according to Apnea-Hypopnea Index (AHI) in mild/moderate OSA (AHI<30) or severe OSA (AHI≥30). All OSA participants were consecutively admitted to the Sleep Disorders Center of the San Raffaele Turro Hospital. After OSA assessment, patients were administered the Italian translation of the Psychology Experiment Building Language EFs tasks and the Epworth Sleepiness Scale (ESS) and *UPPS-P Impulsive Behavior Scale* (*UPPS-P*). Participants' sleepiness was considered severe if a score of 10 or higher on the ESS was observed.

Results: Confirming and extending previous reports, daytime sleepiness affected both attention performance (U=239.00, p<.05; Vargha & Delaney A=0.66), and inhibition performance (U=199.00, p<.05; A=0.70). No effect of AHI was observed on both variables (U=326.00, p>.05; A=0.55; U=330.00, p>.05; A=0.53, respectively). Conversely, self-reported impulsivity was associated with OSA severity (U=30.50, p<.001; A=0.86), but not with daytime sleepiness (U=84, p>.05; A=0.63).

Conclusion: Our findings showed the importance of considering both OSA severity and sleepiness in order to better understand EFs and impulsivity related to OSA.

Support: none

0729

SLEEP APNEA DIAGNOSIS AND SEVERITY AND THEIR IMPACT ON COGNITION AFTER TBI: A VA TBI MODEL SYSTEMS STUDY

Silva, M. A.¹ Brennan, E. M.² Noyes, E.² Royer, A.³ Nakase-Richardson, R.¹

¹James A Haley Veterans' Hospital, MHBSS, Tampa, FL, ²James A Haley Veterans' Hospital, Research Service, Tampa, FL, ³James A Haley Veterans Hospital, Research Service, Tampa, FL, ⁴James A Haley Veterans' Hospital, MHBSS, Tampa, FL.

Introduction: For persons with moderate-to-severe traumatic brain injury (TBI), chronic cognitive impairment contributes to long term disability. Health comorbidities may contribute to the neurologic burden in TBI. Indeed, obstructive sleep apnea (OSA) is associated with neuropathological and cognitive changes. The objective of this study was to examine the relationship between OSA and cognition after TBI.

Methods: Participants were prior inpatient rehabilitation patients drawn from the Tampa VA TBI Model Systems longitudinal study. Post-discharge interviews occurred 2 to 6 years post-TBI. Participants reported whether they were diagnosed with OSA and completed the Brief Test of Adult Cognition by Telephone (BTACT) which measures recall, working memory, processing speed, fluency, and reasoning. Participants with polysomnography (PSG) were separately analyzed to examine the impact of sleep apnea severity (i.e., Apnea-Hypopnea Index [AHI]) on cognition.

Results: Participants (N=104) were mostly male (95.2%), age M=37.7 (SD=12.5), Education M=13.6 years (SD=2.1), and 45.2% were diagnosed with OSA. Participants with and without OSA did not differ by age, education, gender, or time since injury at time of BTACT (ps > .05). ANCOVAs were conducted examining OSA diagnosis on BTACT subscale scores, covarying TBI severity level, but results did not reach statistical significance (ps > .05). A subset of participants with OSA had PSG (n=27), AHI score quartiles = 6.7/10.4/21.6. Higher AHI was associated with poorer reasoning (Spearman $\rho = -0.45$, p = .019). Nonsignificant results were found for word recall (Spearman $\rho = -0.36$, p = .074) and processing speed (Spearman $\rho = -0.36$, p = .069).

Conclusion: Severity of sleep apnea may influence aspects of cognition among persons with TBI, although these results are preliminary and need replication with a larger and more representative sample.

Support: This work was supported by the Veterans Health Administration Central Office VA TBI Model Systems Program of Research and subcontract from General Dynamics Information

Technology [W91YTZ-13-C-0015, HT0014-19-C-0004] from the Defense and Veterans Brain Injury Center (DVBIC); and from the United States Department of Veterans Affairs [W81XWH-13-2-0095]; and from the United States Department of Defense Congressionally Directed Medical Research Programs; and from the Patient Centered Outcomes Research Institute (PCORI) [CER-1511-33005].

0730

INDIVIDUALS RECEIVING METHADONE FOR MEDICATION-ASSISTED TREATMENT OF OPIOID USE DISORDER SHOW EVIDENCE OF RESPIRATORY DEPRESSION

Finlay, M.^{1,2} Wilson, M.^{1,3} Erwin, J. A.^{1,2} Hansen, D. A.^{1,2} Layton, M. E.^{1,2} Quock, R. M.⁴ Van Dongen, H.^{1,2}

¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ³College of Nursing, Washington State University, Spokane, WA, ⁴Department of Psychology, Washington State University, Pullman, WA.

Introduction: A well-established consequence of opiate use is respiratory depression during sleep, with a high prevalence of central sleep apneas. Medication-assisted treatment (MAT) is a widely used therapy for opioid use disorder (OUD) designed to reduce withdrawal symptoms and drug cravings. We investigated the presence of respiratory depression during sleep in patients receiving methadone-based opioid replacement treatment as part of a MAT program for OUD. **Methods:** N=6 individuals (5 females, ages 43.8 \pm 12.8y, BMI 27.2 \pm 4.1kg/m²), who were within 90 days of methadone initiation, underwent in-laboratory overnight polysomnography (8h TIB, 22:00-06:00). Apneaic and hypopneic events were determined using AASM criteria.

Results: The average Apnea-Hypopnea Index (AHI) was 16.5 ± 9.0 events/h, with 2 individuals exceeding the threshold of moderate sleep apnea (>15 events/h). 89.5% of the observed apnea-hypopnea events occurred during NREM sleep. Of all events, $57.1\pm16.3\%$ were central apneas; and of all obstructive, central, and mixed apnea events, $93.0\pm14.3\%$ were central apneas.

Conclusion: Individuals with OUD receiving methadone-based MAT may be at risk of respiratory depression during sleep, as evidenced by the frequent occurrence of central sleep apneas. Such risk could be a contributing factor in opioid overdose deaths. Currently, performing respiratory assessments during sleep is not considered standard of care in MAT programs. Our preliminary data suggest that monitoring and treatment of respiratory depression during sleep may be indicated in OUD patients on methadone-based MAT.

Support: Supported in part by a seed grant from the Washington State University Office of Research Advancement and Partnerships.

0731

OBSTRUCTIVE SLEEP APNEA WITH PREDOMINANT RESPIRATORY EFFORT RELATED AROUSALS: THETA POWER IN C3- M2 AND C4-M1 DERIVATION COMPARISON IN MALE AND FEMALE

Hura, K.¹ Singh, H.² Sahota, P.³ Thakkar, M.¹

¹University of Missouri, columbia, MO, ²university of missouri, University of Missouri, MO, ³University of Missouri, University of Missouri, MO.

Introduction: Theta power in electroencephalography has been studied as a correlate to REM sleep. An increase in theta power

during REM sleep has been observed in patients during recovery sleep after sleep deprivation. Emotional memories appear to be processed and consolidated during REM sleep. The role of hippocampal theta wave activity during REM sleep on emotional memory processing is limited. The importance of theta power has not been well characterized in patients with obstructive sleep apnea (OSA) with predominant respiratory Effort Related Arousals (RERAs). This report aims to study the theta power in patients with OSA with predominant Respiratory Effort Related Arousals (RERAs) with an apnea-hypopnea index (AHI) of < 5.

Methods: We have identified 38 patients with baseline polysomnograms performed from December 2019 to July 2019 with AHI < 5 and a Respiratory Disturbance Index (RDI) of at least 5 or greater. Patients with chronic hypoxemic respiratory failure, hypoventilation and predominant central sleep apnea were excluded from the study. Total power of frequency in bands was obtained for theta waves (4-8 Hz) and total waves (1-30 Hz). Relative theta power was calculated on the last REM sleep using C3-M2 and C4-M1 derivations. Paired two-tailed t-Test was performed on the theta power in C3-M2 and C4-M1 in both the sexes. **Results:** Initial analysis was performed in 38 patients out of which 20 were male and 18 female. Among males, (Mean \pm SEM) age was 52.3 (±2.9); Epworth Sleepiness Scale (ESS) of 6.6 (±1.1), AHI of 2.1 (± 0.3), and RDI of 7.3 (± 0.3). Whereas in female (Mean \pm SEM) age was 46.8 (± 2.8), ESS of 7.7 (±1.4), AHI of 2.3 (±0.35), and RDI of 6.9 (\pm 0.4). Statistically significant difference was noted in the theta power between the C3-M2 and C4-M1 derivations with P value of 0.03 and 0.04 in male and female respectively. However, no significant difference was found when C3-M2 and C4 -M1 was compared between male and female. Further, statistical analysis will be performed after gathering data from a larger sample size. Conclusion: There was significant difference between C3-M2 and C4-M1; overall no difference was found between sexes. Support: none

0732

QUANTITATIVE CHARACTERIZATION OF SLEEP DISORDERED BREATHING DYNAMICS

Chen, S.¹ Eden, U.¹ Prerau, M.²

¹Boston University, Boston, MA, ²Harvard Medical School, Boston, MA.

Introduction: Sleep-disordered breathing (SDB) is a dynamic process in which the rate of respiratory events is highly influenced by numerous covariates, such as sleep stage, body position dominance, time of night, and overall instability of sleep architecture. Additionally, respiratory event rate likely has history dependence, such that the likelihood of a respiratory event is influenced by the timing of previous events. Despite its dynamic nature, clinical diagnosis collapses the complex process of SDB to a single number measuring the average rate of respiratory event occurrence— the apnea-hypopnea index (AHI). Thus, potentially valuable information is being lost by ignoring SDB temporal dynamics and history dependence.

Methods: We apply a general point process framework to sleep apnea events to develop parametric models of a time-varying instantaneous apnea rate given clinical covariates (e.g. EEG power, sleep stage, body position). Develop models of apnea history dependence, describing the likelihood of events given past event times. In doing so, we are able to compute an "instantaneous AHI", which measures the moment-by-moment event rate, which evolves temporally as a function of other clinical observations as well as event history. We apply our model to data from the MESA cohort (**include number of subjects, male/female here**). We then applied dimensionality-reduction methods to assess any population phenotypes.

Results: For every subject, we computed a time-varying AHI for each time point in their polysomnogram and estimated the influence of each of the measured covariates on the instantaneous rate. Results showed that the greatest predictor of apnea events were related to history dependence. Clustering analysis showed no distinct clusters, but rather a constant gradient of changes in history dependence.

Conclusion: These results suggest that the greatest predictor of an apnea event onset is the timing previous event. Moreover, the way in which previous events influence subsequent events can be used as a means of phenotyping, paving the way towards identifying optimal personalized treatment.

Support: National Institute of Neurological Disorders and Stroke Grant R01 NS-096177

0733

RETROSPECTIVE PAIN REPORTS IN OSA PATIENTS: ROLES OF DEPRESSIVE SYMPTOMS, POLYSOMNOGRAPHIC AND SELF-REPORT SLEEP MEASURES

Weingarten, J. A. Dubrovsky, B. Cunningham, J. Chin, W. Howladar, A. Gikashvili, L.

New York-Presbyterian Brooklyn Methodist Hospital, Department of Medicine, Division of Pulmonary and Critical Care, Center for Sleep Disorders, Brooklyn, NY.

Introduction: Exploring the relationship between OSA and pain, some studies showed hyperalgesia, and others, hypoalgesia. It was proposed that apnea-related sleep fragmentation causes hyperalgesia, and hypoxemia, hypoalgesia. However, SpO2 nadir had opposite relationships with pain measures in different studies. A 2018 review of over 1000 studies reported lack of consistent relationship between OSA and pain variables. Further, OSA was shown to relate to depressed mood, which may alter pain perception. Presently, retrospective reports of pain are analyzed as a function of polysomnographic and self-report sleep variables and depressive symptomatology in patients evaluated for OSA.

Methods: A total of 1,166 patients (923 women, 1136 minorities, 18-97 y.o., age M=53.1 \pm 15.2, BMI M=34.4 \pm 8.7) undergoing an overnight PSG filled out the Center for Epidemiologic Studies Depression Scale-Revised (CESDR), ISI, PSQI, ESS, and Chronic Pain Grade Scale yielding pain intensity (PI) and functional effect (FE) scores. PI and FE were separately regressed onto age, sex and BMI, followed by PSG and self-report variables meeting p<0.1 criterion. AHI and SpO₂nadir were forced into the models.

Results: Mean AHI=29.6±34.7, range 0-167/hr, 72.3% had AHI≥5. Higher PI related to higher AHI (p=0.005, R²<1%), lower total arousal index (TAI, p=0.006, R²<1%), higher total sleep time (TST, p=0.003, R²<1%), higher PSQI (p<0.001, R²=5%), and higher CESD (p=0.001, R²<1%), without interactions with sex. Higher FE related to higher AHI (p=0.004, R²<1%), lower TAI (p<0.001, R²=1%), higher PSQI (p<0.001, R²=3%, and higher CESD (p<0.001, R²=2%). Sex had a significant interaction only with AHI (p=0.032); the FE-AHI relationship was significant in women (p=0.012), but not in men.

Conclusion: On retrospective reports of pain in this large sample, higher AHI related to greater pain intensity in both sexes and to greater functional effect in women only. Unexpectedly, higher pain

measures were also related to lower TAI and higher TST. Higher depressive symptomatology and subjective sleep disturbance on PSQI were related to greater pain intensity and its functional effect. Only a small portion of the variance in pain measures was accounted for by PSG and self-report variables. **Support:** none

0734

DOES NOISE MASKING IMPROVE SLEEP CONSOLIDATION IN PATIENTS WEANING FROM PROLONGED MECHANICAL VENTILATION?

Shaikh, H.^{1,2} Chung, P.² Jubran, A.^{2,1} Tobin, M.^{2,1} Laghi, F.^{2,1} ¹Hines VA Hospital, Hines, IL, ²Loyola University Medical Center, Maywood, IL, ³Hines VA Hospital, Hines, IL.

Introduction: Sound masking is a noise reduction strategy that adds a mixed-frequency blend of ambient sound to the environment and may improve sleep. Critically ill patients often cite noise as one of the main factors preventing sleep while they are cared for in an intensive care unit (ICU). The effect of sound masking on sleep in patients weaning from prolonged mechanical ventilation is unknown.

Methods: 12-hour overnight polysomnography was obtained in eight patients undergoing weaning from prolonged mechanical ventilation. None had hearing impairment, delirium, sedation or agitation. In random order, patients were exposed to sound masking half of the recording time. Noise events were defined a 10dB increase from baseline or any sound peak over 75dB. Arousals or awakenings were attributed to noise if they occurred within 5 seconds of the noise event.

Results: Environmental sound was 61.7 ± 0.9 dB (mean±SE) during sound masking and 55.9 ± 1.4 dB during no sound masking. During sound masking, there were fewer sound events per hour of sleep when compared to no sound masking (4.1/hr vs 9.3/hr p=0.03). The percentage of sound events leading to a subsequent arousal or awakening with sound masking was less than during no sound masking:11% vs 22% (p=0.04). Arousal index and fragmentation index (arousal and awakenings/hr of sleep) were similar between the two conditions. In a post-study survey, five patients reported improved sleep quality with sound masking while the remaining three reported no difference.

Conclusion: Sound masking decreases sound-induced arousal from sleep in patients being weaned from prolonged mechanical ventilation.

Support: Veterans Administration Research Service

0735

HYPERGLYCEMIA IS ASSOCIATED WITH AN ALTERED SLEEP STRUCTURE IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Xue, P.¹ Gao, Y.² Zhou, J.³ Tang, X.³

¹West China hospital of Sichuan University, Chengdu, CHINA, ²Department of Endocrinology and Metabolism, West China Hospital, Sichuan University, Chengdu, CHINA, ³Sleep Medicine Center, West China Hospital, Sichuan University, Chegndu, China, Chengdu, CHINA.

Introduction: Although obstructive sleep apnea (OSA) is associated with abnormal glycometabolism, current evidence on the association between hyperglycemia and abnormal sleep structure is still limited among patients with OSA. We sought to evaluate whether hyperglycemia was associated with abnormal sleep architecture.

Methods: A total of 452 patients with OSA, who were free of previously diagnosed diabetes mellitus, were consecutively recruited. All participants underwent overnight polysomnography and 75-g oral glucose tolerance test. Patients were divided into normal glucose tolerance (NGT) and hyperglycemia (i.e. prediabetes and type 2 diabetes) according to the ADA criteria. The association between hyperglycemia and sleep architecture was examined using logistic regression model.

Results: Of 452 patients, 283 (63%) had hyperglycemia (age 43.9 \pm 11.1) and 169 (37%) had NGT (age 40.1 \pm 11.1). Compared to the NGT group, the hyperglycemia group had older age (P < 0.05), higher body mass index (27.5 \pm 4.1 vs. 26.33 \pm 4.4; P < 0.05) and higher AHI (apnea-hypopnea index) (57.41 \pm 28.6 vs. 48.3 \pm 28.2; P < 0.05). There were no differences in total sleep time, the percentage of time spent in rapid eye movement (REM) or non-rapid eve movement (NREM) sleep between groups. However, patients with hyperglycemia had more microarousal events, especially during the NREM sleep (214 (range 19-662) events/h vs. 148 (range 37-600) events/h; P < 0.05). In addition, sleep variables related to oxygen saturation measures, such as the percentage of time spent with oxygen saturation ≤80%, were significantly greater during the REM sleep in patients with hyperglycemia (1.4 (total range 0-91.1) % vs. 1.1(0-78.6) %; P < 0.05). After adjusting potential confounders, logistic regression analyses showed that the presence of hyperglycemia was independently associated with the number of microarousals in NREM sleep (OR = 1.01, 95% CI = 1.00-1.02, P = 0.02).

Conclusion: Hyperglycemia is independently associated with abnormal sleep architecture among patients with OSA. Patients with hyperglycemia have significantly increased sleep fragmentation in NREM sleep and significantly increased hypoxia in REM sleep.

Support: This work was supported by the National Natural Science Foundation of China (81700087).

0736

SELF-REPORTED SLEEP IN OSA PATIENTS: ROLES OF POLYSOMNOGRAPHIC MEASURES AND DEPRESSIVE SYMPTOMS

Dubrovsky, B. Weingarten, J. A. Cunningham, J. Howladar, A. Chin, W. Gikashvili, L.

New York-Presbyterian Brooklyn Methodist Hospital,

Department of Medicine, Division of Pulmonary and Critical Care, Center for Sleep Disorders, Brooklyn, NY.

Introduction: Sleep fragmentation is typical in OSA, which is commonly co-morbid with insomnia and depression. A complex interaction between these conditions may be also gender-dependent. Moreover, self-report measures of sleep quality and insomnia, such as PSQI and ISI, may relate to depression symptoms more than polysomnographic sleep disturbance. The present aim is to ascertain relative contributions of polysomnographic variables and depression symptoms to PSQI and ISI in a large sample of OSA patients. The interaction between depressive symptomatology and gender in their relationships with subjective sleep is also analyzed.

Methods: A total of 1,166 patients (923 women, 1136 minorities, 18-97 y.o., age M=53.1 \pm 15.2, BMI M=34.4 \pm 8.7) undergoing an overnight PSG filled out the Center for Epidemiologic Studies Depression Scale-Revised (CESDR), ISI and PSQI. ISI and PSQI were separately regressed onto age, sex and BMI, followed by PSG variables meeting p<0.1 criterion when tested individually, followed by CESDR and CESDR-by-sex interaction.

Results: Mean AHI=29.6±34.7, range 0-167/hr, 72.3% of patients had AHI≥5. The PSQI final model included total sleep time (TST), sleep efficiency (SEF), WASO, PLM index, CESDR and CESDR-by-sex. Only CESDR and CESDR-by-sex were significant (p<0.001, p=0.023, respectively). Higher CESDR predicted higher PSQI in both sexes (both p<0.001), accounting for a greater portion of PSQI variance in men (R²=39%) than in women (R²=29%). The ISI final model included TST, N3%, REM%, SEF, WASO, total arousal index, AHI, PLM index, CESDR and CESDR-by-sex. Higher ISI related to lower TST (p=0.042, R²<1%), higher REM% (p=0.016, R²<1%), and higher CESDR (p<0.001, R²=42%). CESDR-by-sex was not significant.

Conclusion: In this large sample, after controlling for demographic variables, PSG parameters had only minimal relationship with self-report insomnia and sleep quality measures. Higher depressive symptomatology was associated with higher subjective sleep disturbance on PSQI and worse insomnia symptoms on ISI in both sexes, accounting for 29-42% of the variance.

Support: none

0737

DIAGNOSTIC VALUE AND FINANCIAL EFFECTS OF RECERTIFICATION POLYSOMNOGRAPHY IN MEDICARE PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Neill, S. E. Majid, R.

University of Texas Health Science Center in Houston, Houston, TX.

Introduction: The annual cost of diagnosis and treatment of obstructive sleep apnea (OSA) exceeds 12.4 billion dollars in the United States. The Centers for Medicare and Medicaid Services (CMS) require that after initiation of positive airway pressure (PAP) therapy patients have physician follow up and comply with specific requirements. Otherwise, continued PAP benefits are terminated and patients must undergo repeat sleep testing to reinstate therapy. Repeat testing can become an economic burden. We hypothesize that restudying patients prior to reinstating PAP therapy does not change the diagnosis and may only result in increased health care costs.

Methods: A chart review of polysomnographic studies (PSG) was performed on Medicare referrals made for the purposes of recertification to the Memorial Hermann Sleep center between October 2018 and 2019. Demographic and diagnostic data (including AHI) were collected. The percentage of patients with a change of diagnosis between the initial study and the recertification study was documented.

Results: 429 Medicare patients were referred for polysomnography. 34 patients were referred for PAP recertification. The average age in the recertification group was 65 years, 47% were male with an average BMI of 33.4 kg/m^2 . The average AHI on the recertification study was 33.5 events/hour (range 7-114). None of the patients sent for PAP recertification by polysomnography had a negative study for OSA.

Conclusion: Repeat PSG did not change the need for PAP therapy in patients originally diagnosed with OSA (all the patients continued to qualify). The mandatory referral of all patients who do not meet the CMS requirements for continued benefits for PAP, represents an extra cost to the health care system without a change in the clinical therapy. This money may better be utilized in providing patient education known to improve adherence to PAP. **Support:** N/A

0738

ADVANCE TAPER OF ANTIDEPRESSANTS PRIOR TO MULTIPLE SLEEP LATENCY TESTING INCREASES THE NUMBER OF SLEEP-ONSET RAPID EYE MOVEMENT PERIODS AND REDUCES MEAN SLEEP LATENCY

Kolla, B.¹ Jahani Kondori, M.² Silber, M.³ Samman, H.⁴

Dhankikar, S.⁵ Mansukhani, M. P.³

¹Mayo Clinic, Rochester, MN, ²Center for Sleep Medicine, Mayo Clinic, MN, ³Center for Sleep Medicine, Rochester, MN, ⁴Phelps Health, Rollo, MO, ⁵Burrell Behavioral Health, Springfield, MO.

Introduction: Patients presenting with excessive sleepiness are frequently on antidepressant medication(s). While practice parameters recommend discontinuation of antidepressants prior to multiple sleep latency testing (MSLT), data examining the impact of tapering these medications on MSLT results are limited.

Methods: Adult patients who underwent MSLT at Mayo Clinic Rochester, Minnesota, between 2014-2018 were included. Clinical and demographic characteristics, medications, including use of rapid eye movement suppressing antidepressants (REMS-AD) at assessment and during testing, actigraphy and polysomnography data were manually abstracted. The difference in number of sleeponset rapid eye movement periods (SOREMS), proportion with \geq 2 SOREMS and mean sleep latency (MSL) in patients who were on REMS-AD and discontinued prior to testing versus those who remained on REMS-AD were examined. At our center, all antidepressants are discontinued 2 weeks prior to MSLT wherever feasible; fluoxetine is stopped 4 weeks prior. Regression analyses accounting for demographic, clinical and other medication-related confounders were performed.

Results: A total of 502 patients (age=38.18±15.90 years; 67% female) underwent MSLT; 178 (35%) were on REMS-AD at the time of assessment. REMS-AD were discontinued prior to testing in 121/178 (70%) patients. Patients tapered off REMS-AD were more likely to have ≥ 2 SOREMS (OR-12.20; 95%CI=1.60-92.94) compared to patients who remained on REMS-AD at the time of the MSLT. They also had shorter MSL (8.77±0.46 vs 10.21±0.28; p>0.009) and higher odds of having ≥ 2 SOREMS (OR=2.22; 95%CI=1.23-3.98) compared to patients not on REMS-AD at initial assessment. These differences persisted after regression analyses accounting for confounders.

Conclusion: Patients who taper off REMS-AD prior to MSLT are more likely to demonstrate ≥2SOREMs and have a shorter MSL. Pending further prospective investigations, clinicians should preferably withdraw REMs-AD before an MSLT. If this is not done, the test interpretation should include a statement regarding the potential effect of the drugs on the results. **Support:** None

0739

EFFICACY AND SAFETY OF AXS-12 IN THE TREATMENT OF NARCOLEPSY: RESULTS FROM A PHASE 2, DOUBLE-BLIND, PLACEBO-CONTROLLED, CROSSOVER TRIAL

O'Gorman, C.¹ Jones, A.¹ Thorpy, M. J.² Tabuteau, H.¹ ¹Axsome Therapeutics, New York, NY, ²Sleep-Wake Disorders Center, Montefiore Medical Center, Bronx, NY.

Introduction: Narcolepsy is a chronic, debilitating, neurological disease characterized by excessive daytime sleepiness (EDS), cataplexy, and sleep-wake dysregulation. Existing treatments are limited, provide variable efficacy, and have significant tolerability issues. AXS-12 (reboxetine) is a potent, and highly selective

norepinephrine reuptake inhibitor with potential for therapeutic differentiation in narcolepsy.

Methods: The CONCERT study was a Phase 2, double-blind, randomized, placebo-controlled, crossover trial of AXS-12 in narcolepsy subjects exhibiting moderate and severe symptoms of cataplexy and EDS. Subjects were randomized (1:1) to treatment with AXS-12 followed by placebo, or placebo followed by AXS-12. AXS-12 dosing was 8mg/day for week 1, escalated to 10mg/day for week 2. Crossover occurred after a one-week down-titration/ washout. The primary endpoint was the change in weekly cataplexy attacks from baseline, averaged over the 2-week treatment period for overall treatment effect. Secondary endpoints included improvements in EDS, cognitive function, sleep quality and sleep-related symptoms.

Results: Twenty-one subjects were randomized. The baseline mean weekly number of cataplexy attacks was 30.0 and mean ESS score was 18.1, reflecting moderate and severe illness on both core symptoms. AXS-12 met the prespecified primary endpoint, demonstrating a statistically significantly greater reduction in the mean number of weekly cataplexy attacks (-13.0 with AXS-12 vs -0.3 with placebo; p<0.001) over 2 weeks of treatment. Statistically significant reductions in EDS were observed for AXS-12 compared to placebo, assessed by changes in the Epworth Sleepiness Scale (-6.0 vs -3.1; p=0.003), number of weekly inadvertent naps (-5.88 vs -0.98; p<0.001). AXS-12 was associated with improved cognitive function (p<0.002) and sleep quality (p<0.007), and reduced night awakenings (p<0.05). Rapidity of effect was observed with significant symptomatic improvements occurring as early as week 1 starting at the lower 8mg dose. AXS-12 was safe and well-tolerated with no serious adverse events or discontinuations due to adverse events.

Conclusion: AXS-12 is a novel approach for the treatment of narcolepsy with the potential for comprehensive clinical symptom management compared to current treatments. In this Phase 2 study, AXS-12 resulted in statistically significant improvements in cataplexy, excessive sleepiness, cognitive function and night awakenings in patients with narcolepsy with a favorable safety profile. **Support:** Axsome Therapeutics.

0740

QUALITY OF LIFE IN PHASE 3, PLACEBO-CONTROLLED, DOUBLE-BLIND, RANDOMIZED WITHDRAWAL STUDY OF JZP-258 IN ADULTS WITH NARCOLEPSY WITH CATAPLEXY

Foldvary-Schaefer, N.¹ Thorpy, M. J.² Dauvilliers, Y.³ Roy, A.⁴ Tang, L.⁵ Skowronski, R.⁶ Šonka, K.⁷ Bogan, R. K.⁸ ¹Cleveland Clinic, Cleveland, OH, ²Albert Einstein College of Medicine, Bronx, NY, ³Sleep and Wake Disorders Centre, Department of Neurology, Gui de Chauliac Hospital, Montpellier, FRANCE, ⁴Ohio Sleep Medicine Institute, Dublin, OH, ⁵Jazz Pharmaceuticals, Palo Alto, CA, ⁶Jazz Pharmaceuticals, Inc, Palo Alto, CA, ⁷First Faculty of Medicine, Charles University and General University Hospital, Prague, CZECH REPUBLIC, ⁸University of South Carolina School of Medicine, Columbia, SC.

Introduction: Narcolepsy negatively impacts health-related quality of life (HRQoL). Sodium oxybate is a standard of care for the treatment of cataplexy and excessive daytime sleepiness in narcolepsy. JZP-258 is an oxybate product candidate with 92% less sodium. Efficacy and safety of JZP-258 were established in a

double-blind randomized withdrawal study in adults with narco-lepsy with cataplexy.

Methods: Participants 18-70 years of age began JZP-258 treatment during a 12-week, open-label, optimized treatment and titration period, followed by a 2-week stable-dose period (SDP). Participants were then randomized to receive placebo or continue JZP-258 treatment during a 2-week, double-blind, randomized withdrawal period (DBRWP). HRQoL assessments included the 36-Item Short Form Health Survey Version 2 (SF-36) and 5-level EuroOoL 5-Dimensions Self-Report Ouestionnaire (EO-5D-5L).

Results: 201 participants enrolled; 134 were randomized and received at least 1 dose of double-blind study medication (efficacy population; placebo, n=65; JZP-258, n=69). Decreased scores (worsening) were observed in participants randomized to placebo compared with participants randomized to continue JZP-258 treatment for the SF-36 physical component summary (median [Q1, Q3], -1.92 [-3.46, 1.73] for placebo and -0.03 [-2.07, 2.41] for JZP-258; nominal P=0.02), SF-36 mental component summary (-1.92 [-6.28, 1.34] for placebo and 1.55 [-1.88, 3.78] for JZP-258; nominal P=0.03), and EQ-5D-5L visual analog scale (-5.00 [-10.0, 5.00] for placebo and 0 [0, 5.00] for JZP-258; nominal P=0.03). No change was observed in the EQ-5D-5L crosswalk index (0 [-0.05, 0.03] for placebo and 0 [-0.01, 0.03] for JZP-258; nominal P=0.39). The overall safety profile of JZP-258 was similar to sodium oxybate.

Conclusion: HRQoL worsened in those randomized to placebo during DBRWP but remained stable in participants who continued JZP-258 treatment.

Support: Jazz Pharmaceuticals

0741

DIAGNOSING NARCOLEPSY TYPE II IN THE ACTIVE DUTY POPULATION

Villarreal, B.¹ Powell, T.¹ Hansen, S.¹

¹Wilford Hall Ambulatory Surgical Center, Lackland AFB, TX, ²Wilford Hall Ambulatory Surgical Center, Lackland AFB, TX.

Introduction: Introduction: There is a high prevalence of sleep disturbances in U.S. military personnel which can significantly impact continued military service. Narcolepsy is a rare sleep disorder and testing results are often variable, especially when cataplexy is absent. Given the high incidence of confounding sleep disorders in military personnel, including insufficient sleep duration and sleepdisordered breathing, a narcolepsy diagnosis may be even more challenging. As this diagnosis is often incompatible with continued military service, extra attention should be given to confounding diagnoses in military personnel.

Methods: This is a retrospective study of patients aged 18-65, who were diagnosed with narcolepsy type 1 or type 2 at an outside location and subsequently underwent repeat evaluation at our facility. Polysomnography, actigraphy, and multiple sleep latency test results from the time of the original diagnosis, if available, and repeat evaluation were reviewed. The initial diagnosis was compared to the results of repeat testing and the prevalence of narcolepsy vs. another sleep disorder was assessed.

Results: Two of twenty-three patients retained a diagnosis of narcolepsy on repeat testing. 10 patients were diagnosed with insufficient sleep syndrome (< 7 hours sleep/night), 5 displayed significant circadian misalignment (including irregular sleep/wake periods or delayed sleep phase), and 8 were diagnosed with mild obstructive sleep apnea. 7 of the 8 OSA patients had supine predominate OSA, which may have contributed to not being diagnosed

on initial testing. The average total sleep time of the group was 6 hours and 49 minutes, consistent with insufficient sleep duration. **Conclusion:** Narcolepsy type 1 and type 2 are exceedingly rare disorders with diagnostic test results that may be influenced by the presence of other common sleep disorders. In this study, re-evaluation resulted in a changed diagnosis in 91 percent of patients, emphasizing the importance of ruling out these confounding diagnoses.

Support:

0742

HYPERSOMNIA SEVERITY INDEX: RELIABILITY AND VALIDITY IN A BEHAVIORAL SLEEP MEDICINE CLINICAL SAMPLE

Amatrudo, G. Puzino, K. Bourchetin, E. Calhoun, S. L. Fernandez-Mendoza, J.

Penn State College of Medicine, Hershey, PA.

Introduction: There is a need for patient-reported outcome measures of central disorders of hypersomnolence (CDH) that adequately assess both essential features and associated daytime impact. The Hypersomnia Severity Index (HSI) was designed to assess severity, distress and impairment of hypersomnolence in persons with psychiatric disorders. Little data is available regarding its psychometric properties in clinical samples with diverse sleep disorders, including CDH.

Methods: 158 consecutive patients (44.11±16.38 years old, 70.9% female, 19.6% minority) who were evaluated at the Behavioral Sleep Medicine (BSM) program of Penn State Hershey Sleep Research & Treatment Center completed the HSI and the Epworth Sleepiness Scale (ESS). All patients were diagnosed using ICSD-3 criteria, with 10 % receiving a diagnosis of CDH, 54% of insomnia disorder (ID) and 36% of other sleep disorders (oSD).

Results: The HSI showed satisfactory internal consistency (Cronbach's α =0.79) and item-total correlations (r=0.42-0.67), except for item 1 (r=0.17). Principal component analysis provided a 2-factor structure (HSI-Symptoms and HSI-Impact) explaining 56.20% of the variance. Convergent validity with ESS was optimal (r=0.65) but greater for HSI-Symptoms (r=0.69) than HSI-Impact (r=0.39). Criterion validity showed significantly higher scores in subjects with CDH (22.63±7.57) and significantly lower scores in subjects with ID (16.96±5.96) as compared to those with oSD (18.65±6.65); however, these divergent scores were primarily driven by the HSI-Symptoms score (p<0.01) rather than the HSI-Impact score (p>0.12).

Conclusion: The HSI shows satisfactory indices of reliability and validity in a clinically-diverse sleep disorders sample. Its criterion validity is supported by its divergent association with insomnia vs. hypersomnia disorders. Future studies should examine cut-off score for the HSI to reliably identify CDH and test its sensitivity to treatment effects.

Support: Department of Psychiatry, Penn State College of Medicine

0743

THE PHARMACOKINETIC ADVERSE EVENT RELATIONSHIP FOR FT218, A ONCE-NIGHTLY SODIUM OXYBATE FORMULATION

Seiden, D. Grassot, J. Monteith, D. Dubow, J. Avadel Pharmaceuticals, Chesterfield, MO.

Introduction: Sodium oxybate is an effective treatment for excessive daytime sleepiness and cataplexy in patients with narcolepsy. The

approved formulation requires twice-nightly dosing: at bedtime and 2.5 - 4 hours later, which results in two distinct Cmax's. FT218 is a controlled-release formulation of sodium oxybate intended for once-nightly dosing, using Avadel's proprietary MicropumpTM technology. The objective of this study was to evaluate the pharmacokinetic-adverse event (AE) relationship for FT218, an investigational once-nightly sodium oxybate formulation.

Methods: Six single-dose, randomized, crossover studies that assessed the pharmacokinetics of FT218 at 4.5, 6, 7.5 and 9 g in healthy voluntters were used in this analysis. Lattice plots, "spaghetti" plots, and scatter plots of individual gamma hydroxybutyrate concentrations and indicators when AEs by system, organ, or class (SOC) were created to determine any PK-AE relationship.

Results: A total of 129 healthy volunteers received single doses of FT218 between 4.5 - 9 g. Most AEs, specifically for the neurological and gastrointestinal SOC, occurred close to T_{max} , during the C_{max} period, which for FT218 was around 1.5-2 hours after dosing. These AEs were known AEs associated with sodium oxbyate. There appeared to be no clear correlation between individual plasma GHB concentrations levels and AEs between subjects. Individual AEs were equally distributed above and below the mean population C_{max} and AUC_{inf} for the dataset.

Conclusion: In general, adverse events for FT218 occurred around T_{max} . There was no clear population toxicokinetic range for when AEs occur with FT218, but mostly individual thresholds. Since it appears AEs are related to C_{max} , and FT218 only has one C_{max} compared to two with the currently available product, it is hypothesized that FT218 will have a favorable safety profile compared to twice-nightly dosing.

Support: This work was supported by Avadel Pharmaceuticals

0744

CARDIAC SAFETY PROFILE OF PITOLISANT IN PATIENTS WITH NARCOLEPSY

Winter, W.¹ Wanaski, S. P.² Patroneva, A.³ Dayno, J. M.³ ¹Charlottesville Neurology and Sleep Medicine, Charlottesville, VA, ²Paragon Biosciences, Chicago, IL, ³Harmony Biosciences, LLC, Plymouth Meeting, PA.

Introduction: Cardiovascular diseases are comorbid in patients with narcolepsy. Cardiovascular adverse effects are of concern with narcolepsy medications because of this comorbidity and most patients require lifelong pharmacotherapy. Pitolisant, a selective histamine 3 (H_3)-receptor antagonist/inverse agonist, increases histamine transmission in the brain. In a QT study of healthy volunteers, pitolisant (35.6 mg/day) led to a mean increase of 4.2 msec in QTc interval. This analysis further characterized the cardiac safety of pitolisant (maximum dose, 35.6 mg/day) in adults with narcolepsy.

Methods: Data were obtained from a pooled analysis of 2 randomized, placebo-controlled, 7- or 8-week studies and from a 12-month, open-label study.

Results: Pooled analysis included 166 patients (pitolisant, n=85; placebo, n=81). Mean change in heart rate from baseline to end-of-treatment was -0.5 beats/min with pitolisant and -0.2 beats/min with placebo (LS mean difference, -0.4; P=0.744). Mean change was also similar for pitolisant versus placebo in systolic (LS mean difference, 0.0; P=0.983) and diastolic (LS mean difference, -0.6; P=0.552) blood pressure, as was mean change in QTc interval (LS mean difference, 0.4; P=0.911). Cardiac adverse events with pitolisant included heart rate increase (n=4), right bundle branch block (n=1), sinus tachycardia (n=1), and palpitations (n=1), and with placebo included

blood pressure increase (n=1). In the long-term study, mean change from baseline in QTc interval was 3.1 msec at Month 6 (n=70) and 6.1 msec at Month 12 (n=67); 3 patients had a postbaseline increase >60 msec but none had QTc >500 msec.

Conclusion: In this analysis, no cardiac safety signals were observed during treatment with pitolisant administered up to the maximum recommended dose. Because concomitant use of pitolisant with other drugs known to increase the QT interval may add to the QT effects of pitolisant, avoid use of pitolisant in combination with these medications.

Support: Bioprojet Pharma and Harmony Biosciences, LLC.

0745

THE PHARMACOKINETICS OF FT218, ONCE NIGHTLY SODIUM OXYBATE: FOOD EFFECT AND BIOAVAILABILITY COMPARED TO TWICE NIGHTLY SODIUM OXYBATE

*Thorpy, M.*¹ *Seiden, D.*² *Grassot, J.*² *Monteith, D.*² *Dubow, J.*² *Corser, B.*³

¹Albert Einstein College of Medicine, New York, NY, ²Avadel Pharmaceuticals, Chesterfield, MO, ³Sleep Management Institute, Cincinnati, OH.

Introduction: Sodium oxybate is an effective treatment for excessive daytime sleepiness and cataplexy in patients with narcolepsy. The FDA approved formulation requires twice-nightly dosing; at bedtime and 2.5 - 4 hours later. FT218 is a controlled-release formulation of sodium oxybate intended for once-nightly dosing, using Avadel's proprietary Micropump[™] technology. The objective of this study was to evaluate the relative bioavailability of investigational once-nightly sodium oxybate, FT218, 6 g, compared to commercially available twice-nightly sodium oxybate and the food effect of FT218.

Methods: Two crossover, single-dose pharmacokinetic studies were conducted in healthy volunteers. The first, a relative bio-availability study (n=28) was completed comparing FT218 6 g to twice-nightly sodium oxybate 6 g (in two divided doses of 3 g). The second, evaluated the food effect (n=16) of FT218 6g in the Fed vs. Fasted state.

Results: FT218 had a lower overall C_{max} than twice-nightly sodium oxybate, while AUC was equivalent. C_{8h} level and variability was comparable between FT218 and twice-nightly sodium oxybate. In the Fed, compared to the Fasted state, FT218 had a longer T_{max} , lower C_{max} and decreased AUC (C_{max} 67%, AUC 86%, T_{max} 1-hour slower than Fasted values). Adverse Events with FT218 were mostly mild or moderate in severity, non-serious and known AEs associated with sodium oxybate. The safety profiles of FT218 and twice-nightly sodium oxybate at 6 g appeared similar.

Conclusion: Once-nightly FT218 at 6 g demonstrated a lower overall C_{max} and similar exposure to twice-nightly sodium oxybate, with similar C_{8h} plasma levels and C_{8h} variability. In the Fed state, AUC and Cmax of FT218 was lower than in the Fasted State. FT218 was generally safe and well tolerated and the safety profile appeared comparable to twice-nightly sodium oxybate.

Support: This work was supported by Avadel Pharmaceuticals.

0746

DYNAMICS OF SLEEP STAGE TRANSITIONS IN PATIENTS WITH NARCOLEPSY AND OTHER HYPERSOMNIAS

Kishi, A.¹ Kitajima, T.² Kawai, R.² Hirose, M.² Iwata, N.² Yamamoto, Y.¹ ¹The University of Tokyo, Tokyo, JAPAN, ²Fujita Health University School of Medicine, Aichi, JAPAN.

Introduction: Narcolepsy is a chronic sleep disorder characterized by excessive daytime sleepiness and abnormal REM sleep phenomena. Narcolepsy can be distinguished into type 1 (NT1; with cataplexy) and type 2 (NT2; without cataplexy). It has been reported that sleep stage sequences at sleep-onset as well as sleepwake dynamics across the night may be useful in the differential diagnosis of hypersomnia. Here we studied dynamic features of sleep stage transitions during whole night sleep in patients with NT1, NT2, and other types of hypersomnia (o-HS).

Methods: Twenty patients with NT1, 14 patients with NT2, and 35 patients with o-HS underwent overnight PSG. Transition probabilities between sleep stages (wake, N1, N2, N3, and REM) and survival curves of continuous runs of each sleep stage were compared between groups. Transition-specific survival curves of continuous runs of each sleep stage, dependent on the subsequent stage of the transition, were also compared.

Results: The probability of transitions from N1-to-wake was significantly greater in NT1 than in NT2 and o-HS while that from N1-to-N2 was significantly smaller in NT1 than in NT2 and o-HS. The probability of transitions from N2-to-REM was significantly smaller in NT1 than in o-HS. Wake and N1 were significantly more continuous in NT1 than in NT2; specifically, N1 followed by N2 was significantly more continuous in NT1 than in NT2 and o-HS. N2 was significantly less continuous in NT1 than in o-HS; this was specifically confirmed for N2 followed by N1/ wake. REM sleep was significantly less continuous in NT1 than in NT2 and o-HS; specifically, REM sleep followed by wake was significantly less continuous in NT1 than in NT2 and o-HS; specifically, REM sleep followed by wake was significantly less continuous in NT1 than in o-HS. Continuity of N3 did not differ significantly between groups.

Conclusion: Dynamics of sleep stage transitions differed between NT1, NT2, and o-HS. Dynamic features of sleep such as sleep instability, persistency of wake/N1, and REM fragmentation may differentiate NT1 from NT2, while N2 continuity may differentiate narcolepsy from o-HS. The results suggest that sleep transition analysis may be of clinical utility and provide insights into the underlying pathophysiology of hypersomnia and narcolepsy. **Support:** JSPS KAKENHI (18K17891 to AK).

0747

STUDY DESIGN OF AN OPEN-LABEL EXTENSION AND SWITCH STUDY FOR ONCE NIGHTLY SODIUM OXYBATE, FT218, IN NT1 AND NT2 PATIENTS

Wells, C. Dubow, J. Fenton, M. Patel, D. Tyler, C. Seiden, D. Avadel Pharmaceuticals, Chesterfield, MO.

Introduction: Avadel Pharmaceuticals has developed FT218, a once nightly sodium oxybate (SO) formulation for the treatment of excessive daytime sleepiness (EDS) and cataplexy in narcoleptic patients. REST-ON, a pivotal efficacy/safety study is expected to be completed in the first half of 2020. Previous Phase 1 studies have demonstrated FT218 delivers a pharmacokinetic profile needed for once-nightly dosing. The purpose of this study is to evaluate long-term safety of FT218 in REST-ON completers and dosing strategy when switching from twice-nightly SO to FT218.

Methods: The study will enroll NT1/NT2 patients who completed REST-ON or patients switching from stable dose of twice-nightly SO to FT218. REST-ON completers will initiate FT218 at 4.5 g and increase by 1.5 g to the highest tolerated dose or the dose deemed effective (up to 9 g). Switch patients will initiate FT218 at the equivalent/closest dose to their current twice-nightly SO and

titrate in accord with safety/efficacy. The study will enroll approximately 250 patients for a duration of two years.

Results: The primary endpoint for REST-ON completers will be safety and tolerability. Secondary endpoints for REST-ON completers will include changes in ESS, reported cataplexy and other REM associated phenomena as well as changes in clinician and patient global impression. For switch patients, endpoints will include percentage of subjects that stay on the same, higher or lower dose of FT218 compared to twice-nightly SO, as well as the percentage of subjects who report a preference for the once-nightly dosing regimen.

Conclusion: The results of this open label extension/switch study will further elucidate the potential benefits of once-nightly FT218 regarding long term safety/tolerability, nocturnal safety/tolerability and efficacy, and importantly provide dosing and preference data for patients switching from twice-nightly SO to once-nightly FT218.

Support: This work was supported by Avadel Pharmaceuticals.

0748

A NOVEL, NON-PHARMACOLOGICAL TREATMENT OPTION FOR PATIENTS WITH IDIOPATHIC HYPERSOMNIA

Paech, G. M.^{1,2} Pradeepan, S.^{1,2} Suthers, B.^{1,2}

¹Department of Respiratory and Sleep Medicine, John Hunter Hospital, Newcastle, AUSTRALIA, ²School of Medicine and Public Health, University of Newcastle, Newcastle, AUSTRALIA.

Introduction: There is some evidence suggesting that patients with hypersomnia have delayed circadian timing, which could explain, at least in part, the excessive sleepiness and impaired daytime performance experienced by these individuals. This study investigated the effects of bright light treatment on improving daytime alertness in patients with idiopathic hypersomnia.

Methods: Participants were scheduled to two in-laboratory sessions (baseline and treatment) consisting of overnight sleep monitoring (polysomnography) followed by maintenance of wake-fulness tests (MWT) and performance testing (10-min psycho-motor vigilance task (PVT)). MWTs were performed at 10:00, 12:00, 14:00 and 16:00 and PVTs were performed at 11:00, 13:00, 15:00 and 15:00. In-laboratory sessions were separated by a two-week at-home treatment period during which participants were instructed to use commercially available light devices for 30-60 min each morning. Participants also completed the Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale (ESS) at baseline and after treatment. Paired t-tests were performed to assess differences in sleep architecture, sleep quality, performance (lapses; reaction time >500ms) and alertness (MWT mean sleep latency) between baseline and treatment.

Results: To date, three participants (2 male) aged 62.7 ± 13.2 (mean±SD) years have completed the study. Lapses (baseline: 17.9±11.9; treatment: 6.3 ± 5.4), ESS (baseline: 17.3±3.1; treatment 12.3±6.8) and PSQI (baseline: 7.7 ± 3.8 ; treatment: 4.0 ± 1.0) all improved with light treatment, although this did not reach statistical significance. There were no statistical differences between baseline and treatment with regards to sleep architecture or mean sleep latency.

Conclusion: Although preliminary, results suggest that bright light treatment may improve subjective sleepiness, subjective sleep quality and performance. There was some variability between individuals, indicating that this treatment may not offer the same

benefits to all patients. Although ongoing, this study suggests that light therapy could be used as an alternative, non-pharmacological treatment option to improve waking functions and sleepiness in hypersomnia patients.

Support: This project is supported by a Hunter Medical Research Institute Research Project Grant

0749

CASE SIMULATIONS IN OBSTRUCTIVE SLEEP APNEA AND NARCOLEPSY: PATIENTS THAT KEEP YOU UP AT NIGHT

Bogan, R. K.¹ Rorie, K.² Faler, W. E.² Gross, T.² Perez, J.² Tordoff, S. A.² Salinas, G.³

¹Bogan Sleep Consultants, LLC, Charleston, SC, ²CME Outfitters, Bethesda, MD, ³CE Outcomes, Birmingham, AL.

Introduction: 3 CME interventions used online case simulations to facilitate the recognition of residual EDS in patients with narcolepsy and OSA and to improve decision-making when developing strategies for long-term management of residual EDS.

Methods: Outcomes data were obtained from 3, 30-minute case simulations on residual EDS. Surveys assessing knowledge, confidence, and behavior were administered pre- and post-activity. A separate evaluation provided demographics and other variables used in the model. Data from a 2-month follow-up survey were analyzed to determine performance effects on the learner population (n = 30) compared to matched controls (n = 30). Statistical comparisons of data from baseline to post-intervention were made using McNemar's tests and paired t-tests. Additionally, predictive modeling was applied to evaluate variables predictive of evidence-based decisions. A longitudinal analysis of results was conducted to evaluate knowledge and performance changes in sleep-related initiatives between 2016 - 2019.

Results: Learners outperformed controls in utilizing the Epworth Sleepiness Scale (ESS) and are more likely to interpret ESS scores to confirm EDS diagnosis and select appropriate treatment options in managing patients with EDS. Further, learners are more likely than controls to engage in shared decision-making with patients. When given a real-world case, learners are more likely than controls to identify symptoms and order correct tests. Learners were also more likely to select best treatment options for the patient more often than non-learners. Continued education needs to focus on treatment options for patients with narcolepsy.

Conclusion: Follow-up assessments were conducted to understand lasting performance in learners attributable to this education. Using data from learners compared to matched controls, we found the education had an effect size of 23% (Cohen's d = 0.33). This indicates that for every 100 clinicians exposed to this education, 23 will perform more according to evidence than if they were not exposed.

Support: The educational activity described in this abstract was supported by an educational grant from Jazz Pharmaceuticals, Inc.

0750

NOCTURNAL SLEEP STABILITY AND CEREBROSPINAL FLUID OREXIN-A LEVELS: SLEEP AND WAKE BOUTS

Barateau, L.¹ Lopez, R.¹ Chenini, S.¹ Rassu, A.¹ Scholz, S.¹ Lotierzo, M.³ Cristol, J.³ Jaussent, I.⁴ Dauvilliers, Y.¹ ¹Sleep Disorder Center, Gui de Chauliac Hospital, Montpellier, FRANCE, ²Sleep Disorder Center, Gui de Chauliac Hospital, Montpellier, FRANCE, ³Department of Biochemistry, Montpellier University Hospital, Montpellier, Montpellier, FRANCE, ⁴INSERM U1061, Montpellier, FRANCE. **Introduction:** The orexin (ORX)/hypocretin system stabilizes sleepwake regulation by sustaining long periods of wakefulness in humans and animals. We aimed to evaluate the relationships between cerebrospinal fluid (CSF) ORX levels and markers of nocturnal sleep stability assessed by polysomnography (PSG) in humans.

Methods: Nocturnal PSG data and CSF ORX levels of 300 drug-free subjects (55% men, 29.9 \pm 15.5 years old, mean ORX levels 155.1 \pm 153.7 pg/mL) with a complaint of hypersomnolence were collected in the National Reference Center for Narcolepsy, France. Several markers of nocturnal sleep stability were analyzed: wake (WB), sleep bouts (SB), and sleep/wake transitions. Groups were categorized according to ORX levels: two categories (\leq 110, >110 pg/mL, the current established threshold of ORX-deficiency), and tertiles (\leq 26,]26;254], >254 pg/mL); and were compared using logistic regression models. Results were adjusted for age, gender and body mass index.

Results: ORX-deficient subjects had more WB, SB, and sleepwake transitions than the others. The WB duration was longer and the SB duration shorter in ORX-deficient category. The proportion of the shortest WB (30 sec) was lower in the ORX-deficient category whereas the proportion of WB above 1 min 30 sec was higher. The proportion of SB \leq 14min was higher among ORXdeficient patients, with opposite results for longer SB. Subsequent analyses performed in the population categorized according to tertiles of CSF ORX-A confirmed all these findings, with a strong dose-response effect of ORX levels in post-hoc comparisons. All results remained highly significant in adjusted statistical models.

Conclusion: This study provides a strong evidence of the direct effect of ORX on nocturnal sleep stabilization in humans. WB and SB are reliable markers of nighttime sleep stability, strongly correlated to CSF ORX-A levels in a dose dependent way. These PSG biomarkers are promising to be applied in clinical and research settings.

Support: none

0751

EPWORTH SLEEPINESS SCALE TEST-RETEST RELIABILITY ANALYSIS IN SOLRIAMFETOL STUDIES

Rosenberg, R.¹ Babson, K.² Menno, D.² Morris, S.² Baladi, M.² Hyman, D.² Black, J.³

¹NeuroTrials Research, Inc., Atlanta, GA, ²Jazz Pharmaceuticals, Palo Alto, CA, ³Stanford Center for Sleep Sciences and Medicine, Palo Alto, CA.

Introduction: The Epworth Sleepiness Scale (ESS) measures excessive daytime sleepiness. This analysis examined test-retest reliability of ESS scores in participants with narcolepsy or obstructive sleep apnea (OSA) in solriamfetol studies.

Methods: Intraclass correlation coefficient (ICC) estimates and 95% confidence intervals (CIs) for ESS scores from two 12-week, placebo-controlled trials (1 narcolepsy; 1 OSA), and one long-term open-label extension (OLE) trial (narcolepsy or OSA) were calculated separately for each trial, based on assessments (at time-point pairs) when scores were expected to be stable (at weeks 4 and 8, 8 and 12, and 4 and 12 in the 12-week trials, and weeks 14 and 26/27, 26/27 and 39/40, and 14 and 39/40 in the OLE). ICCs were analyzed for the overall population in each trial and by treatment and adherence to primary OSA therapy. An ICC >0.7 has been recommended as a quality criterion for acceptable test-retest reliability.

Results: In the 12-week narcolepsy trial, ICCs (95% CI) were 0.83 (0.79, 0.87) for weeks 4 and 8 (n=199), 0.87 (0.83, 0.90) for weeks 8 and 12 (n=196), and 0.81 (0.76, 0.85) for weeks 4 and 12 (n=196).

In the 12-week OSA trial, ICCs (95% CI) were 0.74 (0.69, 0.78) for weeks 4 and 8 (n=416), 0.80 (0.76, 0.83) for weeks 8 and 12 (n=405), and 0.74 (0.69, 0.78) for weeks 4 and 12 (n=405). In the OLE trial, ICCs (95% CI) were 0.82 (0.79, 0.85) for weeks 14 and 27/26 (n=495), 0.85 (0.82, 0.87) for weeks 27/26 and 40/39 (n=463), and 0.78 (0.74, 0.81) for weeks 14 and 40/39 (n=463). Treatment (solriamfetol combined/placebo) or adherence to primary OSA therapy did not impact reliability.

Conclusion: In 3 large clinical trials of participants with narcolepsy or OSA, the ESS demonstrated an acceptable level of testretest reliability.

Support: Jazz Pharmaceuticals

0752

JZP-258 DOSE TITRATION AND TRANSITION FROM SODIUM OXYBATE IN A PLACEBO-CONTROLLED, DOUBLE-BLIND, RANDOMIZED WITHDRAWAL STUDY IN ADULT PARTICIPANTS WITH NARCOLEPSY WITH CATAPLEXY

Foldvary-Schaefer, N.¹ Bogan, R. K.² Thorpy, M. J.³ Huang, L.⁴ Skowronski, R.⁴ Dauvilliers, Y.⁵

¹Cleveland Clinic, Cleveland, OH, ²University of South Carolina School of Medicine, Columbia, SC, ³Albert Einstein College of Medicine, New York, NY, ⁴Jazz Pharmaceuticals, Inc., Palo Alto, CA, ⁵Sleep and Wake Disorders Centre, Department of Neurology, Gui de Chauliac Hospital, Montpellier, FRANCE.

Introduction: Sodium oxybate (SXB) is a standard of care for the treatment of cataplexy and excessive daytime sleepiness in narco-lepsy. JZP-258 is an oxybate product candidate (at same concentration as SXB) with 92% less sodium. JZP-258 dose adjustment during titration was evaluated.

Methods: At study entry, participants were taking SXB only, SXB+other anticataplectics, anticataplectics other than SXB, or were cataplexy treatment-naive. JZP-258 treatment began during a 12-week, open-label optimized treatment and titration period. Participants taking SXB only or SXB+other anticataplectics transitioned to JZP-258 at the same gramfor-gram dose as SXB and titrated to an efficacious and tolerable (optimal) dose from weeks 3-12. Participants taking other anticataplectics or who were anticataplectic-naive initiated JZP-258 at 4.5 g/night and were titrated to an optimal dose at 1-1.5 g/night/week (maximum total dose, 9 g/night). A 2-week stable-dose period and 2-week, double-blind, randomized withdrawal period followed.

Results: During the stable-dose period, total nightly JZP-258 dose (median [range]) was higher in participants taking SXB at study entry (SXB-only, 7.5 g [4.5-9.0], n=45; SXB+other anticataplectics, 9.0 g [6.0-9.0], n=14) compared with those not taking SXB (other anticataplectics, 7.5 g [4.5-9.0], n=23; anticataplectic-naive, 7.0 g [3.0-9.0], n=67), and dose adjustments were fewer. In most (69%) participants taking SXB at study entry who entered the stable-dose period, no change in dose was required (median [range] number of adjustments was 0 ([0-8]); for those with a change in dose, most changes were within one titration step (1.5 g/night). In participants not taking SXB at study entry, the median (range) number of adjustments was 3.0 (0-7).

Conclusion: Most participants taking SXB at study entry transitioned to JZP-258 treatment at the same dose with retained effectiveness. Participants not previously taking SXB achieved a tolerable and efficacious dose of JZP-258 after a median of 3 adjustments. **Support:** Jazz Pharmaceuticals

0753

CATAPLEXY-FREE DAYS IN A PHASE 3, PLACEBO-CONTROLLED, DOUBLE-BLIND, RANDOMIZED WITHDRAWAL STUDY OF JZP-258 IN ADULTS WITH NARCOLEPSY WITH CATAPLEXY

Dauvilliers, Y.¹ Foldvary-Schaefer, N.² Bogan, R. K.³ Šonka, K.⁴ Profant, J.⁵ Huang, L.⁵ Thorpy, M. J.⁶

¹Sleep and Wake Disorders Centre, Department of Neurology, Montpellier, FRANCE, ²Cleveland Clinic Lerner College of Medicine, Cleveland, OH, ³University of South Carolina School of Medicine, Columbia, SC, ⁴First Faculty of Medicine, Charles University and General University Hospital, Prague, CZECH REPUBLIC, ⁵Jazz Pharmaceuticals, Inc., Palo Alto, CA, ⁶Albert Einstein College of Medicine, New York, NY.

Introduction: Sodium oxybate (SXB) is a standard of care for the treatment of cataplexy and excessive daytime sleepiness in narcolepsy. JZP-258 is an oxybate product candidate with 92% less sodium. This analysis evaluated cataplexy-free days/week, as a measure of treatment impact, in a placebo-controlled randomized withdrawal study of JZP-258 treatment in patients with narcolepsy. Methods: Treatment for cataplexy at study entry included 1) SXB (SXB-only); 2) SXB plus other anticataplectics (SXB+other); 3) anticataplectics other than SXB (other anticataplectics); or 4) cataplexy treatment-naive (anticataplectic-naive). Participants (aged 18-70 years with narcolepsy with cataplexy) began JZP-258 treatment during a 12-week, open-label, optimized treatment and titration period (OLOTTP), followed by a 2-week stable-dose period (SDP). Participants were randomized to receive placebo or continue JZP-258 treatment during a 2-week, double-blind, randomized withdrawal period (DBRWP).

Results: Of 201 enrolled participants, 134 comprised the efficacy population (placebo, n=65; JZP-258, n=69). Median (Q1, Q3) cataplexy-free days/week at first week of OLOTTP (while initiating JZP-258) by prior treatment were SXB-only, 5.8 (2.0, 7.0); SXB+other, 6.4 (5.0, 7.0); other anticataplectics, 4.0 (1.8, 6.0); anticataplectic-naive, 3.5 (0, 5.8). At end of SDP (on stable dose of JZP-258), median (Q1, Q3) cataplexy-free days/week were 6.0 (3.5, 7.0), 6.1 (1.4, 7.0), 6.0 (2.6, 7.0), and 6.2 (4.0, 7.0), respectively. Prior to randomization, there was no difference in median cataplexy-free days/week between participants to be randomized to placebo (6.0 [3.5, 7.0]) or JZP-258 treatment (6.0 [3.0, 7.0]); during DBRWP, median cataplexy-free days/week decreased in participants randomized to placebo (3.5 [0, 5.83]) but remained similar in participants randomized to continue JZP-258 treatment (5.6 [2.8, 7.0]). The overall safety profile of JZP-258 was similar to SXB.

Conclusion: Number of cataplexy-free days/week increased with JZP-258 treatment in participants previously naive to oxybate. Number of cataplexy-free days/week decreased during placebo exposure in participants randomized to placebo.

Support: Jazz Pharmaceuticals

0754

COGNITIVE BEHAVIORAL THERAPY FOR HYPERSOMNIA (CBT-H): A FEASIBILITY STUDY FOR IMPROVING HEALTH-RELATED QUALITY OF LIFE

Ong, J. C. Dawson, S. C. Mundt, J. M. Adkins, E. Moore, C. Northwestern University, Chicago, IL.

Introduction: The purpose of this study was to conduct a feasibility trial for a novel cognitive behavioral therapy (CBT-H) aimed at improving health-related quality of life (HRQoL) in people with hypersomnia.

Methods: Participants were 35 adults (32 female, mean age=32.0 years, SD=12.9) with an established diagnosis of Narcolepsy Type 1 (n=12), Type 2 (n=11), or Idiopathic Hypersomnia (n=12). Participants were assigned to individual (n=19) or group (n=16, 3-5 per group) format of a 6-session, manualized CBT-H, delivered using live videoconferencing. Key components of CBT-H included structuring daytime behaviors (e.g., planned naps), emotion regulation techniques, and energy management strategies. Outcome measures for HRQoL included PROMIS measures for depression, anxiety, self-efficacy, and social isolation. Other clinical outcome measures included the Patient Health Questionnaire (PHQ) and Epworth Sleepiness Scale (ESS). Exit interviews were used to collect qualitative data to inform acceptability of the intervention.

Results: Intent-to-treat analyses were conducted on the entire sample with the last observation carried forward for 3 participants who did not provide post-treatment data. Paired-samples t-test revealed a significant reduction on PROMIS depression (t[34]=2.05, p=0.0486, d=-0.35), and significant increases on PROMIS general self-efficacy (t[34]=3.64, p=0.0009, d=0.62) and self-efficacy managing social interactions (t[34]=2.14, p=0.0396, d=0.36). Significant reductions were also observed on the ESS (t[34]=2.07, p=0.0458, d=-0.35) and PHQ (t[34]=4.42, p<.0001, d=-0.75). Mixed-design ANOVAs revealed no significant differences on hypersomnia diagnosis or treatment format. Qualitative data supported the acceptability of telehealth delivery with mixed opinions regarding the format and number of sessions.

Conclusion: These findings support the acceptability of a novel CBT-H delivered using a telehealth model and the feasibility of reducing excessive sleepiness and improving HRQoL, particularly in the domains of self-efficacy and depression, in people with narcolepsy and idiopathic hypersomnia.

Support: This study was supported by grant 185-SR-17 from the American Sleep Medicine Foundation.

0755

PITOLISANT (WAKIX) IS AN EFFECTIVE ANTI-CATAPLEXY AGENT IN NARCOLEPSY TYPE 1

Meskill, G. J.

Comprehensive Sleep Medicine Associates, Houston, TX.

Introduction: Harmony Biosciences initiated the Pitolisant Expanded Access Clinical Evaluation (PEACE) program to allow treatment with pitolisant in adult patients with narcolepsy while pitolisant was an investigational medication in the United States. Starting in March 2019, Comprehensive Sleep Medicine Associates (CSMA) offered enrollment to patients who met the inclusion/exclusion criteria and who were deemed appropriate based on clinical judgment. All patients who enrolled were taking at least one standard-of-care agent for narcolepsy at enrollment. Many of the enrolled patients had refractory/challenging cases of narcolepsy. On August 14, 2019, Wakix received FDA approval for the treatment of excessive daytime sleepiness in adult patients with narcolepsy, which contrasts with the European label that states that Wakix is indicated for the treatment of narcolepsy in adults with or without cataplexy.

Methods: CSMA enrolled 21 patients in the PEACE program. The charts for all 10 narcolepsy type 1 (NT1) patients were reviewed. The 2 patients who did not have follow up after starting pitolisant were excluded.

Results: Of the 8 NT1 patients who had at least one follow up visit after initiating pitolisant, 6 reported substantial improvement or complete resolution of cataplexy compared to baseline. For example, one patient's wife stated, "I forgot my husband was funny because he would avoid telling jokes until he started pitolisant." Another stated, "I have not had an episode of cataplexy since starting pitolisant." 5 of these patients were taking an anticataplectic agent at the time of starting pitolisant (sodium oxybate 3, venlafaxine 2).

Conclusion: While a relatively small sample size, these results demonstrate that in a "real world" uncontrolled population of refractory/challenging NT1 patients, pitolisant is an effective anti-cataplectic agent. As there are relatively few treatment options for NT1, clinicians should consider use of pitolisant for patients with cataplexy, and further consideration for adding an indication for pitolisant to treat cataplexy is warranted.

Support: Harmony Biosciences (PEACE trial)

0756

A ONE YEAR OBSERVATIONAL EARLY ACCESS PITOLISANT STUDY OF EXCESSIVE DAYTIME SLEEPINESS IN NARCOLEPSY

Stultz, D. J. Osburn, S. Burns, T. Stanley, N. Walton, R. Pawlowska-Wajswol, S. J. Moomaw, S. Stultz Sleep & Behavioral Health, Barboursville, WV.

Introduction: Pitolisant is a H3 receptor antagonist/inverse agonist that has been FDA approved for excessive daytime sleepiness in narcolepsy at doses of either 17.8 mg or 35.6 mg per day.

Methods: 13 patients (3 males and 10 females) were studied having an average age of 46.8 years, with the majority receiving a dose of 35.6 mg Pitolisant. One patient received 17.8 mg throughout the year, and another advanced after 6 months to the 35.6 mg dose due to hepatic issues. 12 of the patients were Caucasian and one was Asian. 100% of the patients had co-existing sleep and psychiatric disorders. 46% had co-existing sleep apnea and were on CPAP/BIPAP. 38.5% had a history of a head injury. 84.6% of the patients had associated cataplexy, 38% had sleep paralysis, 92% had disrupted nocturnal sleep, and 46% had hypnogogic hallucinations. Throughout the year the patients were monitored using the Epworth Sleepiness Scale (ESS). Nine patients completed the 12-month ESS scales. 12/13 were on other medications to treat narcolepsy prior to starting Pitolisant. 6/13 were on sodium oxybate, 7/13 were on an antidepressant, and 11/13 were on either a stimulant, modafanil, or armodafanil. Only one patient was on Pitolisant alone.

Results: The patient's average ESS score at onset was 16.2 Statistically significant findings using paired t-tests were documented. After one-month ESS scores decreased to an average of 13.2 (t=2.38, 9df, P=.04). At 3 months it was 12.4 (t=2.81, 10df, P=.02), at 6 months it was 12.75 (t=4.69, 11df, P<.001) and at 12 months the average score was 13.11 (t=2.55, 8df, P=.03) documenting clinically meaningful decrease of ESS by >/= 3 points. Three patients had ESS scores </=10 at 12 months.

Conclusion: Improvement on ESS was documented at one month and sustained for one year in patients diagnosed with having nar-colepsy both with and without cataplexy.

Support: **No support was given for this study. Dr. Stultz is a speaker for Harmony Biosciences and has served on their advisory committee. She is also a speaker for Jazz Pharmaceuticals.

INCREASING NUMBER OF CASES WHO HAD BOTH HYPERSOMNOLENCE DISORDERS AND DEVELOPMENTAL DISORDERS WITH OREXIN MEASUREMENTS

Imanishi, A.¹ Yoshizawa, K.¹ Tsutsui, K.¹ Omori, Y.² Ono, T.³ Ito Uemura, S.⁴ Mishima, K.¹ Kondo, H.⁵ Kanbayashi, T.⁵ ¹Akita University School of Medicine, Akita, JAPAN, ²Tokyo Metropolitan Geriatric Hospital, Tokyo, JAPAN, ³Sleep & Circadian Neurobiology Laboratory, Stanford University,, California, CA, ⁴Akita University Graduate School of Health Sciences, Akita, JAPAN, ⁵International Institute for Integrative Sleep Medicine (IIIS), Tsukuba University,, Tsukuba, JAPAN.

Introduction: Recently, attention has been paid to the relationship between developmental disorders such as attention deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD), and sleep disorders. We meet many developmental disorder patients who complaint hypersomnolence. Among these patients, cases with coexistence of central hypersomnia and developmental disorders, or developmental disorder alone were increased. Therefore, we first investigated patients with the complaint of hypersomnolence, who were also suspected developmental disorders. Furthermore, we have been measuring CSF orexin in 17 cases suspected of both disorders to investigate orexin levels of these patients.

Methods: 86patients who complained of EDS with suspicion of developmental disorders had been examined. In order to diagnose hypersomnolence disorders, PSG and MSLT were performed. Psychological examinations were performed for diagnosing developmental disorders. We have been measuring for CSF orexin in 17 cases suspected both hypersomnolence and developmental disorders. We examined the onset of hypersomnolence and the clinical history of these ADHD or ASD cases for more details.

Results: In 86 examined cases, developmental disorders coexisted in 30 cases. Among 30 cases, ADHD were 18, ASD were 6 and both diagnosed were 6 cases. Among them, 20 cases diagnosed as having coexistence of hypersomnia (8: narcolepsy, 12: IHS) and developmental disorders (ADHD:12, ASD:4, ADHD/ASD:4). In 17 cases with orexin measurements, 10 cases coexisted ADHD and 4 cases coexisted ASD. Two cases diagnosed as both ADHD and ASD. In 10 ADHD cases, 3 cases had low orexin levels, and 7 cases had normal orexin levels. Other 7 ASD cases had normal orexin levels. Conclusion: ADHD has a higher rate of central hypersomnia (12/18) compared with ASD and the rate of narcolepsy was also high (5/12). While patients in ASD was diagnosed as IHS (3/6), narcolepsy cases were not observed. It became clear that the majority of patients had developmental disorder or had a tendency for developmental disorder before the onset of hypersomnolence. Although it is possible that ADHD/ASD symptoms may be exacerbated by orexin dysfunctions, ADHD/ASD may not newly occur. There were cases with low orexin levels, but it seems that narcolepsy happened to coexist with developmental disorders. Support: a

0758

QUANTIFICATION OF LATE REM PERIODS IN PATIENTS WITH PROLONGED SLEEP DURATION

Blattner, M. S. August, J. Chopra, S. Dalal, L. Luthra, S. Cunningham, L. Dunham, K. Thomas, R. J. Sleep Medicine; Beth Israel Deaconess Medical Center, Boston, MA. **Introduction:** Evaluation of hypersomnia includes polysomnography followed by mean sleep latency testing (MSLT). As consistent with guidelines as applied in most centers, the overnight portion of the study will be terminated to begin sleep latency testing. For patients with prolonged sleep duration, this interruption could result in REM sleep on nap testing that reflects continuation of their biological night, rather than abnormalities in REM sleep pressure/regulation.

Methods: We reviewed 42 consecutive extended (unrestricted) sleep studies for patients with a total sleep time greater than 600 minutes. For studies with sleep onset before midnight, we evaluated for REM period onset after 6AM, the number of REM periods after 6AM and 8AM, and the time of the final REM period onset. **Results:** 42 hypnograms were reviewed for patients undergoing evaluation of hypersomnia, median age 32 years (range 19-92) with a median total sleep time of 663 minutes (range 602-832), of these 28/42 (67%) had sleep onset before midnight (12 AM) and were included in the analysis. 27/28 (96%) of hypnograms reviewed had REM sleep after 6 AM, 24/28 (86%) had REM sleep after 8 AM, with the onset of the final REM period ranging from 4:46 AM-12:30 PM for patients with sleep onset time before midnight (12 AM).

Conclusion: These data suggest that termination of overnight polysomnography to complete mean sleep latency testing, as is standard in most sleep labs, may influence the presence of REM sleep on MSLT for patients with prolonged total sleep duration. These results may have implications for the interpretation of MSLT for patients with long sleep duration, and may explain why a given individual may test as type II narcolepsy or idiopathic hypersomnia unpredictably on repeat testing.

Support: Sleep Medicine Fellowship at BIDMC

0759

PHASE 2 PROOF OF CONCEPT STUDY OF SUVN-G3031, A HISTAMINE H3 RECEPTOR INVERSE AGONIST FOR THE POTENTIAL TREATMENT OF NARCOLEPSY

Nirogi, R. Goyal, V. Jayarajan, P. Bhyrapuneni, G. Ravula, J. Jetta, S. Shinde, A.

Suven Life Sciences, Hyderabad, INDIA.

Introduction: SUVN-G3031 is a potent inverse agonist at histamine H3 receptor (H3R) with selectivity over 70 other targets. SUVN-G3031 has excellent pharmacokinetics in rats and dogs. SUVN-G3031 demonstrated dose dependent receptor occupancy in rats with marked wake-promoting and anticataplectic effects in orexin knockout mice supporting its potential therapeutic utility in the treatment of narcolepsy. Long-term safety studies in animals and Phase 1 evaluation for safety, tolerability and pharmacokinetics demonstrated no concern for further development of SUVN-G3031.

Methods: SUVN-G3031 is currently being evaluated in a Phase 2 proof of concept study in USA for the treatment of narcolepsy with and without cataplexy (ClinicalTrials.gov Identifier: NCT04072380). This is a double-blind, placebo-controlled, parallel-group, multicenter study to evaluate the safety, tolerability, pharmacokinetics and efficacy of SUVN-G3031 in comparison to placebo in patients with narcolepsy with and without cataplexy. Participants with an ESS score of \geq 12; and mean MWT time of < 12 min are being randomized at a ratio of 1:1:1 to 2 mg SUVN-G3031, 4 mg SUVN-G3031 or placebo. Patients are to be stratified based on the type of narcolepsy. Each patient will receive study drug once daily for 14 days. The primary objective of the study is to evaluate the effectiveness of SUVN-G3031 compared to placebo as

measured by an improvement in the maintenance of wakefulness test (MWT) score. Various secondary, exploratory, safety endpoints and steady state plasma concentrations will be evaluated.

Results: This study has been initiated in Q3 2019 and subject recruitment is expected to be completed by Q2 2020.

Conclusion: This study is a phase 2 clinical trial evaluating the efficacy and safety of SUVN-G3031 as a monotherapy in patients with narcolepsy with and without cataplexy. Safety and efficacy results from the study are expected in Q3 2020.

Support: None

0760

SUVN-G3031, A POTENT AND SELECTIVE HISTAMINE H3 RECEPTOR INVERSE AGONIST: SAFETY, TOLERABILITY AND PHARMACOKINETICS FOLLOWING SINGLE AND MULTIPLE ASCENDING DOSES IN HEALTHY ADULT SUBJECTS

Bhyrapuneni, G. Goyal, V. Pandey, S. Muddana, N. Palacharla, R. Ajjala, D. Ravula, J. Jetta, S. Badange, R. Benade, V. Nirogi, R. Suven Life Sciences, Hyderabad, INDIA.

Introduction: SUVN-G3031 is a potent and selective histamine H3 receptor inverse agonist currently being developed for the treatment of narcolepsy. SUVN-G3031 produced robust wake promoting and anticataplectic effects in animal model relevant to the disease. This supports its therapeutic utility in the treatment of sleep related disorders like narcolepsy with and without cataplexy. **Methods:** Two Phase 1 studies were conducted to assess safety, tolerability and pharmacokinetics (PK) of SUVN-G3031. In the first study, single ascending doses of 0.1 mg to 20 mg SUVN-G3031 were administered to healthy subjects. For multiple ascending dose cohorts, doses of 1 mg to 6 mg were administered for 14 days. In the second Phase 1 study, effects of food, gender and age on the PK of SUVN-G3031 were assessed.

Results: SUVN-G3031 absorbed rapidly following single oral administration and the exposures (C_{max} and AUC) were dose proportional at the tested doses between 0.1 mg to 20 mg. SUVN-G3031 attained steady state on day six and achieved projected efficacy concentrations following repeated administrations. Food, gender and age had no effect on pharmacokinetics of SUVN-G3031. SUVN-G3031 was well tolerated up to 20 mg/ day single dose and 6 mg repeated dose in healthy adult subjects. There were no serious adverse events reported by any subject during Phase 1 studies.

Conclusion: SUVN-G3031 was well tolerated in humans with adequate plasma exposures for efficacy and has favorable pharmacokinetics suitable for once a day oral administration. SUVN-G3031 is currently being evaluated in a Phase 2 study as monotherapy for the treatment of narcolepsy with and without cataplexy (ClinicalTrials.gov Identifier: NCT04072380). **Support:** None

0761

INDEPENDENT COMPONENTS ANALYSIS AND GRAPH THEORETICAL ANALYSIS IN PATIENTS WITH NARCOLEPSY

Xiao, F.¹ Zhao, L.¹ Han, F.²

¹Peking University People's Hospital, Beijing, CHINA, ²Peking University People's Hospital, Beijing, CHINA.

Introduction: To evaluate resting state functional connectivity and topological properties of brain network in narcolepsy compared with healthy controls.

Methods: Resting state fMRI was performed in 26 adult narcolepsy patients and 30 matched healthy controls. MRI data was first analyzed by group independent component analysis, then a graph theoretical method was applied to evaluate topological properties within whole brain. Small-world network parameters and nodal topological properties were measured. Altered topological properties in brain areas between groups were selected as ROI-seeds, then functional connectivity among these ROI-seeds were compared between groups. Partial correlation analysis was performed to evaluate the relationship between sleepiness severity and functional connectivity or topological properties in the narcolepsy.

Results: 21 independent components out of 48 components were obtained. Compared with healthy controls, narcolepsy exhibited a significant decreased functional connectivity within the executive and salience network, while increased functional connectivity in bilateral frontal lobe within executive network can be detected in narcolepsy. There were no differences in small-world network properties between narcolepsy and healthy controls. The altered brain areas in nodal topological properties were mainly located in inferior frontal cortex, basal ganglia, anterior cingulate, sensory cortex, supplementary motor cortex and visual cortex between groups. In the partial correlation analysis, nodal topological properties in putamen, anterior cingulate and sensory cortex as well as functional connectivity between these brain regions were correlated with the severity of sleepiness (sleep latency, REM sleep latency and ESS) among narcolepsy.

Conclusion: Altered connectivity within executive network and salience network were found in narcolepsy. Functional connection changes between left frontal cortex and left caudate nucleus may be one of the parameters describing the severity of narcolepsy. Nodal topological properties alterations in left putamen and left posterior cingulate, changes in functional connectivity between left supplementary motor area and right occipital as well as changes in functional connectivity between left anterior cingulate gyrus and bilateral postcentral gyrus can be considered to be a specific indicator for evaluating the severity of narcolepsy.

Support: National Natural Science Foundation of China (81700088)National Program on Key Basic Research Project of China (973 Program, 2015CB856405)

0762

EFFICACY OF PITOLISANT IN PATIENTS WITH HIGH BURDEN OF NARCOLEPSY SYMPTOMS

Davis, C. W.¹ Kallweit, U.² Krahn, L. E.³ Vaughn, B.⁴ Thorpy, M. J.⁵

¹Harmony Biosciences, LLC, Plymouth Meeting, PA, ²Universität Witten/Herdecke, Center for Narcolepsy and Hypersomnias, Institute of Immunology, and Center for Biomedical Education and Research, Witten, GERMANY, ³Mayo Clinic, Phoenix, AZ, ⁴Rho, Durham, NC, ⁵Albert Einstein College of Medicine, Bronx, NY.

Introduction: Recent literature suggests that histamine may play an important role in narcolepsy. This post hoc analysis evaluates the efficacy of pitolisant, a histamine 3 (H_3)-receptor antagonist/ inverse agonist, in patients with high burden of the main narcolepsy symptoms.

Methods: Data were pooled from 2 randomized, placebocontrolled, 7- and 8-week studies of pitolisant (individually titrated; maximum dose, 35.6 mg/day) in adults with narcolepsy. Analyses included 3 independent patient subgroups: baseline score of >16 on the Epworth Sleepiness Scale (ESS), sleep latency of ≤ 8

minutes on the Maintenance of Wakefulness Test (MWT), and ≥ 15 cataplexy attacks per week.

Results: The analysis populations included 108 patients for the ESS (pitolisant, n=54; placebo, n=54), 105 for the MWT (pitolisant, n=59; placebo, n=46), and 31 for cataplexy (pitolisant, n=20; placebo, n=11). Mean change in ESS from baseline was significantly greater for pitolisant (-6.1) compared with placebo (-2.6; P=0.0002). A significantly greater percentage of pitolisanttreated patients were classified as treatment responders: for ESS score reduction $\geq 3.68.5\%$ in the pitolisant group versus 35.2%in the placebo group (P=0.0006); for final ESS score $\leq 10, 35.2\%$ versus 9.3%, respectively (P=0.0026). Mean increase in sleep latency on the MWT was significantly greater for pitolisant (7.0 minutes) compared with placebo (3.4 minutes; P=0.0089). Decrease in mean weekly rate of cataplexy was significantly greater for pitolisant (baseline, 21.8; final, 3.9) compared with placebo (baseline, 20.9; final, 18.2); the rate ratio was 0.35 (95% CI, 0.26-0.47; P < 0.001). The adverse event profile in the analysis populations was consistent with the known safety profile for pitolisant; headache was the most common adverse event in pitolisant-treated patients (10.0%-20.4%).

Conclusion: In patients with severe symptom burden, pitolisant produced significantly greater improvements in excessive daytime sleepiness and cataplexy compared with placebo, highlighting the important role of histamine in narcolepsy.

Support: Bioprojet Pharma and Harmony Biosciences, LLC.

0763

EFFECTS OF SOLRIAMFETOL ON DRIVING PERFORMANCE IN PARTICIPANTS WITH NARCOLEPSY

Vinckenbosch, F.¹ Lammers, G.² Overeem, S.³ Chen, D.⁴ Wang, G.⁴ Carter, L.⁴ Zhou, K.⁴ Ramaekers, J.¹ Vermeeren, A.¹

¹Maastricht University, Maastricht, NETHERLANDS, ²Sleep-Wake Centre SEIN, Zwolle, NETHERLANDS, ³Kempenhaeghe, Heeze, NETHERLANDS, ⁴Jazz Pharmaceuticals, Palo Alto, CA.

Introduction: Patients with narcolepsy have an increased risk of automobile accidents. Solriamfetol, a dopamine/norepinephrine reuptake inhibitor, is approved in the US (Sunosi[®]) for adults with excessive daytime sleepiness (EDS) associated with narcolepsy (75-150 mg/day). This study evaluated the effects of solriamfetol on on-road driving performance in participants with narcolepsy.

Methods: In each period of this randomized, double-blind, placebo-controlled, crossover study (NCT 02806908; EudraCT 2015-003931-36), driving performance during an on-road driving test (a 1-hour drive on a public highway) was assessed at 2 hours and 6 hours postdose following 7 days of treatment with solriamfetol (150 mg/day \times 3, then 300 mg/day \times 4) or placebo. For assessment of driving performance, the primary endpoint was standard deviation of lateral position (SDLP), a measure of "weaving," at 2 hours postdose. Comparisons (solriamfetol vs placebo) used a Wilcoxon signed-rank test.

Results: The study included 24 participants (54% male; mean age, 40 years); 22 were included in the analyses of SDLP data. At 2 hours postdose, SDLP for solriamfetol (median, 19.08 cm) was statistically significantly lower than that for placebo (median, 20.46 cm; P=0.0022; incomplete driving tests: solriamfetol, n=4; placebo, n=7), indicating a better performance with solriamfetol. At 6 hours postdose, SDLP for solriamfetol (median, 19.59 cm) was not statistically significantly different from that for placebo (median, 19.78 cm; P=0.1245; incomplete driving tests: solriamfetol, n=3; placebo, n=10). Common adverse events (\geq 5%) were headache,

decreased appetite, somnolence, sleep disorder, agitation, nausea, and palpitations.

Conclusion: Solriamfetol (300 mg/day) improved SDLP, an important measure of driving performance, at 2 hours after administration in participants with narcolepsy. **Support:** Jazz Pharmaceuticals

0764

PITOLISANT IN THE TREATMENT OF PATIENTS WITH NARCOLEPSY: A 2-YEAR, PROSPECTIVE, OBSERVATIONAL, SINGLE-CENTER STUDY

Triller, A. Hof zum Berge, A. Finger, B. Kallweit, U. Center for Narcolepsy and Hypersomnias, Institute of Immunology, and Center for Biomedical Education and Research, Universität Witten/Herdecke, Witten, GERMANY,

Introduction: Pitolisant, a selective histamine H_3 receptor antagonist/inverse agonist, increases histamine release in the brain. The efficacy of pitolisant in adults with narcolepsy was demonstrated in randomized, placebo-controlled trials. This study evaluated long-term use of pitolisant in clinical practice.

Methods: This prospective, open-label, 2-year, observational study was conducted at a major narcolepsy center in Germany and enrolled adults with a diagnosis of narcolepsy who had no prior treatment with pitolisant. Assessments included excessive day-time sleepiness (Epworth Sleepiness Scale [ESS]), weekly rate of cataplexy (WRC), and health-related quality of life (Short-Form Veterans RAND [VR-36]).

Results: The study enrolled 147 patients: mean age, 29.9 years; 57.1% female, 65.3% with cataplexy, and 66.7% with disrupted nighttime sleep. In patients who were tested, CSF hypocretin-1 was <110 pg/mL in 70.8% (51/72), and 79.4 % (77/97) were HLA-DQB1*0602 positive. The pitolisant dose was 35.6 mg/d in 38.1% of patients at Month 3, and 73.5% at Month 24. Most patients received concomitant narcolepsy medications (63.3% at baseline; 79.6% at month 24). Mean ESS score decreased from 16.2 at baseline to 12.4 at Month 12 and 12.6 at Month 24. Mean WRC was reduced by 31% at Month 24. Significant improvement in quality of life was noted at Months 12 and 24 on VR-36 subscales that assess general health perception, vitality, and social function. In all, 38 patients (25.8%) discontinued from the study before Month 24: 15.0% for lack of efficacy and 10.8% due to adverse events. The most common adverse events were disrupted nighttime sleep (29.3% of patients), headache (15.5%), and nausea (12.2%).

Conclusion: These real-world data show that long-term treatment with pitolisant (usually with 35.6 mg/d) was efficacious for reducing EDS and cataplexy and improving quality of life in patients with narcolepsy. Treatment was generally well tolerated.

Support: Writing support funded by Harmony Biosciences, LLC.

0765

RELIABILITY AND VALIDITY STUDY OF THE CHINESE VERSION OF NARCOLEPSY SEVERITY SCALE FOR ADULT PATIENTS WITH NARCOLEPSY TYPE 1

Li, C.^{1,2} Zhang, C.² Han, F.²

¹Peking University School of Nursing, Beijing, CHINA, ²Peking University People's Hospital, Beijing, CHINA.

Introduction: Narcolepsy is a chronic sleep disorder that can affects significantly patient functioning, involving social, work, and affective life. At present, many drugs have been developed to treat narcolepsy efficiently. But there is no Chinese version of Narcolepsy

Severity Scale (NSS) available yet, for that the aim of this study is to translate the NSS into Chinese and evaluate reliability and validity of the NSS in adult patients with narcolepsy type 1 (NT1).

Methods: NSS was translated according to the standard procedures of double-back translation and cross-cultural adaptation steps. The NSS was administered to 62 adult patients (42 males, 20 females; mean age 34 years; range 19 to 67 years) with NT1 from April 2019 to December 2019. The validity of the scale was assessed by the exploratory factor analysis, discriminant validity and convergent validity. The reliability was assessed by the Cronbach's α coefficient and test-retest reliability.

Results: Three common factors were extracted and 15 items explained 57.4% of the total variance. Cronbach's α coefficient for total scale was 0.767 and Cronbach's α for three dimensions ranged from 0.729 to 0.787. Scores were significant difference between treated and untreated group in dependent samples (p=0.036), but no differences in the independent samples (p>0.05). The NSS had good correlations with Epworth Sleepiness Scale (r=0.302, p=0.017) and Insomnia Severity Index (r=0.526, p=0.000). The NSS showed good test-retest reliability (r=0.72, p=0.029).

Conclusion: The Chinese version of NSS was proved to be valid and reliable and can be used to evaluate the severity and consequences of symptoms in Chinese adult patients with NT1. **Support:**

0766

SAFETY AND TOLERABILITY OF PITOLISANT IN THE TREATMENT OF ADULT PATIENTS WITH NARCOLEPSY: FINAL ANALYSIS OF AN OPEN-LABEL, EXPANDED ACCESS PROGRAM IN THE UNITED STATES

*Bauer, E. D.¹ Davis, C. W.¹ Patroneva, A.¹ Dayno, J. M.¹ Thorpy, M. J.*²

¹Harmony Biosciences, LLC, Plymouth Meeting, PA, ²Albert Einstein College of Medicine, Bronx, NY.

Introduction: Pitolisant Expanded Access Clinical Evaluation (PEACE) provided adult patients with narcolepsy access to treatment with pitolisant while it was an investigational medication in the United States.

Methods: Pitolisant was titrated to 35.6 mg/day (or the highest tolerable dose) over a 3-week period. Dose adjustments were permitted at the discretion of the treating physician based on patient response. Treating physicians followed their standard of care and were required to report adverse events (AEs). Demographic and baseline information for all enrolled patients, and safety results available through October 30, 2019, are reported here (presentation will include final data from the PEACE program).

Results: In all, 623 patients (67.9% female; 84.6% white; mean age, 40.0 years; narcolepsy type 1, 51.5%) were treated with pitolisant in the PEACE program. Nearly all patients (98.4%) had been previously treated with other narcolepsy medications (88.1% with ≥ 2 narcolepsy medications). Overall, 35.2% of patients discontinued from the program; 16.7% due to an AE and 12.2% for lack of effect. At Month 1, 97.3% of patients remained in the study, 88.2% at Month 3, 76.5% at Month 6, 66.9% at Month 9, and 55.0% at Month 12. In all, 256 (41.1%) patients experienced ≥ 1 AE; majority (52.5%) of these AEs occurred early in treatment (by Week 3). The most commonly reported AEs were headache (9.8% of AEs), nausea (6.6%), anxiety (5.6%), and insomnia (4.7%).

Conclusion: In the PEACE program, patient characteristics were generally reflective of the US narcolepsy patient population. The safety and tolerability profile of pitolisant was similar to that seen

in the clinical development program, with no new safety signals identified. The program ceased enrollment in August 2019 after the US approval of pitolisant for the treatment of excessive daytime sleepiness in adult patients with narcolepsy.

Support: Harmony Biosciences, LLC.

0767

ANALYSIS OF THE EFFECT ON BLOOD PRESSURE AND HEART RATE WHEN ADDING SOLRIAMFETOL (SUNOSI) TO STIMULANT THERAPY

Meskill, G. J.¹ Meskill, S. D.²

¹Comprehensive Sleep Medicine Associates, Houston, TX, ²Texas Children's Hospital, Houston, TX.

Introduction: Solriamfetol is a non-stimulant wakefulnesspromoting agent (WPA) indicated for the treatment of excessive daytime sleepiness in adult patients with obstructive sleep apnea or narcolepsy. It acts by inhibiting reuptake of dopamine and norepinephrine. Since many patients with excessive daytime sleepiness take stimulants, clinicians commonly ponder the safety of adding solriamfetol in this population due to concern of increased blood pressure and/or heart rate (HR).

Methods: We conducted a retrospective chart review and identified 18 patients who had solriamfetol added to their stimulant therapy. Of those, 7 to date have had a follow-up appointment after the addition of solriamfetol (6 on 150mg, 1 on 75mg). We collected the blood pressure and HR readings at the appointment immediately prior to and following the addition of solriamfetol and conducted a paired t-test.

Results: The systolic blood pressure (SBP) had a mean difference of -0.57 (95% CI -9.6 to 8.5, p=0.88), diastolic blood pressure (DBP) 1.7 (95% CI -4.4 to 7.9, p=0.52), mean arterial pressure (MAP) 0.95 (95% CI -5.6 to 7.5, p=0.73), and HR 6.6 (95% CI -0.07 to 13.2, p=0.052).

Conclusion: The addition of solriamfetol to stimulant therapy did not lead to a significant increase in SBP, DBP, MAP, or HR. **Support:** none

0768

TIME COURSE OF IMPROVEMENT IN EXCESSIVE DAYTIME SLEEPINESS AND CATAPLEXY DURING TREATMENT WITH PITOLISANT IN PATIENTS WITH NARCOLEPSY

*Roy, A.*¹ *Davis, C. W.*² *Vaughn, B.*³ *Dayno, J. M.*² *Dauvilliers, Y.*⁴ *Schwartz, J.*⁵

¹Ohio Sleep Medicine Institute, Dublin, OH, ²Harmony Biosciences, LLC, Plymouth Meeting, PA, ³Rho, Durham, NC, ⁴National Reference Center for Narcolepsy, Montpellier, FRANCE, ⁵Bioprojet Pharma, Paris, FRANCE.

Introduction: This analysis evaluated the efficacy of pitolisant over time in three 7- to 8-week, randomized, placebo-controlled studies of adults with narcolepsy.

Methods: Patients in all 3 studies (HARMONY-1, HARMONY-1bis, HARMONY-CTP) experienced excessive daytime sleepiness (EDS) at study baseline; patients in HARMONY-CTP also experienced ≥3 cataplexy attacks/week. Pitolisant was titrated to a maximum dose of 35.6 mg/day (HARMONY-1, HARMONY-CTP) or 17.8 mg/day (HARMONY-1bis). Change from baseline in mean Epworth Sleepiness Scale (ESS) score (3 studies) and mean weekly rate of cataplexy (WRC; 1 study) was compared for pitolisant versus placebo.

Results: In the higher-dose HARMONY-1 (pitolisant, n=31; placebo, n=30) and HARMONY-CTP (pitolisant, n=54; placebo, n=51) studies, ESS score improvement was significantly greater with pitolisant versus placebo beginning at Week 2 (LS mean difference, -2.8; P=0.015) and Week 3 (LS mean difference, -2.0; P=0.005), respectively. In the lower-dose HARMONY-1bis study (pitolisant, n=66; placebo, n=32), significant separation from placebo was first observed at Week 7 (LS mean difference, -2.3; P=0.044). At end-of-treatment, LS mean difference in ESS score change from baseline was -3.1 (P=0.022) in HARMONY-1, -3.4 (P<0.001) in HARMONY-CTP, and -2.2 (P=0.030) in HARMONY-1bis. In HARMONY-CTP, LS mean WRC with pitolisant was 11.7 at baseline, 4.6 at end-of-treatment, and 5.1 after a 1-week, placebowashout period. Improvement in WRC was significantly greater with pitolisant versus placebo beginning at Week 2 (LS mean difference, -5.3; P=0.004) and continued through end-of-treatment (LS mean difference, -6.2; P<0.001); there was no evidence of rebound cataplexy after placebo-washout (LS mean difference, -4.9; P=0.027).

Conclusion: During pitolisant treatment, improvement in EDS occurred sooner (within first few weeks) and was more robust in studies that permitted titration to the maximum recommended dose (35.6 mg/day). The rate of cataplexy attacks decreased early during treatment, with no evidence of rebound when pitolisant was withdrawn.

Support: Bioprojet Pharma and Harmony Biosciences, LLC.

0769

FDG-PET IMAGING IN NARCOLEPSY TYPE 1, IDIOPATHIC HYPERSOMNIA, AND NON-SLEEPY CONTROLS

Trotti, L. Meltzer, C. Rye, D. Nye, J. Emory University School of Medicine, Atlanta, GA.

Introduction: Functional imaging of narcolepsy type 1 (NT1) has shown disparate results, with evidence for both regional hyper- and hypo-metabolism. A FDG-PET study of idiopathic hypersomnia (IH) demonstrated regional hypermetabolism within the salience network.

Methods: Patients with NT1 (n=14) or IH (n=16) were recruited, with age-matched, non-sleepy controls (HC, n=8). Patients discontinued treatment for \geq 5 half-lives. Participants underwent injection of ¹⁸F-fludeoxyglucose (FDG) in a dimly-lit room and were asked to remain awake, seated quietly. Simultaneous 6-channel EEG, EOG, and EMG were collected. Participants were alerted by a loud noise if sleep onset was observed. Thirty minutes after injection, patients underwent 36-minute PET scan. Images were spatially normalized to MPRAGE images and analyzed for group differences using SPM8.

Results: Groups were similar in age (NT1: 30.0 (+/-SD 8.3), IH: 36.3 (+/-12.4), HC: 33.2 (+/-16.2), p=0.29) and gender (%women, NT1: 71%, IH: 87.5%, HC: 62.5%, p=0.37). Patients were sleepier than controls by Epworth (NT1: 18.2 (+/-3.5), IH: 15.8 (+/-3.2), HC: 5.0 (+/-2.7), p<0.0001) and MSLT mean latency (NT1: 2.0 (+/-1.4), IH: 5.1 (+/-1.7), HC: 14.6 (+/-2.6), p<0.0001). Despite attempts to remain awake, NT1 patients had difficulty maintaining wakefulness during uptake, obtaining 6.2 (+/-5.9) minutes sleep versus <1 minute for the other groups. Compared to controls, NT1 patients demonstrated increased activation in bilateral precentral gyri, left postcentral gyrus, left middle frontal gyrus, right insula, right inferior and superior temporal gyri, right fusiform gyrus, and bilateral inferior frontal gyri. Compared to controls, IH patients

demonstrated increased activation in bilateral precuneus, bilateral inferior and middle frontal gyri, left middle and superior temporal gyri, left inferior parietal lobule, and left anterior cingulate.

Conclusion: Different patterns of metabolic activity are seen in two hypersomnia disorders, implying disease-specific activity rather than non-specific sleepiness. Inadvertent sleep during uptake is more common in NT1.

Support: K23 NS083748

0770

<OLFACTORY DISORDERS IN INDIVIDUALS WITH DIAGNOSIS OF NARCOLEPSY>

Truzzi, G. M.¹ Naufel, M. F.¹ Coelho, F. M.¹

¹Universidade Federal de São Paulo, São Paulo, BRAZIL,

²Universidade Federal de São Paulo, São Paulo, BRAZIL.

Introduction: Narcolepsy is a sleep disorder characterized mainly by excessive daytime sleepiness, cataplexy, sleep paralysis, hypnagogic-hypnopompic hallucinations and sleep fragmentation. Besides those most known manifestations, other findings may be present in patients with narcolepsy, such as weight gain, reduction in eating satiety and psychological alterations. It is also observed that part of the hypocretin-producing cells projections are present in the olfactory bulb, and the conditions appears to be related to changes in olfaction.

Methods: a cross sectional study was performed in patients diagnosed with narcolepsy followed up by the excessive daytime sleepiness outpatient clinic of the discipline o Sleep Biology and Medicine of the Department of Psychobiology of the Federal University of São Paulo, Brazil. Olfaction was assessed by the University of Pennsylvania Smell Identification Test (UPSIT) following an interview and nasal cavity examination. Patients with conditions and disorders that may cause hyposmia were excluded.

Results: 77 patients were assessed, of which 56 had type-1 narcolepsy and 21 with type-2 narcolepsy. The results were compared with the test's reference data. Most patients with type-1 and type-2 narcolepsy presented scores compatible with some degree of olfactory impairment. No significant difference was observed between the scores of patients with type-1 and type-2 narcolepsy.

Conclusion: The present study shows most patients with narcolepsy have some degree of olfactory impairment. This impairment doesn't appear to be explained by alterations in the hypocretineric cells as pointed out in previous studies. The changes in olfaction in people with narcolepsy may cause the satiety alterations often observed in them. Other mechanisms involved with the genesis of hyposmia in those patients should be studied further.

Support: AFIP - Associação Fundo de Incentivo à Pesquisa

0771

EXPLAINING SELF-REPORTED HYPERSOMNOLENCE IN SEASONAL DEPRESSION

Wescott, D. L.¹ Hasler, B. P.² Franzen, P. L.² Roecklein, K. A.¹ ¹University of Pittsburgh, Pittsburgh, PA, ²University of Pittsburgh Medical Center, Pittsburgh, PA.

Introduction: Hypersomnolence is commonly reported in Major Depressive Disorder with Seasonal Pattern (Seasonal Affective Disorder; SAD). However, self-reported hypersomnolence may conflate long sleep duration, daytime sleepiness, fatigue, increased time in bed, or maladaptive sleep cognitions, undermining treatment efforts. **Methods:** Eighty-eight participants ages 18-65 years old were recruited during the winter (SAD = 43, Control = 45). Depression severity was assessed by a clinician rated interview, and self-reported hypersomnolence was determined by assessing self-reported sleep duration. Participants wore an Actiwatch for 4-14 days and completed self-report measures of daytime sleepiness and fatigue. We performed a hierarchical linear regression to determine which factors best explain self-reported winter hypersomnolence: actigraphic total sleep time (TST), time in bed (TIB), depression severity, sleepiness, or fatigue. Due to collinearity of TST and TIB, we separated those variables into two models predicting hypersomnolence.

Results: SAD participants endorsed greater hypersomnolence than controls during the winter (B = .714; p < .001). In model 1, TST (OR(1,14) = .024, p <.001) and daytime sleepiness (OR(1,14) = .208, p = .03) significantly predicted the presence of self-reported winter hypersomnolence above and beyond age, gender, depression, and fatigue. In model 2, only TIB (OR(1, 14) = .021, p = .001) was a significant predictor. Post-hoc analyses indicated that fatigue and depression severity significantly predicted self-reported hypersomnolence when entered separately into the model. Sleepiness accounted for the largest change in pseudo- R^2 in bth models.

Conclusion: We found evidence for the multifaceted etiology of self-reported hypersomnolence. Daytime sleepiness, sleep duration, time in bed, and the shared variance between fatigue and depression severity all explained self-reported hypersomnolence. Treatment of hypersomnolence should include actigraphy, and should be individually tailored based on presentation.

Support: NIMH K.A.R. MH103303

0772

EFFECTS OF SOLRIAMFETOL ON 24-HOUR BLOOD PRESSURE PATTERNS IN PARTICIPANTS WITH EXCESSIVE DAYTIME SLEEPINESS ASSOCIATED WITH NARCOLEPSY

Strollo, P. J.¹ Malhotra, A.² Strohl, K.³ Pepin, J.⁴ Schweitzer, P.⁵ Lammers, G.⁶ Hedner, J.⁷ Baladi, M.⁸ Carter, L.⁸ Bujanover, S.⁸ Menno, D.⁹ Dauvilliers, Y.¹⁰

¹University of Pittsburgh/Veterans Administration Pittsburgh Health System, Pittsburgh, PA, ²Division of Pulmonary, Critical Care and Sleep Medicine, University of California San Diego, La Jolla, CA, ³Case Western Reserve University, Cleveland, OH, ⁴Grenoble Alpes University Hospital, Grenoble, FRANCE, ⁵Sleep Medicine and Research Center St. Luke's Hospital, Chesterfield, MO, ⁶Sleep-Wake Centre SEIN, Zwolle, NETHERLANDS, ⁷Sahlgrenska University Hospital, Gothenburg, SWEDEN, ⁸Jazz Pharmaceuticals, Palo Alto, CA, ⁹Jazz Pharmaceuticals, Philadelphia, PA, ¹⁰Gui-de-Chauliac Hospital, Montpellier, FRANCE.

Introduction: Solriamfetol is a dopamine and norepinephrine reuptake inhibitor indicated to improve wakefulness in adult patients with excessive daytime sleepiness associated with narcolepsy (75-150 mg/d) or obstructive sleep apnea (37.5-150 mg/d). Previous studies reported small mean increases in blood pressure (BP); however, the time course of these effects has not been evaluated. In addition, effects on BP dipping, which has been shown to be a risk factor for adverse cardiovascular outcomes, have not been evaluated. These analyses evaluated the effects of solriamfetol treatment on BP using 24-hour ambulatory blood pressure monitoring (ABPM) and on the percentage of narcolepsy patients with a nondipping BP profile.

Methods: Twenty-four-hour ABPM was conducted at baseline and week 8 in a 12-week randomized controlled trial in participants with narcolepsy (n=236).

Results: At week 8, increases in BP were apparent in the 150 and 300 mg dose groups from 8 AM until 4 PM and 6 PM, respectively. At baseline, 52% (placebo) and 48% (combined solriamfetol) of participants were non-dippers (defined as <10% decrease in mean arterial pressure [MAP] during sleep). There was no increase in the percentage of non-dippers at week 8 relative to baseline (placebo, 44%; combined solriamfetol, 39%). Results were similar when dipping was defined by changes in systolic BP and diastolic BP.

Conclusion: The effects of solriamfetol on BP at the highest approved dose of 150 mg/d are transient across the day. Solriamfetol was not observed to have an increase in non-dipping classification in participants with narcolepsy at any dose studied.

Support: Jazz Pharmaceuticals

0773

WHY DID PANDEMRIX TRIGGER NARCOLEPSY? A STRUCTURAL APPROACH.

Peris Sempere, V. Ambati*, A. Luo*, G. Lin, L. Mignot, E.* Stanford university Center for Sleep Sciences and Medicine, Palo Alto, CA.

Introduction: The 2009 Pandemrix influenza A pH1N1 vaccine has been linked to an increased number of Narcolepsy type I onsets in children across Europe whereas administration of a very similar adjuvanted vaccine, Arepanrix, had little effects in Canada. One possible explanation for the difference may be vaccine composition differences that could modify peptide binding to narcolepsy associated HLA-DQ0602 allele, as viral extracts for these two vaccines used distinct processes in different factories. Other explanations may involve differences in vaccination timing in relation to the pandemic H1N1 infection wave, or other environmental factors. We have previously compared the amino acid sequence of the Hemagglutinin (HA) component of the Pandemrix and the 2010 Arepanrix vaccine, finding possible contributors, but excluding most of these after DQ0602-tetramer analysis of T cell reactivity in narcolepsy versus controls.

Methods: Mass spectrometric characterization of multiple additional batches of Pandemrix and Arepanrix used during 2009 influenza pandemic vaccination campaign was performed.

Results: In addition to confirming previously published results such as increased deamidation of hemagglutinin (HA) (146N>D) in Pandemrix (p=2.1e-9), we identified novel differences, including a significant 2-fold post-translation deamidation increase in 277N in Arepanrix versus Pandemrix (p=0.032), together with increased 2-fold glycosylation in the 286-323 positions in Arepanrix (p=0.00036). The 277 N to D/isoD substitution is located in pocket 1 of the binding core of a strong binder NAGSGIIIS, (< 10% rank) for HLA-DQ0602 allele and abolishes epitope binding. The increased glycosylation in Arepanrix occurs in the immediate flanking area of the same 277N epitope and could also reduce DQ0602 presentation of the same epitope through differential binding and/or proteolysis of HA in this region of the molecules. As CD4 T cells recognizing this epitope have been reported to be significantly increased in narcolepsy versus DQ0602 controls, with possible mimicry with homologous hypocretin sequence.

Conclusion: These changes could explain why Arepanrix was less narcolepsy inducing. Confirmatory studies, as well as studies of all novel changes observed, are ongoing, but this is a promising result.

Support: Wake Up Narcolepsy

0774

FACTORS ASSOCIATED WITH THE CONTINUOUS USE OF PSYCHOTROPIC TREATMENTS FOR NARCOLEPSY

*Ohayon, M. M.*¹ *Krystal, A. D.*² *Black, J.*³ *Shapiro, C. M.*⁴ *Sullivan, S.*⁵ *Swick, T. J.*⁶ *Wells, C. C.*⁷ ¹Stanford University, Palo Alto, CA, ²Department of Psychiatry, UCSF School of Medicine, San Francisco, CA, ³Jazz Pharmaceuticals Inc, Jazz Pharmaceuticals inc, CA, ⁴Department of Psychiatry, University of Toronto, Toronto, ON, CANADA, ⁵SERI, Palo Alto, CA, ⁶Neurology and Sleep Medicine Consultants, Houston, TX, ⁷Sleepmed, Inc., Macon, GA.

Introduction: Narcolepsy is a debilitating disorder characterized by excessive sleepiness and cataplexy episodes. There is no cure for this disease. Current treatments focus on controlling the symptoms with CNS stimulants for sleepiness and antidepressants and/ or CNS depressants for cataplexy. This study examines the factors that can contribute to the cessation of narcolepsy treatment. **Methods:** The study includes 291 narcoleptic individuals who were interviewed twice, approximately five to seven years apart, in Wave 1 (W1) and Wave 2 (W2). Telephone interviews were conducted with the help of the Sleep-EVAL system; narcolepsy individuals were initially evaluated and diagnosed by a Sleep Specialist. **Results:** At W1, 49.2% of narcoleptic individuals were taking a

CNS stimulant; at W2, 37% of narcoleptic individuals were taking a CNS stimulant; at W2, 37% of narcoleptic individuals were taking a CNS stimulant. The use was chronic (i.e., present at W2 and W1) for 52.7% of the W2 subjects. CNS depressants were used by 19.1% at W1 and 17% at W2. Of the W1 subjects, 67.6% still reported using CNS depressants at W2. In terms of antidepressants, 38.6% and 29.6% of subjects reported using these medications at W1 and W2 respectively. Of those taking antidepressants at W2, 58.9% reported chronic use (ie, were also on antidepressants at W1). At least one of the aforementioned medication classes was used by 72% of participants at W1 and 56.1% at W2. Chronicity of nocturnal awakenings (RR: 2.7), the frequency of cataplexy episodes (RR: 2.3) and the chronicity of hypnopompic hallucinations (RR: 2.8) were associated with long-term use of narcolepsy treatment.

Conclusion: Narcolepsy treatments are mostly taken to long term. Some narcoleptics individuals were able to reduce or stop treatment either because the intensity of symptoms decreased or because they developed coping mechanisms to deal with the symptoms.

Support: NIH (R01NS044199), the Arrillaga Foundation and Jazz Pharmaceuticals Inc.

EFFECTS OF STRESS, SLEEP AND DEPRESSION ON RESILIENCE OF FEMALE NURSES WORKING IN SHIFT AND FIXED WORK SCHEDULES IN GENERAL HOSPITAL

Eun, H. Shin, H.

Presbyterian Medical Center-Jesus Hospital, Jeonju, KOREA, REPUBLIC OF.

Introduction: Healthy sleep is important and can have a positive effect on resilience. The aim of the present study was to compare the differences in resilience between two group nurses in rotating shift and daytime fixed work schedules and to investigate stress perception, coping factors, social and psychological health, and sleep factors that may affect resilience.

Methods: A total of 400 female nurses having rotating shift and daytime fixed work schedules at two hospital were surveyed from June 12, 2017 to June 12, 2018. Perceived stress scale(PSS), stress coping short form(Brief COPE), psycho-social wellbeing Index short form(PWI-SF) or general health questionnaire-18(GHQ-18), center for epidemiologic studies depression scale(CES-D), STAI-X-1 in state-trait anxiety inventory(STAI), Pittsburgh sleep quality index(PSQI), Epworth sleepiness scale(ESS), insomnia severity index(ISI), Conner Davidson resilience scale(CD-RISC) applied. Independent t-test, paired t-test, Pearson correlation analysis, and multiple regression analysis were applied to the results of the final 373 questionnaires of 400 nurses in two general hospitals. Results: As a result of comparing the variable statistics between the two groups of rotating shift and daytime fixed work nurses, there were statistically significant differences in all variables except perceived stress, sleep quality, and daytime sleepiness. Factors that had a significant correlation with resilience were stress coping strategies, depression, and insomnia severity(p<0.001). In multiple regression analysis, the larger positive reframing $1(\beta=0.206, p<0.001)$, the less depression (β = -3.45, p<0.001), and the higher psychosocial health level(β =0.193, p <0.001). As acceptance coping2 increases(β =0.129, p<0.05), as daytime sleepiness decreases(β =-1.17, p<0.05), and as active coping2 increases(β =0.118, p<0.05), as the positive reframing2 increases(β =0.110, p<0.05), the resilience increased.

Conclusion: In this study, it was found that resilience was higher in daytime fixed workers than in shift workers. In addition, specific stress coping strategies and sleep, depression, and anxiety factors were found to be associated with resilience.

Support: Key words: Shift work · Female nurse · Resilience · Sleep · Stress · Depression

0776

DIFFERENCES IN WELL-BEING IN DAYWORKERS COMPARED TO SHIFT WORKERS: A STUDY OF UNITED STATES NAVY SAILORS

Shattuck, N. L. Matsangas, P.

Human Systems Integration Program, Operations Research Department, Naval Postgraduate School, Monterey, CA.

Introduction: On United States Navy (USN) ships, most sailors are shift workers, required to support 24/7 operations. However, $\sim 15\%$ of the ship's company are solely dayworkers who do not work in shifts. It is often assumed that the quality of life for dayworkers is better than that of shift workers. This study compared the well-being of dayworkers with that of shift workers.

Methods: Longitudinal, naturalistic observations were made of sailors (N=926; 18-59 years of age, ~80% males, ~84% enlisted personnel) on seven US Navy ships while performing their normal

underway duties. Sleep-related attributes (actigraphy, Epworth Sleepiness Scale, Pittsburgh Sleep Quality Index, Insomnia Severity Index), mood (Profile of Mood States), and work/rest patterns were assessed.

Results: Dayworkers (DW; n=98) were on average older (p=0.001) and more senior (p=0.001) than shift workers (SW). Of the dayworkers, 31% reported elevated daytime sleepiness (SW:45.5%; p=0.008), 64.2% were poor sleepers (SW:82.6%; p<0.001), and 26.3% had ISI score≥15 (SW:24.8%; p=0.782). Also, 13.8% of the dayworkers had ESS>10 and ISI \geq 15 (SW:16.8%; p=0.626). Dayworkers had better mood (Total Mood Disturbance, anger/ hostility, vigor, fatigue; all p<0.050), slept more (7.03±0.74hrs sleep/day; SW:6.52±1.03hrs; p<0.001) and had more consolidated sleep (1.1 \pm 0.3 sleep episodes/day; SW:1.4 \pm 0.6; p<0.001). Yet, split sleep was commonplace for both groups (DW:~62%; SW:~92%). The two groups do not differ (all p>0.300) in their use of caffeinated beverages (82%-86%), use of nicotine products (30%-36%), or having a regular exercise routine (69%-75%). In both groups, ~9% of sailors drank caffeinated beverages, used nicotine products and did not have an exercise routine (p=0.999). Dayworkers worked 10.1 hours/day, i.e., 1.7 hours/day less than watchstanders (p<0.001).

Conclusion: Quality of life of dayworkers is a bit better when compared to shift workers, but sleep-related issues are evident in almost all US Navy sailors. Living and working on a naval vessel takes a toll on almost everyone aboard. A culture change is required! **Support:** Supported by the Naval Medical Research Center's Advantation Medical Research Center's

Advanced Medical Development Program, the US Navy 21st Century Sailor Office, and the US Navy OPNAV N1.

0777

VARIATIONS IN VIGILANCE AND SLEEP AMONG UNDERGROUND MINE WORKERS DURING 14 CONSECUTIVE NIGHT SHIFTS

Laberge, L.¹ Lavigne, A. A.² Auclair, J.¹ Hébert, M.² ¹ÉCOBES - Recherche et transfert, Cégep de Jonquière, Jonquière, QC, CANADA, ²Centre de recherche CERVO, Université Laval, Québec, QC, CANADA.

Introduction: Adverse effects of night shift work are well known but there is scarce data on how vigilance and sleep vary across a large number of consecutive night shifts.

Methods: In summer, 38 underground miners (mean age (SD): 36.8 (13.9) years) wore an actigraph, filled out the Morningness-Eveningness questionnaire, and completed a Visual Analog Scale capturing subjective vigilance (very sleepy to very alert) 4 times per shift (19:00, 22:00, 02:00, and 05:30) for 14 consecutive night shifts. Mixed effects linear regression models were used to account for repeated measures.

Results: Mean vigilance level is lower at 22:00, 02:00 and 05:30 than at the beginning of the shift at 19:00 (p<0.001). Also, a more pronounced decrease in vigilance during the night was observed among older workers compared to younger workers (p<0.05). Moreover, workers with greater eveningness have higher vigilance at the beginning of the first night shift at 19:00 (p<0.001), but their decline in vigilance level during the night is faster than that observed in workers with greater morningness (p<0.01). Interestingly, the mean vigilance decline observed at 02:00 and 05:30 (compared to 19:00) is slowed down for each additional night shift (p<0.001). Furthermore, mean sleep efficiency is negatively associated with morningness and gradually decreases across consecutive night

shifts (p<0.05). In addition, mean sleep duration is shorter in older workers and is positively associated with morningness (p<0.05).

Conclusion: Results show a progressive improvement in vigilance of mine workers assigned to a large number of consecutive 12-hour night shifts from 2 am onwards. This may probably be ascribed to an adjustment in homeostatic sleep propensity consecutive to the partial sleep deprivation associated with time spent traveling to the remote site before the first shift. However, circadian adjustment is unlikely considering the strong morning light exposure experienced daily after the night shift.

Support: College and Community Innovation Program of the Natural Sciences and Engineering Research Council of Canada (NSERC) (CU12I 472201-14)

0778

WORKPLACE YOGA PROGRAM FEATURES AND ASSOCIATIONS WITH SHIFT WORK AND SLEEP AMONG NURSING STAFF

Zhang, Y.¹ Thind, H.¹ Kim, S.¹ Soup, A.¹ Punnett, L.¹ Duffy, J.² ¹University of Massachusetts Lowell, Lowell, MA, ²Brigham and Women's Hospital, Boston, MA.

Introduction: Nursing staff are at risk for impaired sleep due to irregular schedules, long work hours, and other occupational stress. Yoga has demonstrated beneficial effects on sleep in healthy adults and patients with chronic diseases. However, yoga interventions are generally offered as 60-75-minute sessions; this long duration might not be suitable as a workplace program for nursing staff. The objective of this study is to examine workplace yoga program features and associations with shift work and sleep among nursing staff.

Methods: Online Qualtrics surveys were distributed among nurses and nursing assistants at a community hospital in the northeast U.S. Hypothetical workplace yoga program features were assessed including general interest, duration, frequency, timing with respect to work shift, and interest in home practice. Sleep duration and disturbances were assessed.

Results: Among the 541 participants (94% female; age 43±13y), over a third reported sleep \leq 6hrs/day (38%) and sleep disturbances (38%), and 79% reported interest in workplace yoga. Among those reporting interest, after work (61%), 30min/session (73%), 3 sessions/week (56%), and interests in home practice (64%) were yoga features endorsed by nursing staff. Night or \geq 12hr shift was associated with less interest in yoga after work, while day or night shift was associated with less interest in yoga after work. Mild sleep disturbances were associated with less interest in yoga after work. Mild sleep disturbances were associated with less interest in yoga after work. Nursing staff with sleep \leq 6hrs/day reported less interest in yoga before work, but more interest in home yoga practice.

Conclusion: Nursing staff reported a high prevalence of short and disturbed sleep and interest in workplace yoga. Workplace yoga programs need to be designed according to nursing staff's interest while considering the effect of shift work and sleep problems reported by nursing staff.

Support: Drs. Yuan Zhang and Jeanne F. Duffy were supported by NIH grant R01 AG044416.

0779

CIRCADIAN ENTRAINMENT IN 12H NIGHT SHIFT WORKERS: THE ENVIRONMENTAL LIGHTS MATTER? *Choi, S.¹ Park, H.² Joo, E.³* ¹Department of Nursing, Samsung Medical Center, Seoul, KOREA, REPUBLIC OF, ²Department of Neurology, Inje University College of Medicine, Ilsan Paik Hospital, Ilsan, KOREA, REPUBLIC OF, ³Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of medicine, Seoul, KOREA, REPUBLIC OF.

Introduction: Shift workers frequently suffer sleep disturbance in relation with the atypical work schedules. In particular, night shift workers are exposed to inevitable sunlight or bright indoor lights during the morning hours that may disturb their daytime sleep. We aimed to find out the optimal environmental lights to facilitate circadian entrainment of 12h-shift workers by assessing melatonin profiles in the morning and daytime sleep quality.

Methods: We enrolled 12h-shift female nurses working at one hospital (n=10, mean age 29.4±3.5 years). The schedules of participants are identical such as day-day-night-night shifts and four consecutive off days. Participants admitted to the laboratory at 9:00 following 2nd night shift schedule. Saliva melatonin was taken six times every 30 minute from 10:00 to 12:30 under two different lighting conditions with organic light emitting diodes (OLED) or light emitting diodes (LED) with 150 lux of light intensity. Dim light condition (10 lux) was conducted as controls. Three sessions were randomly ordered with 8-10 days intervals. Participants were allowed to sleep after 12:30 with light off and woke up ad libitum. Results: Melatonin concentration had decreased gradually from 10.6±11.4 to 5.0±8.2 pg/ml. Among three different lighting conditions, there were no statistical differences in salivary melatonin and sleep parameters recorded by polysomnography. Circadian entrainment in night shift workers was defined as that salivary melatonin concentration had maintained above 5 pg/ml at the time to bed. 20% of sessions (6/30) were classified as circadian entrainment (CE) and 80% (24/30) were as non-entrainment (NE). Mean melatonin concentration was 22.1±11.2 in CE and 3.9±4.1 pg/ml in NE (p<.001). CE showed significantly shorter sleep latency (0.5±0.3 vs. 1.5 \pm 1.4 min, p=.025) and wakefulness after sleep onset (13.6 \pm 6.3 vs. 27.9 \pm 16.8%, p=.015), and higher sleep efficiency (94.6 \pm 2.6 vs. 88.7 \pm 6.3%, p=.011) than NE. The number of each lighting condition was not different between CE and NE (p=.847).

Conclusion: This is a preliminary study with small number of participants. We found that environmental lights including dim light did not affect daytime sleep of 12h shift workers. Instead, daytime sleep quality was influenced by circadian entrainment with higher melatonin concentration in the morning. **Support:**

0780

EFFECT OF A SLEEP/CIRCADIAN FRIENDLY PROTOCOL ON THE OUTCOMES OF PATIENTS ADMITTED TO THE MEDICAL INTENSIVE CARE UNIT: A RANDOMIZED CONTROL TRIAL

Wakefield, C.¹ Morrow, J.² Hamati, F.² Sanzo, G.² Peterson, S.³ Balk, R. A.⁴ Burgess, H.⁵ Swanson, G.² Keshavarzian, A.² ¹Rush University Medical Center, Chicago, IL, ²Rush University Medical Center, Department of Internal Medicine, Division of Digestive Diseases and Nutrition, Chicago, IL, ³Rush University Medical Center, Department of Clinical Nutrition, Chicago, IL, ⁴Rush University Medical Center, Department of Internal Medicine, Division of Pulmonary, Critical Care, and Sleep Medicine, Chicago, IL, ⁵University of Michigan, Sleep and Circadian Research Laboratory, Lansing, MI. **Introduction:** Critically ill patients often suffer from fragmented sleep due to light and noise in the intensive care unit (ICU). We investigated whether placing patients in a sleep/circadian friendly protocol improved patient-reported sleep quality and ICU outcomes.

Methods: Patients were included if they were expected to have a >24-hour stay. Exclusion criteria included age <18 years, pregnancy, frequent overnight assessments, poor prognosis, pre-existing cognitive impairment, and severe psychological disorders. Patients were randomized to a sleep/circadian protocol or control protocol. Demographic, treatment, sound (dB), light (lux), patient-reported sleep, and medical outcomes were collected. Those who were not maintained in the sleep/circadian protocol were combined with the controls for statistical analysis. Univariate analysis was performed with Chi-Square test and Wilcoxon rank-sum test.

Results: Sixty-one patients were enrolled: 28 randomized to sleep/ circadian protocol, 33 to control protocol. 14 subjects were excluded from analysis due to developing criteria for exclusion. Nine patients (median age 50 years, 6 females) maintained in the sleep/ circadian protocol, 13 patients were not maintained in sleep/circadian protocol and 25 patients in control protocol (median age 64 years, 20 females). There was no difference in age, gender, or race between groups (p > 0.05). Median ICU first overnight dB level (10:00pm-6:00am) was 44.2 (±2.7) for sleep/circadian protocol and 50.5 (\pm 4.9) for combined group (p= 0.008). Median ICU first overnight lux (10:00pm-4:00am) was 2.28 (±61.3) in sleep/circadian protocol and $18.42 (\pm 90.9)$ in combined group (p=0.0374). A continued stay in the sleep/circadian protocol resulted in decreased total decibel level (p=0.0018) decreased total overnight lux exposure (p=0.0025) and decreased reported awakenings in first night (p = 0.0175). There were no differences in ICU length of stay, inpatient mortality, or readmission rates between groups (p>0.05). Three patients developed delirium, all originally randomized to control group.

Conclusion: We report on the successful outcomes associated with a sleep/circadian friendly protocol within a large tertiary center medical ICU which resulted in significant decreases in object-ively assessed noise and light, and subjectively reported nocturnal awakenings. We continue to collect data to determine if the sleep/ circadian friendly protocol should be permanently implemented in the ICU.

Support: None.

0781

IMPACT OF SUVOREXANT ON TOTAL DAYTIME SLEEP HOURS IN SHIFT WORKERS: A RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED CLINICAL FIELD TRIAL

Zeitzer, J. M.¹ Joyce, D. S.¹ Sterkel, A. L.¹ Quevedo, Y. L.² Hernandez, B.² Holty, J.¹

¹Stanford University, Palo Alto, CA, ²VA Palo Alto Health Care System, Palo Alto, CA.

Introduction: Many shift workers have an inability to sleep during the daytime following a night shift not due to insomnia or lack of sleep pressure, but because a circadian signal promoting wakefulness is hampering their ability to maintain sleep. We have previously hypothesized that the neuropeptide hypocretin-1 is, in part, responsible for the physiologic expression of this circadian wake signal. As such, it was our intent to determine whether a pharmacologic blockade of hypocretin would enable shift workers to obtain more daytime sleep.

Methods: Nineteen shift workers took part in a placebocontrolled, double-blind field study of suvorexant. Following two weeks of baseline, participants received 10 mg suvorexant/placebo for one week and were titrated upward to 20 mg suvorexant/placebo for an additional two weeks. Subjective (diaries) and objective (actigraphy) sleep were monitored throughout. No restrictions were placed on participants' schedules.

Results: Both subjective and objective measures of total sleep time significantly improved in the active vs. the placebo condition, increasing by 2.08 ± 0.47 hours (diary) or 1.04 ± 0.53 hours (actigraphy) by the end of the 10 mg condition, and increasing by 2.97 ± 0.56 hours (diary) or 2.16 ± 0.75 hours (actigraphy) by the end of the 20 mg condition. Physician ratings of change in the severity of symptoms similarly improved in the active group. There were no adverse events reported in the active condition.

Conclusion: Robust changes in total sleep time were observed after administration of suvorexant, a dual-hypocretin antagonist, prior to daytime sleep in a field study of shift workers. The very large changes in total sleep time, coupled with the permissive nature of the therapeutic mechanism (i.e., suppressing wake rather than inducing sleep) indicate that this could be a viable and important therapy for shift workers.

Support: Merck Sharpe and Dohme investigator-initiated study #53236

0782

DURATION OF SUN EXPOSURE IN MEDICAL STUDENTS IMPACTSCHRONOTYPE

Geoca, A.¹ Dowling, M.² Jain, V.³

¹George Washington University School of Medicine and Health Sciences, Washington, DC, ²George Washington University Milken Institute School of Public Health, Washington, DC, ³George Washington University, Washington, DC.

Introduction: Timing of the human sleep-wake cycle is determined by social constraints, biological processes (sleep homeostasis and circadian rhythmicity) and environmental factors, particularly natural and electrical light exposure. However the effect of environmental factors, especially duration of sun exposure, on circadian rhythmicity remains unknown. We aimed to study the relationship between duration of sun exposure and chronotype among medical students.

Methods: Eighty-six GW medical students (62 F [71%], 24 M [29%]; ages 21-33 y [mean 24.4 y]) completed the Munich Chronotype Questionnaire (MCTQ). Mid points, the middle of the participants reported bedtime and wake time on workdays, were used to determine chronotype. Two independent groups based on the mean split (mean = 45) of the distribution of minutes of sun exposure were created: 45 minutes or more (n=31) vs less than 45 minutes (n = 55) of sun exposure. Independent samples t-test was performed to compare the measured midpoint with the following pairs of groups of reported work day sunlight exposure (in minutes): less than 45 min (n = 55) versus 45 min or more (n = 31).

Results: In the total sample (n = 86), no significant association between duration of sun exposure and midpoints was found using Pearson correlation. However, medical students with reported sun exposure of greater than or equal to 45 minutes a day had a significantly earlier chronotype compared to students reporting a sun exposure of less than 45 minutes a day [mid point 2.196 (SD 0.085) versus a mid point 3.386 (SD 1.084); t(69) = 2.021; p = .047].

Conclusion: We found that, in GW medical students, greater amounts of sun exposure during the day was advancing the

circadian rhythm. This may have implications on sleep duration and quality. Support: NA

0783

RELATIONSHIP BETWEEN CHRONOTYPE AND SLEEP DURATION AMONG MEDICAL STUDENTS

Geoca, A.¹ Dowling, M.² Jain, V.³

¹George Washington University School of Medicine and Health Sciences, Washington, DC, ²George Washington University Milken School of Public Health, Washington, DC, ³George Washington University, Washington, DC.

Introduction: Previous literature has supported the claim that longer sleepers have later chronotypes. It is also thought that later chronotypes may obtain less sleep during workdays. We aimed to study the association between sleep duration and chronotypes in The George Washington University (GWU) medical students.

Methods: Eighty-six medical students at GWU (62 F [71%], 24 M [29%]; ages 21-33 y [mean 24.4 y]) filled out the Munich Chronotype Questionnaire (MCTQ). Midpoint of the bedtime and wake times during workdays was used to determine chronotype. Subjects were split into two groupsbased on the median of the distribution (Md=7.5) of the self-reported sleep duration variable; those who sleep less than 7.5 hours (short sleepers), and those who sleep 7.5 hours or longer (long sleepers). Independent samples t-test was used to compare the chronotype measurements of the long sleepers (n=39) versus short sleepers (n=41).

Results: Short sleepers had a mean of 6.48 (SD=0.72) hours of sleep while long sleepers had a mean of 8.11 (SD=0.53) hours of sleep. The range of chronotype measures was wider in the long sleepers (1.25 to 7.25; range=6) compared to that in the short sleepers group (1.42 to 5.280; range=3.86). We found no significant mean differences in chronotype between those who sleep less than 7.5 hours (mean=3.188, SD=0.858) and those who sleep 7.5 hours or longer (mean=3.201, SD=1.20) [t(77)=0.056; p = .956].

Conclusion: Sleep duration among medical students was not associated with their chronotype. This is in opposition to other research findings of decreased sleep duration among later chronotypes. Our findings need to be replicated in a larger sample. **Support:** NA

0784

ASSOCIATIONS OF SLEEP REGULARITY AND CHRONOTYPE WITH HYPERTENSION AMONG AFRICAN AMERICANS IN THE JACKSON HEART SLEEP STUDY

Johnson, D. A.¹ Guo, N.² Redline, S.² ¹Emory University, Atlanta, GA, ²Brigham and Women's Hospital, Boston, MA.

Introduction: Emerging evidence suggests that disparities in sleep regularity, a marker of circadian disruption, contributes to hypertension disparities; however, data among African Americans are limited. We examined associations of sleep regularity and chronotype with hypertension among African Americans in the Jackson Heart Sleep Study (JHSS).

Methods: Participants underwent 7-day actigraphy, completed questionnaires, and had seated blood pressure (BP) measured as part of the JHSS (2012 - 2016). Sleep regularity was defined as the standard deviation (SD) of actigraphy-measured sleep onset timing or sleep duration. Chronotype was assessed by the

Morningness-Eveningness Questionnaire. Prevalent hypertension was defined as either a systolic BP \geq 130 mmHg or diastolic BP \geq 80mmHg, antihypertensive medication use, or self-report of diagnosed hypertension. Multivariable logistic regression models were fit to estimate the prevalence odds ratio (OR) and 95% confidence intervals for the associations of hypertension with sleep regularity measures (SD of sleep onset timing and sleep duration) and chronotype adjusted for covariates.

Results: Participants (n=830) on average were 63.4 years (SD:10.7), mostly female (66.3%) and hypertensive (85.8%). Compared to individuals with sleep onset SD < 30 minutes, higher adjusted odds of hypertension was observed with increasing variability: OR:1.87 (CI:0.99-3.56); OR:2.16 (1.06-4.39), and OR:2.41 (1.12-5.20), for $SD > 30 \& \le 60, > 60 \& \le 90$ and > 90 minutes, respectively. Among non-shift workers, definite morning and evening types compared to intermediates had higher adjusted odds of hypertension, OR:1.71 (1.04-2.83) and OR:2.56 (1.12-5.84), respectively. There were no observed associations for the SD of sleep duration with hypertension. Conclusion: Increased sleep onset variability and extreme chronotypes were associated with prevalent hypertension, supporting interventions targeting sleep hygiene recommendations promoting regular sleep. Future research is needed to understand sleep patterns and risk of cardiovascular disease according to chronotype.

Support: NHLBI K01HL138211 and HL110068-03S1

0785

ASSOCIATIONS BETWEEN CIRCADIAN PREFERENCE AND SLEEP-RELATED THOUGHTS: DATA FROM THE 2015 SLEEP IN AMERICA POLL

Jeon, B.¹ Luyster, F. S.² Chasens, E. R.³

¹University of Pittsburgh, Pittsburgh, PA, ²University of Pittsburgh, School of Nursing, Pittsburgh, PA, ³University of Pittsburgh, School of Nursing, Pittsburgh, PA.

Introduction: Evening types of sleep tend to have poorer sleep quality and sleep habits than morning types. Maladaptive beliefs or thoughts about sleep can affect one's sleep and may differ between evening and morning types. We examined the association between the circadian preference and sleep-related thoughts in U.S adults. Methods: A secondary analysis used survey data from the 2015 National Sleep Foundation's Sleep in America Poll. Questions included normal bedtime and wake-up time for week/work days and weekend/non-work days. Circadian preference was determined by midpoint of sleep calculated as midpoint of sleep on weekends corrected for average nightly sleep duration. Participants were excluded if their sleep midpoint was from noon to midnight. Midpoint of sleep was divided into two groups using median split ("earlier" vs. "later"). Sleep-related thoughts were "worry about getting a good sleep", "overwhelming thoughts about getting enough sleep", "motivation to get sleep", and "concern about serious physical consequences due to poor sleep"; responded often/ always or extremely to somewhat for these items were coded as maladaptive. Logistic regression analysis controlling for sociodemographics, sleep duration, and sleep disturbance (PROMIS Scale; higher scores = greater sleep disturbance) was conducted to examine the relationships between midpoint of sleep and sleeprelated thoughts.

Results: The sample (N = 1011) was primarily White (73.6%), male (50.9%), college educated (62.2%), married/partnered (67.6%) with a mean age of 51.65 ± 17.05 years. Mean midpoint of sleep in "earlier" type was 2:33AM and 5:29AM in "later" type. "Later"

type had shorter sleep duration on weekdays and longer sleep duration on weekends than "earlier" type (p < .01), but average sleep duration was similar between two types. "Later" type had more "worry" and "overwhelming thoughts" (p < .05) about sleep. In logistic models, midpoint of sleep was significant only for "concern" (p = .02).

Conclusion: In this study, late chronotype was associated with increased sleep disturbances and greater variability in sleep duration. The relationship between the timing of sleep and thoughts about the impact of impaired sleep remains unclear and an area for further study with objective measures.

Support:

0786

NON-24 HOUR SLEEP WAKE SYNDROME: A COHORT ANALYSIS

Dalal, L. Yuenan, N. Pogach, M. Thomas, R. J. Beth Israel Deaconess Medical Center, Boston, MA.

Introduction: Although commonly described in the blind population, diagnosis in sighted individuals can be difficult due to perceived rarity and underlying co-morbid conditions. Our objective was to identify the characteristics of N24 rhythm individuals, and responses to varied treatments.

Methods: Patients were identified to have non-24-hour sleep wake syndrome (N24) via history, sleep diaries or digital logs, paired melatonin profiling as well as actigraphy through retrospective chart review at the Beth Israel Deaconess Medical Center, Sleep Disorders Clinic.

Results: 37 patients were identified from 2007 to 2019 with N24 syndrome, BMI of 28, and 67% male. The mean age of onset was within the teenage years (16), and age at diagnosis of 35 years. Paired melatonin profiles (24-hour salivary melatonin, 3-hourly, separated by 7 days, in the subject's own home) showed "movement". Depression and anxiety were seen in 54% and 29% of the co-hort respectively. 75% (28) of the patients had a treatment strategy involving light, and 54% (20) included melatonin. The combination of melatonin and light led to a clinical improvement in 41% of individuals under that regimen (17). Low dose lithium (8 subjects) enhanced melatonin/light responses. A strategy of combining the orexin antagonist suvorexant with melatonin or ramelteon (3 subjects) helped stabilize the circadian rhythm. Tasimelteon treatment has been initiated in 4 subjects.

Conclusion: These data suggest that while comorbid psychiatric conditions are prevalent, a significant proportion of the cohort did not have associated psychiatric disease. Patients reported onset of symptoms in the teenage years, however there was significant delay to diagnosis. Besides light/melatonin, orexin antagonism and low dose lithium may have benefits, but require more systematic assessments. Paired melatonin estimations could be considered as a definitive testing strategy.

Support:

AUTONOMIC DYSFUNCTION AND PHENOCONVERSION IN IDIOPATHIC/ISOLATED REM SLEEP BEHAVIOR DISORDER

*McCarter, S.*¹ *Gehrking, T.*¹ *St. Louis, E.*² *Suarez, M.*¹ *Boeve, B.*² *Silber, M.*² *Low, P.*¹ *Singer, W.*¹

¹Department of Neurology, Mayo Clinic, Rochester, MN, ²Department of Neurology and Sleep Medicine, Mayo Clinic, Rochester, MN.

Introduction: REM-sleep behavior disorder (RBD) is a common finding among patients with synucleinopathies. We aimed to determine the degree of autonomic dysfunction in patients presenting with idiopathic RBD (iRBD), and the predictive value of autonomic dysfunction for phenoconversion to a defined neurodegenerative disease.

Methods: We searched our electronic medical record for patients diagnosed with iRBD who also underwent standardized autonomic function testing within 6 months of iRBD diagnosis, and who had clinical follow-up of at least 3 years following iRBD diagnosis. Patients who received a diagnosis of phenoconversion within 3 months of autonomic testing were excluded. The composite autonomic severity score (CASS) was derived and compared between phenoconverters and non-converters using chi-square and Wilcoxon rank-sum tests.

Results: We identified 18 patients who fulfilled in- and exclusion criteria. Average age at autonomic testing was 67 ± 6.6 years. Twelve (67%) patients phenoconverted during the follow-up period; 6 developed PD, the other 6 DLB. Fifteen (83%) patients had at least mild autonomic dysfunction. There were no significant differences between overall converters and non-converters in total CASS or CASS subscores. However, iRBD patients who developed DLB had significantly higher total and cardiovagal CASS scores compared with those who developed PD (p <0.05), and a trend for higher adrenergic CASS scores compared to those who developed PD and those who did not phenoconvert (p=0.08 for each).

Conclusion: Autonomic dysfunction was seen in 83% of iRBD patients, and more severe baseline cardiovagal and adrenergic autonomic dysfunction in iRBD was associated with phenoconversion to DLB but not PD. Prospective studies are needed to confirm the value of autonomic testing for predicting phenoconversion and disease phenotype in iRBD. **Support:**

0788

INCREASED RAPID EYE MOVEMENT DENSITY IN CHINESE PATIENTS WITH PARKINSON'S DISEASE AND RBD

Zhang, L. Zhu, J.

Nanjing Brain Hospital Affiliated to Nanjing Medical University, Nanjing, CHINA.

Introduction: Impaired rapid eye movement sleep is common among patients with Parkinson's disease (PD). However, information on rapid eye movement density (REM density) among PD patients is currently lacking. The current study sought to characterize REM density in PD patients and to examine the associations between REM density sleep parameters and clinical manifestations. **Methods:** We retrospectively recruited 172 PD patients. All participants were assessed with a two-night polysomnography, and REM density was calculated. Clinical assessments were completed in PD patients before polysomnography. **Results:** Rapid eye movement sleep behavior disorder (RBD) were observed in 93 patients (54.1%). The disease duration, UPDRS part III score, Hoehn and Yahr (H-Y) stage, and HAMA, HAMD, and PDQ-39 scores in the Parkinson's disease patients with rapid eye movement sleep behavior disorder (RBD) were significantly higher than in the patients without RBD (P<0.05). The REM density was also significantly higher in the RBD patients than in the patients without RBD (P<0.05). NREM sleep stage 3 time (N3 time) and percentage of N3 time of total sleep time (N3%) were higher in patients without RBD. The forward binary logistic regression model showed that REM density, UPDRS-III score and N3 sleep time were associated with RBD in the PD patients.

Conclusion: Our results confirm the high prevalence of RBD in patients with PD. Increased REM density was the main risk factor of RBD.

Support: Special Funds of the Jiangsu Provincial Key Research and Development Projects (grant No. BE2018610)

0789

DECREASED SIGMA BAND POWER DURING NREM SLEEP IN REM SLEEP BEHAVIOR DISORDER

LEE, Y^1 Lee, B^2

¹Dept. of Clinical Neurosciences Laboratory, ASAN Medical Center, Seoul, KOREA, REPUBLIC OF, ²Dept. of Psychiatry, Korea University Anam Hospital, Seoul, KOREA, REPUBLIC OF.

Introduction: REM sleep Behavior Disorder (RBD) is characterized by dream enacting behaviors and a loss of atonia during REM sleep. Early detection of RBD is important because it is considered premonitory symptoms neurodegenerative disorders. In this study, we investigated the slow and fast sigma band power of patients with RBD using frequency analysis.

Methods: Twenty patients who were diagnosed as RBD according to the ICSD-3 criteria and 20 age-matched controls who underwent polysomnography (PSG) for other sleep disorders (insomnia, snoring) and showed normal to mild obstructive sleep apnea (OSA). NREM sleep EEG data was extracted and N1 sleep data was excluded to minimize arousal artifact. Fast Fourier transform-based spectral power analysis was used to compute the power spectral densities of the EEG in the MATLAB environment. The sigma bands were divided into 2 discrete bands: slow sigma (11 to 13 Hz) and-fast sigma (13 to 15 Hz). Mann-Whitney U test by SPSS was used.

Results: RBD patients (61.9 ± 7.1 years old; 12 men) had a significantly lower sigma band power than the control group (61.5 ± 1.1 years old; 11 men) in central region (p = 0.028). Particularly, the slow sigma band power showed a bigger difference in all regions except O1 (F3 = 0.017, F4 = 0.027, C3 = 0.004, C4 = 0.009, O2 = 0.017).

Conclusion: Sigma power was lower in the RBD patients than in the control. It suggests that RBD has impaired cortical activity. Thus, decreased spindle activity during NREM sleep may be a potential biomarker of RBD.

Support:

0790

THE CLINICAL SIGNIFICANCE OF HEPCIDIN AS A PREDICTIVE VALUE FOR TREATMENT RESPONSES IN RESTLESS LEGS SYNDROME

Im, H.

Dontan Sacred Heart Hospital, Hallym University Medical Center, Hwaseong, KOREA, REPUBLIC OF.

Introduction: Restless legs syndrome (RLS) is a common sensory motor neurological disorder that is related to iron-dopamine dysregulation and immune system alteration. Hepcidin is the key regulatory hormone of systemic iron homeostasis and is related to inflammatory processes. We aimed to evaluate the clinical utility of hepcidin as a diagnostic biomarker and index of therapeutic responses in RLS patients after dopaminergic treatment.

Methods: Non-anemic and drug-naive RLS patients (n=18) and healthy controls (n=15) were enrolled. Hepcidin (pre-prohepcidin) and iron-related values in serum were measured upon the first visit in both groups and 12 weeks later after dopaminergic treatment in 12 RLS patients. Information about sociodemographic characteristics, sleep-related profiles, mood, and anxiety was obtained upon the first visit in all participants as well as after treatment in RLS patients.

Results: Hepcidin levels exhibited no significant differences between patients with drug-naïve RLS and healthy controls at a diagnosis (7.1 ± 2.4 vs. 7.0 ± 3.2 ng/ml, p = 0.978). Decreased hepcidin levels were significantly associated with decreased RLS severity ($\beta = 0.002$, 95% CI = 0.00–0.00, p = 0.005) and improved quality of life ($\beta = 0.002$, 95% CI = 0.00–7.01, p = 0.044) in a dosedependent manner after 12 weeks of treatment with a dopamine agonist. This association was independent of age, sex, inflammatory markers, sleep quality, insomnia, daytime sleepiness, depression, and anxiety.

Conclusion: This study demonstrates a role of hepcidin as a predictor of therapeutic responses in **RLS** patients.

Support: This work was supported by the Korea Health technology R&D Project through the Korea Health Industry Development Institute, funded by the Ministry of Health & Welfare, South Korea [grant number HI17C2072].

0791

EFFECTS OF LOW FREQUENCY ELECTRICAL STIMULATORS AS NONPHARMACOLOGICAL TREATMENT IN RESTLESS LEGS SYNDROME

Cho, Y.¹ Lee, Y.² Ku, J.² Kim, K.²

¹Keimyung University Dongsan medical center, Daegu, KOREA, REPUBLIC OF, ²Keimyung University School of Medicine, Daegu, KOREA, REPUBLIC OF.

Introduction: Non-pharmacological treatments for restless legs syndrome (RLS) is a treatment option for patients who have not yet started medical treatment, who do not respond to their prescribed medication, or who suffer from adverse effects of medication. This study aims to investigate the clinical effectiveness of low frequency electronic stimulators (LFES) as a non-pharmacological treatment.

Methods: This is a randomized, single-blind study. After screening 64 patients according to the inclusion/exclusion criteria, a total of 46 patients participated in the study. The participants were separated into an active group and a sham group with 22 and 24 members, respectively. The stimulation was administered using the tapping mode (3Hz) present on the machines used, and symptom changes were measured in both groups. The effects of the stimuli were analyzed with repeated measures ANOVA.

Results: Symptom severity was significantly reduced in the active group, and showed significant interaction effects in the time multiple group. Although both the active and sham groups reported improved symptoms upon receiving longer periods of treatment, the effect of the LFES was greater in the active group.

Conclusion: Analyzing the effects of LFES by dividing the active and sham groups revealed that LFES treatment resulted in symptom improvement when using effective stimulation intensity. LFES can be a non-pharmacological treatment option for RLS. **Support:** None

0792

MANDIBULAR MOVEMENT MONITORING WITH ARTIFICIAL INTELLIGENCE ANALYSIS FOR THE DIAGNOSIS OF SLEEP BRUXISM

Martinot, J.^{1,2} Le-Dong, N.³ Cuthbert, V.¹ Denison, S.³ Gozal, D.⁴ Pepin, J.⁵

¹Sleep Laboratory, CHU UCL Namur Site Sainte-Elisabeth, Namur, BELGIUM, ²Institute of Experimental and Clinical Research, UCL Bruxelles Woluwe, Bruxelles, BELGIUM, ³Sunrise, Namur, BELGIUM, ⁴Department of Child Health and Child Health Research Institute, University of Missouri, Columbia, MO, ⁵Université Grenoble Alpes, Inserm, CHU Grenoble Alpes, HP ², Grenoble, FRANCE.

Introduction: Sleep bruxism (BXM) is the result of rhythmic muscular masticatory activity (RMMA) and can be captured by masseters surface electromyography (sEMG). Despite the multiple adverse negative consequences of BXM, a simple reliable home diagnostic device is currently unavailable, with in laboratory audio-video polysomnography (type I PSG) remaining the gold standard diagnostic tool. Mandibular movements (MM) recordings during sleep can readily identify RMMA, are simple to set up and can be easily repeated from night to night. Here, we aimed to identify stereotypical MM in patients with BXM, and to develop RMMA automatic detection and BXM diagnosis using an artificial intelligence-based approach.

Methods: MM were recorded by a dedicated sensor (Sunrise, Namur, Belgium) in 12 patients with BXM during type I PSG. The Sunrise system consists of a coin-sized hardware that is comfortably placed on the subject's chin. Its embedded inertial measurement unit communicates via Bluetooth with a smartphone and automatically transfers MM signals to a cloud-based infrastructure at the end of the night. Data processing and analysis are then performed in Python programming language. A time series cluster analysis was applied to sequences of masseters sEMG and MM signals during BXM episodes (n=300) and during spontaneous micro-arousals (n=300). Then, a convolutional neuronal network (CNN) was developed to identify BXM and distinguish it from spontaneous micro-arousals while exclusively relying on MM signal.

Results: Based on the cluster analysis, BXM periods were characterized by a specific pattern of MM signals (higher frequency and amplitude), which was closely associated with the sEMG signals but clearly differed from the MM signal patterns during microarousals. CNN-based classifier distinguished the BXM events from other RMMAs during micro-arousals and respiratory efforts with an overall accuracy of 91%.

Conclusion: Sleep bruxism can be automatically identified, quantified, and characterized with mandibular movements analysis supported by artificial intelligence technology.

Support: This work was supported by the French National Research Agency (ANR-12-TECS-0010), in the framework of the "Investissements d'avenir" program (ANR-15-IDEX-02). https://life.univ-grenoble-alpes.fr.

NEARLY 25% OF RESTLESS LEGS SYNDROME (RLS) PATIENTS NATIONALLY TREATED WITH DOPAMINE AGONISTS ARE TAKING HIGHER DOSES THAN THE MAXIMUM RECOMMENDED BY FDA AND EXPERT GUIDELINES

Winkelman, J. W.

Massachusetts General Hospital, Boston, MA.

Introduction: Dopamine agonists (DAs) have been first-line therapy for restless legs syndrome (RLS) for 30 years. Long-term DA use is associated with augmentation, a dose-related iatrogenic worsening of RLS, which results in a vicious cycle of escalating DA dosing to manage worsening symptoms. The objective of this study was to investigate rates of high-dose DA prescribing in patients with RLS nationally.

Methods: Patients with a diagnosis of RLS (ICD-10 G255.81), and no diagnosis of Parkinson's disease, treated with rotigotine, pramipexole, and ropinirole, alone or in combination for >180 days, were identified from National Prescription Audit database from October 2017-September 2018. Daily total dosage was grouped into low (FDA-approved) and high (above FDA-approved) dose ranges, respectively (rotigotine: >0- \leq 3mg/>3mg; pramipexole: >0- \leq 0.75mg/>0.75mg; ropinirole: >0- \leq 4mg/>4mg). When DAs were used in combination with each other, dose-equivalent algorithms were used (rotigotine/ropinirole/pramipexole, 1:1:4).

Results: Prescriptions for 244,423 patients meeting inclusion criteria were sampled, constituting 71,181,466 therapy days. For all patients on DA therapy for RLS, 23.5% (57,552/244,423) were prescribed higher than FDA-recommended doses. For patients on DA monotherapy, high-dose prescriptions were provided to 42.7% (819/1919) on rotigotine, 40.7% (38,342/94,275) on pramipexole, and 11.5% (16,900/146,355) on ropinirole. Nearly one-quarter (23.2%) of all pramipexole monotherapy prescriptions were for very high dosages (>1.25 mg). For patients on DA combination therapy, high-dose prescriptions were provided to 79.6% (1,491/1,874).

Conclusion: Roughly 25% of RLS patients treated with DAs were prescribed doses above FDA and expert guideline-recommended maximum doses. Patients on DA combination therapy had substantially higher rates of cumulative high-dose prescriptions. High-dosage DA use is likely due to dose escalation in response to augmentation, risking further augmentation and adverse events such as impulse control disorders. Prescriber education on risks of high-dose DA prescribing for RLS is important.

Support: Arbor Pharmaceuticals, LLC

0794

REDUCTION IN RESTLESS LEGS SYNDROME SYMPTOMS WITH NON-INVASIVE PERIPHERAL NERVE STIMULATION

*Charlesworth, J. D.*¹ *Baker, F. C.*² *Kolotovska, V.*³ *Adlou, B.*¹ *de Zambotti, M.*² *Ismail, M.*² *Raghunathan, S.*¹ *Singh, H.*³ *Buchfuhrer, M. J.*⁴

¹Noctrix Health, Inc., San Francisco, CA, ²SRI International Human Sleep Research Lab, Menlo Park, CA, ³Sleep Medicine Specialists of California, San Ramon, CA, ⁴Stanford University School of Medicine, Stanford, CA.

Introduction: Restless Legs Syndrome (RLS) is a sensorimotor neurological condition characterized by an uncontrollable urge to move the legs that interferes with falling and staying asleep. For the over 5 million Americans with clinically significant RLS, these

symptoms occur multiple nights per week, significantly impair quality of life, increase the prevalence of depression and anxiety, and increase suicide risk. FDA-approved medications for RLS are associated with progressively worsening RLS symptoms and numerous adverse events, whereas existing medical device treatments have limited efficacy.

Methods: We evaluated a novel neurostimulation intervention for RLS developed by Noctrix Health; electrical stimulation was applied non-invasively and bilaterally to the peroneal nerve of patients with moderate-to-severe primary RLS. Stimulation parameters were engineered to maximize therapeutic efficacy while minimizing interference with sleep. To assess the therapeutic efficacy of this technique, we conducted a multi-site randomized patient-blinded crossover trial comparing active neurostimulation treatment to a sham device. Following a lab visit for calibration, optimization, and training, each patient was instructed to self-administer each treatment - active and sham - for 14 consecutive nights at home.

Results: Active neurostimulation treatment resulted in a clinically significant reduction in RLS severity of 4.2 points on the International RLS Rating Scale (IRLS) relative to sham (P<0.01), comparable to FDA-approved medications. Moreover, 79% of patients demonstrated a clinically significant improvement on the Clinical Global Impressions-Improvement scale (CGI-I) compared to 7% for sham (P<0.01).

Conclusion: To our knowledge, this is the first sham-controlled study demonstrating a clinically significant reduction in RLS severity resulting from a non-pharmacological intervention. This therapeutic effect was sustained over 2-weeks of in-home patient-administered usage, indicating consistent efficacy. A medical device based on this technology could be a promising alternative or complement to medications.

Support: Funding was provided by Noctrix Health, Inc.

0795

THE RELATIONSHIP BETWEEN RESTLESS LEGS SYNDROME AND HYPOTHYROIDISM

Ahmed, N. Kandil, M. Elfil, M. Jamal, A. Koo, B. Yale University, New Haven, CT.

Introduction: The diurnal nature of RLS and its response to dopamine hint that hormones may be central in RLS pathophysiology. Hypothyroidism has been linked to RLS, but studies are few and limited in scope. We sought to determine whether restless legs syndrome (RLS) is more prevalent in persons with hypothyroidism and whether hypothyroidism is more prevalent in persons with RLS.

Methods: Persons with hypothyroidism and controls without hypothyroidism were recruited through Research Match, an on-line registry of potential clinical research participants, and assessed for RLS using the Cambridge Hopkins questionnaire. Persons with RLS and controls without RLS were recruited through the RLS Foundation and Research Match and assessed for hypothyroidism by self-report of physician diagnosis. RLS severity was assessed using the International RLS Study Group Severity Scale and cause of hypothyroidism was assessed by self-report.

Results: 266 hypothyroid subjects and 321 controls were comparable in age (52.3 ± 13.4 vs. 53.9 ± 11.7 ; p=0.14) and gender (91.7% vs. 91.3% women; p=0.85). 354 RLS and 313 control subjects were comparable in age (59.1 ± 13.2 vs. 58.2 ± 13.6 ; p=0.41) and gender (80.8% vs. 78.3%; p=0.42). Hypothyroid participants compared to controls had significantly higher RLS prevalence (14.3% vs. 8.1%; p=0.02). RLS participants compared to non-RLS controls had significantly higher hypothyroidism prevalence (22.3% vs.

13.8%; p=0.005). RLS severity was similar in persons with and those without hypothyroidism. Among 73 persons with RLS and comorbid hypothyroidism, 14 previously were hyperthyroid compared to 0 out of 37 persons with hypothyroidism without RLS (p=0.004).

Conclusion: RLS prevalence is increased in individuals with hypothyroidism prevalence is increased in individuals with RLS. Persons with comorbid hypothyroidism and RLS are significantly more likely than those with hypothyroidism alone to have had hyperthyroidism prior to hypothyroidism. The association between RLS and thyroid disease is likely to shed light on the complex biological mechanisms underlying RLS pathophysiology. **Support:** None

0796

DIPYRIDAMOLE FOR THE TREATMENT OF RLS: PRELIMINARY RESULTS OF A CROSS-OVER, PLACEBO-CONTROLLED, DOUBLE-BLIND TRIAL

Garcia-Borreguero, D.¹ Granizo, J.¹ Ferre, S.² ¹Sleep Research Institute, Madrid, SPAIN, ²Integrative Neurobiology Section, NIDA, NIH, Baltimore, MD.

Introduction: Recent animal models of restless legs syndrome (RLS) suggest that brain iron deficiency is associated with a hypoadenosinergic state, with downregulation of adenosine A_1 receptors (A1R) in the striatum. Dipyridamole is a non-selective inhibitor of ENT1/ENT2 (the main reuptake mechanism of adenosine) and increases thereby extracellular adenosine. We hypothesized that treatment with dipyridamole would improve RLS symptoms more than placebo.

Methods: We performed a randomized, double-blind, placebocontrolled clinical trial on 15 previously untreated idiopathic RLS patients that underwent a two week treatment with either 300 mg dipyridamol or placebo (randomized for the order of treatments), following a cross-over design. Treatment started with a dose of 100 mg/day, followed by a forced up-titration to 200 mg on day 4, and to 300 mg on day 7. Severity was assessed at baseline, day 7 and day 14 of each treatment condition by means of the IRLS and CGI scales. The primary endpoint was therapeutic response (50% improvement in IRLS total score).

Results: Fifteen patients (nine women), never before treated with dopaminergic agents, completed both arms of the study. Mean age was 60,2 (\pm 7,1), and the mean duration of illness was 6,03 (\pm 3,1) yrs. IRLS score improved during treatment with dipyridamole from a mean (\pm S.D.) of 24,36 (\pm 3,3) to 9,78 (\pm 3,4), in contrast to a change from 23,9 (\pm 3,8) to 16,6 (\pm 3,7) under placebo (p< 0.05). Corresponding improvements on the CGI were 61,3% and 11,8% for dipyridamole and placebo, respectively. 7/ 15 patients improved by more than 50% during treatment with dipyridamole vs. 4/15 under placebo. Main side effects were abdominal cramps, diarrhea, dizziness, and flushing.

Conclusion: These preliminary results suggest that dipyridamole is an effective agent on RLS symptoms. It also provides evidence of hypoadenosinergic mechanisms playing a central role in RLS. **Support:** No funding was provided for this investigation.

0797

RESTLESS LEGS SYNDROME AND PERCEIVED OLFACTORY AND TASTE DYSFUNCTION: A COMMUNITY-BASED STUDY

Zhuang, S.¹ Yuan, X.² Ma, C.³ Yang, N.² Liu, C.¹ Na, M.³ Winkelman, J. W.⁴ Wu, S.² Gao, X.³ ¹The Second Affiliated Hospital of Soochow University, Suzhou, CHINA, ²Kailuan General Hospital, Tangshan, CHINA, ³The Pennsylvania State University, University Park, PA, ⁴Massachusetta General Hagnital and Harward Medical School

⁴Massachusetts General Hospital and Harvard Medical School, Boston, MA.

Introduction: Restless legs syndrome (RLS), an under-recognized sensorimotor condition worldwide, is thought to be a prodromal symptom of Parkinson's disease as suggested by previous evidences. However, its association with prodromal chemosensory impairments, including olfactory or taste dysfunction, has remained largely unknown. Few studies of small sample sizes were conducted in predominantly Caucasian populations and results were inconsistent.

Methods: We performed a cross-sectional analysis including 90,337 Chinese adults free of neurodegenerative diseases in the Kailuan study in 2016. Presence of RLS was defined using revised RLS diagnostic criteria and further verified by Cambridge-Hopkins questionnaire for RLS. Perceived olfactory and taste dysfunction was collected via a questionnaire. The association between RLS and perceived olfactory and taste dysfunction was assessed using logistic regression model, adjusting for potential cofounders such as age, sex, smoking, alcohol consumption sleep conditions and medical history.

Results: RLS was associated with high odds of having perceived olfactory and/or taste dysfunction (adjusted odds ratio =5.92; 95% confidence interval, 3.11, 11.3). The significant association persisted when using Cambridge-Hopkins questionnaire for RLS (adjusted odds ratio =5.55; 95% confidence interval, 2.37-13.0) or when excluding participants with major chronic diseases.

Conclusion: RLS was associated with increased odds of perceived olfactory and taste dysfunction.

Support: This research was supported by start-up grant from the College of Health and Human Development and the Department of Nutritional Sciences, Pennsylvania State University, the Institute for CyberScience Seed Grant Program, Pennsylvania State University, and Natural Science Foundation of Hebei Province (H2018209318).

0798

"UNCOVERING MY FEET ALLEVIATES MY RESTLESS LEGS": IMPACT OF DIFFERENT ACTIVITIES AND CONDITIONS ON RESTLESS LEGS SYMPTOMS

Karroum, E. G.¹ Leu-Semenescu, S.^{2,3} Amdur, R.⁴ Arnulf, I.^{2,3} ¹Department of Neurology, The George Washington University School of Medicine and Health Sciences, Washington, DC, ²Sleep Disorders Unit, Pitié-Salpêtrière Charles Foix University Hospital, APHP, Paris, FRANCE, ³Sorbonne University, Paris, FRANCE, ⁴Department of Surgery, The George Washington University School of Medicine and Health Sciences, Washington, DC.

Introduction: The restless legs syndrome (RLS) is a resting wake state disorder with inactivity/decreased movement as an aggravating factor and activity/increased movement as an alleviating factor. Other activities and conditions may impact RLS symptoms but have not been systematically studied.

Methods: Fifty-six patients with primary severe RLS (age: 64.1 ± 11.3 ; 66% women) responded about the effect of 20 activities/conditions on their RLS symptoms. Responses were assigned a numerical value: Aggravation (-1), No effect/Don't know (0), Alleviation (+1), with calculating a mean effect score for each

activity/condition and using a sign test to determine if that score was significantly above or below zero (no effect). Responses were further analyzed based on age, age at RLS onset, duration of RLS, RLS severity, gender, Familial/Non-familial RLS, and Painful/Non-Painful RLS. Association of continuous variables and categorical variables with each activity/condition was examined using Spearman correlation test and Fisher exact test, respectively. Bonferroni p threshold was set at p=0.00036.

Results: Activities/conditions with significant (p<0.0001) positive mean effect scores were: Feet uncovering (0.70); Leg massaging (0.63); Cold showers (0.54); and Manual activities (0.46). Activities with significant negative mean effect scores were: Vehicle passenger (-0.80); Show attendance (-0.70); Bedsheets weight on legs (-0.57); Watching TV (-0.54); High ambient temperature (-0.45); During meals (-0.39) (all p<0.0001); and Bedsheets rubbing on legs (-0.34; p=0.0002). Activities/conditions with no significant (all p>0.00036) mean effect scores were: Driving (0.00); Gambling (0.02); Professional activities (0.13); Hot showers (0.13); Using computer (0.14); Low ambient temperature (0.21); Sexual activities (0.27); Mental activities (0.29); and Sports activities (0.34). There was no significant association between each activity/condition and age, age at RLS onset, duration of RLS, RLS severity, gender, Familial/Non-familial RLS, or Painful/Non-Painful RLS.

Conclusion: There is a wide range of impact of different activities/ conditions on RLS symptoms. These could be further considered in the non-pharmacological treatment or prevention of RLS symptoms.

Support: This study was not funded.

0799

A POPULATION-BASED STUDY OF ADULT PATIENTS WITH RECURRENT, CLINICIAN DIAGNOSED SLEEPWALKING AND/OR SLEEP RELATED EATING DISORDER

Krahn, L. E.¹ Tashman, Y. S.¹ Lyng, P. J.¹ Lloyd, R. M.² Silber, M. H.²

¹Mayo Clinic, Scottsdale, AZ, ²Mayo Clinic, Rochester, MN.

Introduction: The ICSD-3 describes NREM-related parasomnias as abnormal sleep related complex movements where motor behavior occurs in the setting of absent or very minimal higher cognitive function. A population-based twin study reported that 3.9% of men and 3.1 % of women sleepwalk (SW) with 30% reported experiencing injury (Hublin 1997). Sleep related eating disorder (SRED) may be a variant of SW with a reported prevalence of 0.5-5 % (Michalska 2016). This study examined SW and SRED in a population-based sample permitting review of associated features, risk factors and outcomes.

Methods: The records-linkage system of the Rochester Epidemiology Project that includes all adults residing in Olmsted County MN was searched for documentation of patients seeking care for at least two episodes of sleepwalking and/or sleep eating. Records from 2007-2016 were included.

Results: 56 cases were identified with 50 (89 %) having SW and 16 (29%) SRED. The gender breakdown was 50% male and 50% female with the mean age at diagnosis of 40 (SD 13). Childhood parasomnia events were reported by 23/36 (62%) and a family history in 21/35 (64%). Data were unavailable for the others. The number of parasomnia incidents was \geq 10 for 21 (37%). A variety of experiences were documented, including leaving the bedroom (39%), injury (25%), and exiting through a window/balcony (5%). Associated factors were sleep deprivation (52%), untreated

obstructive sleep apnea (34%), antidepressant use (41%), zolpidem use (18%), circadian disruption (14%) and alcohol use (9%). Polysomnography was conducted for 41 (74%). Documented interventions were medication discontinuation (18%) and starting a benzodiazepine (16%). In 21%, no treatment was given.

Conclusion: This population-based study of adults with clinician diagnosed recurrent SW and SRED revealed equal rates in men and women. The majority of patients with SRED also had SW. 25% of cases were associated with injuries.

Support: Rochester Epidemiology Project supported by NIH R01 AG034676 and AG052425 and the Mayo Foundation.

0800

POSTTRAUMATIC STRESS DISORDER AND REM SLEEP WITHOUT ATONIA IN VETERANS

Lee, E.¹ Yoon, I.² Choi, H.¹

¹Veteran Health Service Medical Center, Seoul, KOREA, REPUBLIC OF, ²Seoul National University Bundang Hospital, Seongnam, Gyeonggi-do, KOREA, REPUBLIC OF.

Introduction: In veterans, the prevalence of rapid eye movement (REM) sleep behavior disorder(RBD) is higher than the general population, and there is some evidence that this is related to posttraumatic stress disorder(PTSD). In addition, trauma related nightmares (TRNs) interfere with REM sleep and are often accompanied by motor activity. (rem sleep without atonia; RSWA). The purpose of this study is to determine whether the frequency of dream enactment behavior(DEB) and RSWA is different according to the presence of PTSD or trauma.

Methods: The patients (n = 2262) who underwent video assisted polysomnography (PSG) and sleep-related questionnaire surveys at Veteran Health Service Medical Center in Republic of Korea were reviewed retrospectively and cross-sectionally. Based on patients diagnosed with PTSD (N = 20; 100% male; 67.9 \pm 8.5 years of age), those exposed to trauma but not diagnosed with PTSD (N = 23; 100% male; age 64.0 \pm 13.4) and trauma unexposed controls (N = 21; 100% male; age 59.86 \pm 10.9) were matched.

Results: In the PTSD group, patients who reported self-reported DEB tended to be more than the traumatic exposure group and the control group (P = 0.022). In-lab video assisted PSG showed no differences in DEB between the three groups, but RSWA. (p = 0.026) After adjusting for age, hypnotics, apnea hypopnea index (AHI), Beck depression inventory (BDI), and periodic limb movement (PLM) arousal factors, RSWA was significantly higher in the PTSD group than in the traumatic exposure group. (p = 0.006)

Conclusion: The result that RSWA was significantly higher in the PTSD group than in the traumatic exposure group suggests that there may be an associated pathophysiology between PTSD and RBD. Longitudinal studies are needed to establish the link between RBD with PTSD and neurodegenerative diseases associated with synucleinopathy.

Support: This study was supported by a VHS Medical Center Research Grant, Republic of Korea. (grant number: VHSMC 19033)

0801

SLEEP, NIGHTMARES, AND THE MAINTENANCE OF POSTTRAUMA SYMPTOMS

Meysing, A. Schulte, M. Youngren, W. Hamilton, N. University of Kansas, Lawrence, KS.

Introduction: Most individuals will experience at least one traumatic event in their lifetime. Although most individuals who experience a trauma will exhibit some posttrauma symptoms, only a small subset will experience long-lasting symptoms. In fact, most research suggests that posttrauma symptoms will gradually reduce over time. However, some individuals can exhibit posttrauma symptoms for an extended period of time. Although research has demonstrated that poor overall sleep quality may explain why some people have trouble gradually recovering from posttrauma symptoms, research has vet to examine specific aspects of sleep that can impair the gradual remission of posttrauma symptoms. This study examined how individual facets of sleep quality (such as nightmares and sleep duration) impact posttrauma symptoms over time. Methods: 944 college students completed an online survey battery that included measures of traumatic experiences, time since trauma (TST), posttrauma symptoms, sleep quality, and the presence of nightmares. Regression analyses were used to examine the interaction of sleep quality sub-facets and time since trauma on posttrauma nightmares (PNMs).

Results: Out of 944 participants, 637 (67%) reported at least one trauma. Of those students, time since trauma (TST) and all other sleep variables significantly predicted posttrauma symptoms (p < 0.05). However, the only significant interaction was nightmares and TST (p < 0.01) where individuals who experienced PNMs had significantly (p < 0.01) higher posttrauma symptom intercept (48.19) than individuals who did not experience PNMs (31.19). Furthermore, individuals who experienced PNMs demonstrated statistically significant flatter slopes than those without nightmares (p < 0.01).

Conclusion: The results reveal that time since trauma predicts a decrease in posttrauma symptoms, whereas nightmares impede this symptom reduction. Interestingly, the interaction finding suggests that nightmares play a critical role in initial symptom expression and recovery. These results demonstrate the importance of identifying and treating nightmares immediately following a trauma.

Support: "none"

0802

TO EXAMINE THE EFFECT OF GABAPENTIN ENACARBIL IN PRIMARY RESTLESS LEGS SYNDROME PATIENTS WHO ARE ON DOPAMINERGIC AGENTS AND **EXHIBITING AUGMENTATION**

Bollu, P.¹ Goyal, M.² Sivaraman, M.³ Taylor, N.³ Yin, L.³ Thakkar, M.² Sahota, P.³

¹University of Missouri, Columbia, MO, ²Harry S Truman VA Hospital, Columbia, MO, ³University of Missouri Health Care, Columbia, MO.

Introduction: Augmentation is defined as worsening of the symptoms of Restless Legs Syndrome after a brief period of initial improvement with dopaminergic agents resulting in either an earlier onset, increase in severity, quicker onset, spread to other body parts. The exact prevalence of this phenomenon is not known and in patients experiencing augmentation, it can pose a difficult diagnostic and therapeutic challenge to the clinician. In our study, we found extended-release gabapentin to be an effective intervention in patients experiencing dopaminergic augmentation

Methods: This is an open-label single-arm study done in patients exhibiting augmentation while on dopaminergic agents. Patients who were enrolled in the study were initiated on oral extendedrelease gabapentin(Horizont) 600 mg at 5 pm at the beginning of the study. At day 90, attempts were made to reduce or discontinue dopaminergic agents. International Restless Legs Syndrome-Rating Scale (IRLS) and Augmentation Severity Rating Scale(ASRS) were recorded at each visit.

Results: A total of 10 patients were enrolled in the study while only 8 patients completed it. Compared to the baseline (visit 2), there is a significant improvement in both the augmentation severity(p= 0.0131) and the IRLS (p=0.0497). Wilcoxon matched-pairs signed rank test was used for statistical analysis.

Conclusion: Extended-release Gabapentin is an effective treatment option in primary RLS patients experiencing augmentation secondary to dopaminergic medication usage.

Support: The study is funded and medication is provided by Arbor Pharmaceuticals.

0803

CLINICAL AND POLYSOMNOGRAPHIC FEATURES OF TRAUMA ASSOCIATED SLEEP DISORDER (TASD)

Sanchez, H. O.¹ Mysliwiec, V.¹ Froese, R. E.¹ Creamer, J. L.¹ Matsangas, P.² Foster, S. N.¹ Hansen, S. L.¹ Brock, M. S.¹ ¹Department of Sleep Medicine, San Antonio Uniformed Services Health Education Consortium, JBSA-Lackland AFB, TX, ²Operations Research Department, Naval Postgraduate School, Monterey, CA.

Introduction: Trauma associated sleep disorder (TASD) is an emerging parasomnia that develops after trauma with clinical features of trauma related nightmares (TRN), disruptive nocturnal behaviors (DNB), and autonomic disturbances. The purpose of this study is to characterize the clinical and polysomnographic (PSG) features of TASD.

Methods: Clinical history and detailed video-PSG review, to include post-PSG nightmare reports, of a cohort of patients with TASD.

Results: Patients (n=40, 32 men, 8 women) were active duty service members with a median age of 38.9 yrs (range 24-57 yrs). Dream content typically related to combat (n=37, 92.5%), with 75% (n=30) reporting TRN and 60% (n=24) reporting dream enactment at least weekly. Self-reported DNB included vocalizations, violent limb movements, thrashing, defensive posturing, and jumping from bed. There was a high rate of comorbid insomnia (n=35, 87.5%), PTSD (n=23, 63.9%), anxiety (n=25, 62.5%), depression (n=20, 50%), OSA (n=19, 47.5%), chronic pain (n=12, 30%), and TBI (n=13, 32.5%). Most patients had REM sleep without atonia (RSWA) (n=33, 82.5%), though a minority had purposeful DNB (n=11, 27.5%). Vocalizations were present in seven (17.5%) patients. Patients with PTSD were more likely to have purposeful DNB (n=9, 100%) than those without PTSD (n=13, 50%; p=0.013), whereas patients with purposeful DNB had markedly less N3 sleep $(1.0\pm11.4\%)$ than those without purposeful DNB ($13.8\pm16.2\%$; p=0.002). There was no significant difference in medications between DNB groups.

Conclusion: TASD is frequently comorbid with other sleep and behavioral health disorders. Characteristics of TASD are often captured on video-PSG during REM sleep, though DNB may be less pronounced than what patients report in their habitual sleeping environment. Clinical and video-PSG correlations are invaluable in assessing patients with TASD. This study, which is the largest cohort to date, provides a further basis for establishing TASD as a unique REM-related parasomnia.

Support: N/A

SLEEP BRUXISM, AWAKE BRUXISM AND SLEEP RELATED BREATHING DISORDERS IN ADULTS WITH DOWN SYNDROME

Giannasi, L.¹ Meira e Cruz, M.² Rezende, T.¹ Dutra, M.³ Nacif, S.⁴ Oliveira, E.⁴ Oliveira, L.⁵ Oliveira, W.⁶ Rode, S.¹ Nazário, L.⁶ Silvestre, P.⁶ Bacigalupo, E.⁶ Amorim, J.⁶ Salgado, M.⁶ Gomes, M.⁶ ¹COAT - Institute of Science and Technology, São José dos Campos, BRAZIL, ²Sleep Unit, Cardiovascular Center of University of Lisbon, Lisbon School of Medicine, Lisbon, PORTUGAL, ³CEBAPE - Institute of Science and Technology, CEBAPE - Institute of Science and Technology, BRAZIL, ⁴Hospital do Servidor Público Estadual de São Paulo, São Paulo, BRAZIL, ⁵University Center of Anápolis-UniEnvagélica, Goiás, BRAZIL, ⁶CEBAPE - Institute of Science and Technology, São José dos Campos, BRAZIL.

Introduction: To our knowledge, no studies have accessed theawake bruxism (AB) and stage by stage sleep bruxism (SB) in adults with Down syndrome. Recent works have shown that portable PSG systems are accurate for SB assessment even in the absence of audiovideo recording. We aimed to evaluate the prevalence of awake bruxism, stage-by-stage sleep bruxism and Sleep Related Breathing Disorders (SRBD) in adults with Down syndrome.

Methods: Twenty-three adults with Down Syndrome (DS) were enrolled in this study. General health, dental status, parafunctional habits and temporomandibular symptoms were assessed. The history of SB/AB was taken from a questionnaire to the caregivers. A portable PSG type II system (Embla Embletta MPR+PG ST+Proxy, Natus, California-USA) was used to perform a fullsleep study at patients' home. RMMA activity was defined as low (>1 and <2 episodes/h of sleep), moderate (>2 and <4 episodes/h of sleep), or high (>4 episodes/h of sleep). PSG diagnose of SB was assumed if RMMA index was >2 episodes/h of sleep.

Results: According to caregiver's report, AB was present in all patients whereas only 13.1% had SB. PSG records showed a SB prevalence of 91.3%, with a mean RMMA index $40.0\pm30.0/h$. Only 2 (8,7%) showed RMMA index of 0.0/h. SB episodes were predominant in N3 and REM sleep stage in 14 and 9 patients, respectively. All but one (95,7%) patient (isolated snoring) presented with OSA (AHI= 32.8 ± 28.6). A unique TMD symptom (pain on palpation) was present in 8,7% of the global sample.

Conclusion: The high prevalence of "definitive SB" together with the high prevalence of OSA and snoring point in favor to the recommendation of routine PSG in adults with DS. Furthermore, the low sensitivity of parent-oriented questionnaires reinforces the need of more accurate assessment tools in order to get a better standard of care in this particular group of patients.

Support: State of Sao Paulo Research Support Foundation - FAPESP grant number: 2017/06835-8

0805

LACK OF ASSOCIATION BETWEEN PERIODIC LIMB MOVEMENTS OF SLEEP AND NEUROIMAGING SIGNATURES OF CEREBRAL SMALL VESSEL DISEASE IN STROKE-FREE COMMUNITY-DWELLING OLDER ADULTS

Slota, K. A.¹ Castillo, P.¹ Mera, R. M.² Del Brutto, V. J.³ Del Brutto, O. H.⁴ ¹Mayo Clinic, Jacksonville, FL, ²Gilead Sciences, Foster City, CA, ³University of Miami, Miami, FL, ⁴Universidad Espiritu Santo, Guayaquil, ECUADOR.

Introduction: Evidence of the relationship between periodic limb movements during sleep (PLMS) and cerebral small vessel disease (cSVD) is limited and inconsistent. Here, we aimed to assess the independent association between PLMS and the different neuroimaging signatures of cSVD.

Methods: Community dwelling adults aged ≥ 60 years enrolled in the Atahualpa Project undergoing PSG and MRI with time intervals ≤ 6 months were included. MRI readings focused on white matter hyperintensities (WMH) of presumed vascular origin, deep cerebral microbleeds (CMB), silent lacunar infarcts (LI), and >10enlarged basal ganglia-perivascular spaces (BG-PVS). Data from single-night polysomnograms were interpreted according to recommendations of the American Academy of Sleep Medicine. Associations between the PLMS index and neuroimaging signatures of cSVD (as dependent variables) were assessed by means of logistic regression models, adjusted for relevant confounders.

Results: A total of 146 individuals (mean age: 71.4 \pm 7.5 years; 64% women) were included. A PLMS index \geq 15/hour were noted in 48 (33%) participants. Moderate-to-severe WMH were present in 33 individuals (23%), deep CMB in 9 (6%), silent LI in 16 (11%), and >10 BG-PVS in 44 (30%). In univariate analyses, silent LI (*p*=0.035) and the presence of >10 enlarged BG-PVS (*p*=0.034) were significantly higher among participants with a PLMS index \geq 15/hour. However, fully-adjusted multivariate models showed no significant association between PLMS index \geq 15/hour and any of the neuroimaging signatures of cSVD.

Conclusion: Conclusions: This study shows no independent association between the PLMS index and neuroimaging signatures of cSVD in stroke-free community-dwelling older adults.

Support: This study was supported by Universidad Espíritu Santo - Ecuador.

0806

PRESCRIPTION CORRELATES OF NIGHTMARE DISORDER AMONG VETERANS

Gross, M.¹ Patel, R.^{1,2} Schwartz, S. W.¹ Sebastião, Y. V.³ Foulis, P.^{1,2} Scheer, D.^{1,4} Taylor, K. A.^{1,5} Anderson, W.^{1,2} ¹University of South Florida, College of Public Health, Tampa, FL, ²James A. Haley Veteran's Hospital, Tampa, FL, ³Nationwide Children's Hospital, Columbus, OH, ⁴Biotech Research Group Inc., Tampa, FL, ⁵Gannon University, Ruskin, FL.

Introduction: In the James A. Haley Veterans Administration (JAHVA) Vista database, the ICD-9 code 307.47 for Nightmare disorder (ND) is infrequently used and appears independently of codes for PTSD. We wanted to determine if certain drugs that may affect sleep are associated with ND.

Methods: All patients with ND visiting JAHVA between 2007 and 2011 were selected along with control patients who visited JAHVA on one of 20 random days, one day each quarter year. Controls were assigned an index date reflecting their selection quarter. Associations with prescriptions for opioids, antidepressants (SSRI's, SSNI's, Tricyclics), antihistamines and benzodiazepine/Z-drugs were initially investigated. Two analyses were performed: risk factor analysis- patients with ND diagnosis dates (cases) or index dates (controls) prior to 2008 were excluded and only prescription dates that preceded the ND diagnosis or index date were considered; treatment analysis- cases and controls with a ND diagnosis

date or index date after 2010 were excluded and only prescription dates that were subsequent to the ND diagnosis or index date respectively were considered. Logistic regression adjusting for age, gender, race and Hispanic ethnicity was used to determine the association between drug groups and ND.

Results: In risk factor analysis (667 cases, 14,739 controls), opioids and antihistamines were significantly less prevalent among would-be ND patients than controls (OR=0.627 and 0.610 respectively); no drug group was predictive of ND. In contrast, all drug groups were significantly associated with ND in treatment analysis (803 cases, 15,530 controls). The strongest associations were seen with benzodiazepine (OR=3.026; 95% CI: 2.472, 3.703) and SSRI (OR=2.789; 95% CI=2.316, 3.358) prescriptions.

Conclusion: Our data suggest that some JAHVA providers may be treating ND with medication, most notably with benzodiazepines/ Z-drugs and antidepressants. The role of anti-histamine and opioid prescriptions needs further elucidation. The ramifications of these treatment decisions should be explored.

Support: This material is the result of work supported with resources and the use of facilities at the James A. Haley Veterans' Hospital.

0807

NREM PARASOMNIAS: RETROSPECTIVE ANALYSIS OF TREATMENT AND OUTCOMES

*Limbekar, N.*¹ *Pham, J.*² *Yusuf, H.*² *Budhiraja, R.*² *Javaheri, S.*² *Epstein, L. J.*² *Pavlova, M.*²

¹Brigham and Women's Hospital & Massachusetts General Hospital, Boston, MA, ²Brigham and Women's Hospital, Boston, MA.

Introduction: NREM parasomnias are relatively common among children and sometimes persist in adulthood. These behaviors may result in injury or have negative impacts on functioning and quality of life thus necessitating treatment. The treatment is challenging given the lack of evidence for frequently used medications such as benzodiazepines (BDZ) or tricyclic antidepressants (TCA). The aim of this retrospective analysis is to determine the most frequently prescribed medications for treatment of NREM parasomnias and evaluate reported outcomes.

Methods: We performed a retrospective chart review of all patients with NREM parasomnia diagnosed within BWH clinics examining the date of diagnosis, date of starting therapy, comorbidities, type of medication prescribed, and the reported change in symptoms or side effects at the individual's follow-up visits.

Results: From 2012 to 2019, 123 patients (64 females, 59 male) at BWH clinics received the diagnosis of NREM parasomnia, including sleepwalking and night terrors. Mean age was 44. Comorbidities included depression=16, anxiety=32, seizures=6, RLS=9, epilepsy=5, insomnia=29, and OSA=57. Initial treatment included safety counseling (72), BDZ (7), TCA (4), and treatment of comorbidity (23). Treatment of OSA only (n=15) was effective in 66% (n=10) and 33% were lost to follow up. Of those with OSA treatment plus BDZ (n=6), treatment was effective in 50% (n=3). Of those receiving BDZ only (n=7), treatment was effective in 43%. Of those receiving Melatonin (8),treatment was effective among 62.5% (n=5). TCAS (n=4) were effective in 3 patients (75%). Treatment of comorbid conditions without pharmacotherapy (23) was effective in 35% (n=8) while the remaining 65% (n=15) were lost to follow up.

Conclusion: Treating comorbid conditions such as OSA, insomnia, RLS, depression, and anxiety is a frequent treatment strategy.

Additional pharmacologic treatment most commonly includes melatonin, BDZs, and TCAs. **Support:** None

0808

HIGH DENSITY EEG CORRELATES OF NREM SLEEP PARASOMNIA EPISODES

Valomon, A.¹ Nakamura, K. P.² De Cuntis, I.¹ Kummerow, E.¹ Bazalakova, M.² Plante, D. T.¹ Riedner, B. A.¹ Jones, S. G.¹ Tononi, G.¹ Boly, M.²

¹UW Madison - Department of Psychiatry, Madison, WI, ²UW Madison - Department of Neurology, Madison, WI, ³UW Madison - Department of Neurology, Madison, WI, ⁴UW Madison - Department of Psychiatry, Madison, WI.

Introduction: Parasomnia episodes (PE) consist of abnormal behaviors during sleep. Using high-density EEG (HDEEG), we sought to quantify topographical differences in spectral power during PE in comparison to wake and sleep.

Methods: 17 adult subjects with a history of NREM sleep parasomnia underwent 256-electrode HDEEG recordings during recovery sleep after 25h of sleep deprivation. PE occurred either spontaneously or when triggered by a sound. Data preprocessing of PE, sleep and wake data included filtering at 1-25 Hz, careful epoch and channel selection, and adaptive mixture independent component analysis (AMICA). We compared topographies of delta (slow wave activity, or SWA) and theta power, alpha power, and beta/delta ratio (a marker of cortical arousal) between states using paired t-tests. All results were thresholded at p<0.05 corrected for multiple comparison using statistical non parametric mapping (SNPM).

Results: Clean data were obtained in 26 PE arising out of N2/N3 sleep in 11 subjects. During PE, delta and theta power were significantly higher than during wake but lower than during sleep in central regions (at uncorrected p<0.05 for sleep vs. PE delta power). Occipital alpha was lower during PE compared to wake, but higher during PE compared to sleep. Finally, beta/delta ratio values during PE were globally higher than in wake, but globally lower than during sleep.

Conclusion: The present results confirm and extend our previous findings of decreased SWA in central areas during baseline sleep in patients with NREM sleep PE. They suggest that higher cortical arousal in central regions may precipitate motor behaviors during PE. Alpha power and beta-delta ratio during PE were intermediate between sleep and wake, suggesting that PE are transitional states with an admixture of cortical arousal and cortical sleep. Future analyses will use source reconstruction to identify the cortical generators of observed scalp differences.

Support: This work was funded by the Swiss National Science Foundation and the Tiny Blue Dot foundation.

0809

A RANDOMIZED DOUBLE-BLIND, PLACEBO CONTROLLED TRIAL WITH CROSS-OVER, TO ASSESS THE EFFICACY OF CORRECTING VITAMIN D DEFICIENCY IN IMPROVING THE SYMPTOMS OF RESTLESS LEGS SYNDROME (RLS).

Wong, J. Gupta, D. Nadhim, A. Bhat, S. Polos, P. JFK Medical Center Neuroscience Institute, Edison, NJ.

Introduction: Recent studies have shown an association of low Vitamin D levels and severity of RLS symptoms. However, effect

Methods: This is an ongoing study at the JFKMC Sleep Clinic. Eligible Patients with RLS include those with vitamin D 25, hydroxy deficiency (<20 ng/ml), or insufficiency (<30 ng/ ml). Such patients will be enrolled in the study after comorbid conditions like iron deficiency and OSA have been adequately corrected. Randomization of the patients will be done by the JFK pharmacy so the patient and provider are blinded to the substance. Substance A or B could be either Vitamin D3 Capsule 50,000 IU, or placebo. Each patient takes A for 6 weeks and then crosses over to B for 6 weeks. Weekly iRLS questionnaires will be collected. Actiwatch Device, to assess activity count per minute, will be worn on the ankle at night for one week time periods: at baseline, at end of 6 weeks of taking A and then at the end of 6 weeks of taking B. Vitamin D levels will also be assessed after each course of supplementation and correlated with subjective and objective findings.

Results: Between July 7, 2019 to current, 50 consecutive patients seen in sleep clinic with RLS were assessed for vitamin D levels. Ages ranged from 23-86 years. 27 patients were female (54%). Two patients met inclusion criteria and have started their 13-week study.

Conclusion: This study will help to establish the role of Vitamin D deficiency as a risk factor for RLS, independent of ferritin levels, and comorbid OSA, in affected individuals. This may help to discover a potentially treatable form of RLS.

Support: No financial support.

0810

SLEEP DISTURBANCES IN RETURNING VETERANS THAT ARE HOMELESS AND COMBAT-EXPOSED

Speed, K. J.¹ Crean, H. J.¹ Bishop, T. J.¹ Hoff, R.² Pigeon, W. J.¹ ¹Center of Excellence for Suicide Prevention, Canandaigua, NY, ²Department of Psychiatry, Yale University School of Medicine, New Haven, CT.

Introduction: Challenges with sleep (i.e., nightmares and insomnia) impact military service members both during and following deployment, but may occur more frequently in combat-exposed individuals. In addition, among the challenges faced following the transition from active duty to Veteran status are periods of home-lessness, which may further contribute to sleep disturbances.

Methods: The present analyses utilized data from the Survey of Experiences of Returning Veterans, a national survey of recently returning combat Veterans focused on the examination of sex differences following exposure to traumatic events. The sample (n = 793) consisted of 58% males; females were oversampled and all branches were represented. Ordinal and multiple linear regressions were used to investigate the role of combat exposure and homelessness in predicting nightmare distress and insomnia severity.

Results: An ordinal regression found that combat exposure (b = -.02, p < 0.001), homelessness (b = -.31, p = 0.010), and insomnia severity (b = -.10, p < 0.001) each significantly predicted nightmare distress. These variables increased risk (SAS parameterizes these models so that negative coefficients are associated with increased risk). Demographic variables were not significantly related to nightmare distress. Similarly, combat exposure ($\beta = .100$, p = .002) was associated with insomnia severity, as was nightmare

distress (β = .522, p < .001). The moderational role of gender and homeless in the above models are also tested.

Conclusion: For those who have been combat exposed, have a history of homelessness, and report insomnia symptoms there is an increased odds of reporting nightmare distress, sleep disturbance is even more likely to occur. Although combat exposure and nightmare distress were predictive of insomnia severity, history of homelessness was not. These findings suggest that nightmare distress and insomnia symptoms are a significant concern in our returning combat-exposed Veterans, with nightmares being even more problematic for those at risk of homelessness.

Support: This study was funded by VHA CSR&D grant ZDA-01. Dr. Speed is supported, in part, by the VA Advanced Fellowship Program in Mental Health Illness Research and Treatment, the VISN 2 Center of Excellence for Suicide Prevention at the Canandaigua VAMC.

0811

PERIODIC LIMB MOVEMENTS IN PATIENTS WITH DYSAUTONOMIA

Tallavajhula, S.

University of Texas Health Science Center, Houston, TX.

Introduction: Periodic Limb Movements in Sleep (PLM) have been thought to result in increase in sympathetic tone in sleep, tachy-cardia, elevated systolic blood pressure and therefore elevation in risk of cardiovascular disease. However, evidence of a direct causa-tive relationship has been elusive. We discuss the presence of PLM in patients with dysautonomia, proposing that autonomic dysfunction may be either a causative factor for PLM or share common pathogenesis with PLM.

Methods: Three patients with dysautonomia, referred to the TIRR Memorial Hermann Hospital neurological sleep disorders center with varying sleep-related symptoms were studied with polysomnography, recorded under AASM guidelines. PLM were scored using standard criteria described in the AASM scoring manual.

Results: The three patients described all carried a diagnosis of dysautonomia, diagnosed by tilt table testing. The first patient was an 18 year old woman with complaints of palpitations during sleep and frequent arousals. Her PLM index was 49.8. Serum ferritin level was pending. The second patient was a 35-year-old woman who complained of nonrestorative sleep. Polysomnogram demonstrated a PLM index of 17.6. Ferritin level was 36 ng/mL. The third patient was a 34-year-old woman with insomnia who was also found to have a PLM index of 15.1. Her serum ferritin level was 24. She underwent therapeutic iron infusions and experienced substantial improvement in subjective sleep quality. None of the patient described symptoms of restless legs syndrome or had any bleeding disorders to explain low ferritin levels.

Conclusion: The patients described above all were relatively healthy young women with symptoms of dysautonomia and disturbed sleep. It would be unusual for young individuals to present with PLM which are commonly associated with advancing age. In at least one patient, iron infusions improved sleep quality dramatically. Work needs to be done to establish relationship between autonomic imbalance and PLM, suggesting that the previously thought unidirectional relationship, [i.e. PLM causing change in sympathetic tone] may actually be bidirectional. **Support:** None

NIGHTMARES: AN INDEPENDENT RISK FACTOR FOR CARDIOVASCULAR DISEASE ULMER. C. S.

DURHAM VA & DUKE MEDICAL CENTERS, DURHAM, NC.

Introduction: Associations between PTSD, sleep and cardiovascular disease are well-established in prior research, but few studies have examined adverse health correlates of nightmares. Nightmares are often called the "hallmark" symptom of PTSD and represent the cardinal sleep-specific manifestation of PTSD. Yet, prior studies have not examined nightmares' independent contribution to cardiovascular disease risks beyond risks conferred by PTSD. The purpose of this study was to examine associations between nightmares and cardiovascular disease in Veterans with and without PTSD.

Methods: Participants were Veterans (N=3876; 78% male) serving since September 11, 2001, 1 or 2 tours of duty (73.5%), aged 38 years (SD=10.4), 31% meeting criteria for current PTSD, with equivalent proportions of African-Americans (48%) and Caucasians (48%). Nightmare frequency was assessed using the Davidson Trauma Scale (DTS), with "frequent" defined as occurring at least 2-3 times per week. Self-reported medical issues were assessed using the National Vietnam Veterans Readjustment Study (NVVRS) Self-report Medical Questionnaire. PTSD diagnosis was established using the Structured Clinical Interview for DSM-V.

Results: Frequent nightmares over the past week were endorsed by 33% of participants. Cardiovascular conditions were endorsed at the following rates: heart problems (6%); diabetes (6.6%); arthrosclerosis (0.5%); hypertension (29.2%); stroke (0.7%); and heart attack (1.2%). After adjusting for age, sex and race, frequent nightmares were associated with heart problems (F=7.50, p=.006), high blood pressure (F=23.84, p<.0001), and heart attack (F=7.19, p=.007). When PTSD was added to the model, these associations remained significant.

Conclusion: We found that frequent nightmares among Veterans are associated with cardiovascular conditions, even after controlling for the effects of PTSD. Additional research is needed to explore mechanisms explaining these associations and determine if reducing nightmare frequency and severity results in improved cardiovascular health.

Support: This work was supported by the Department of Veterans VISN 6 MIRECC and ADAPT Centers at the Durham VA Health Care System. The views expressed in this article are those of the authors and do not necessarily represent the views of the Department of Veterans Affairs.

0813

CIRCADIAN REST-ACTIVITY RHYTHM IN ISOLATED REM SLEEP BEHAVIOR DISORDER

Stefani, A.

Innsbruck Medical University, Innsbruck, AUSTRIA.

Introduction: Isolated REM sleep behavior disorder (iRBD) is a parasomnia characterized by abnormal behaviours occurring during REM sleep. Several studies showed that iRBD is a prodromal stage of synucleinopathies. Therefore identifying iRBD in the general population is of utmost importance. Aim of this study was to explore whether the assessment of circadian rest-activity rhythm features, measured by actigraphy, can distinguish iRBD patients from patients suffering from disorders characterized by other pathological motor activity during sleep and healthy controls. **Methods:** Nineteen subjects with video-polysomnographic (v-PSG) diagnosis of iRBD, 39 subjects with other disorders with motor activity during sleep (19 restless leg syndrome -RLS- and 20 untreated sleep apnea syndrome patients -SAS) and 16 healthy controls underwent 2-week actigraphy, v-PSG, and completed RBD screening questionnaires. Nonparametric analyses were applied to assess rest-activity rhythm features; daytime napping was also evaluated. The diagnostic value of nonparametric measures has been assessed through ROC curve.

Results: iRBD patient showed lower sleep efficiency, increased WASO and increased frequency of prolonged activity bouts compared to RLS and controls, while no difference emerged with SAS patients. Moreover, iRBD patients presented increased occurrence of estimated nap in comparison to RLS, SAS and controls. The nonparametric measure I<O distinguished iRBD patients from RLS, SAS and controls with an area under the curve greater than that of RBD screening questionnaires.

Conclusion: The nonparametric index I<O is able to distinguish iRBD patients from patients with other pathological motor activity during sleep and controls, confirming its potential use as an objective measure suitable to screen large at-risk populations.

Support: This study was supported by a grant from the Austrian Science Fund (FWF) to Birgit Högl, I 2120-B27.

0814

A NEW DIAGNOSTIC TOOL FOR ISOLATED REM SLEEP BEHAVIOR DISORDER: AUTOMATED 3D VIDEO ANALYSIS OF LOWER LIMB MOVEMENTS DURING REM SLEEP

Stefani, A.

Innsbruck Medical university, Innsbruck, AUSTRIA.

Introduction: The differentiation of isolated REM sleep behavior disorder (iRBD) or its prodromal phase (prodromal RBD, pRBD) from other disorders with motor activity during sleep is critical for identifying α -synucleinopathy in an early stage. Currently, definite RBD diagnosis requires video-polysomnography (vPSG). Aim of this study was to evaluate automated 3D video analysis of leg movements during REM sleep as objective diagnostic tool for iRBD.

Methods: A total of 122 participants (40 iRBD, 18 pRBD, 64 with other disorders with motor activity during sleep) were recruited among patients undergoing vPSG at the Sleep Disorders Unit, Department of Neurology, Medical University of Innsbruck. 3D videos synchronous to vPSG were recorded. Lower limb movements rate, duration, extent and intensity were computed using a newly developed software.

Results: The analyzed 3D movement features were significantly increased in subjects with iRBD compared to pRBD and other disorders with motor activity during sleep. Minor leg jerks with a duration <2 seconds discriminated with the highest accuracy (90.4%) iRBD from other motor activity during sleep. Automatic 3D analysis did not differentiate between pRBD and other disorders with motor activity during sleep.

Conclusion: Automated 3D video analysis of leg movements during REM sleep is a promising diagnostic tool for identifying subjects with iRBD in a sleep laboratory population and is able to distinguish iRBD from subjects with other motor activities during sleep. For future application as a screening, further studies should investigate usefulness of this tool when no information about sleep stages from vPSG is available.

Support: This study was funded by the Austrian Science Fund (FWF), Project KLI 677-B31.

VIOLENT PARASOMNIAS IN PATIENTS WITH REM WITHOUT ATONIA. PARASOMNIA OVERLAP DISORDER VS PURE REM BEHAVIOR DISORDER

Simmons, J. H.^{1,2} Meskill, G. J.^{1,2} Lavender, M. G.¹ ¹Comprehensive Sleep Medicine Associates, Houston, TX, ²Sleep Education Consortium, Houston, TX.

Introduction: REM-Behavior-Disorder (RBD) patients are known for parasomnias causing self-injury. On literature review, harm to others using a weapon is not well established. Some opinions state REM-parasomnias do not consist of elaborate actions, such firing a gun. This has significant ramifications in forensic medicine when RBD is a consideration. We reviewed our RBD patients to identify instances in which a gun was used during a parasomnic event to characterize clinical features associated with such behaviors.

Methods: We reviewed over 57 RBD cases from Texas, between 2014-2017 seeking parasomnias in which a gun was used.

Results: We found two patients in whom a gun was used during a parasomnia, representing < 3.5% of cases. Case-1: 59 y/o F with a 5 year hx of parasomnias of screaming, thrashing, roaming and one instance in which she pointed an unloaded gun at her husband saving she was going to kill him. She had no recollection of the event. NPSG demonstrated REM without atonia, mild OSA (1a AHI of 11/hr) and frequent PLMS. Case-2 presented to a sleep center in 1989 at 33 y/o with 3 year Hx of EDS, found to have mild OSA unresponsive to PAP Tx, then diagnosed with narcolepsy. He later developed cataplexy and progressed to developing parasomnias 15 years later. He demonstrated REM without atonia on a CPAP re-titration NPSG study done in part for his parasomnias, 20 years after original assessment. PLMS were also demonstrated. His parasomnias consisted of yelling, screaming, roaming and one time he woke up finding bullet holes in his closet door with no recollection of firing his gun, which he kept near his bed.

Conclusion: RBD is associated with a wide range of parasomnic events, almost never captured in the laboratory. These patients had clear RBD findings. It is possible they had Parasomnia Overlap Disorder in which Non-REM parasomnias occur in patients with RBD. PLMS and or OSA may contribute by fragmenting sleep. Nonetheless, it is clear that RBD patients can have elaborate parasomnias involving the use of weapons. More attention of this is noteworthy since reports are lacking in the literature.

Support: N/A

0816

SLEEP CORRELATES OF NIGHTMARES AMONG VETERANS

Patel, R. K.^{1,2} Schwartz, S. W.¹ Sebastiao, Y. V.³ Andrews, A.^{1,2} Foulis, P. R.^{1,2} Anderson, W. M.^{1,2}

¹University of South Florida, Tampa, FL, ²James A. Haley Veterans Hospital, Tampa, FL, ³Nationwide Children's Hospital, Columbus, OH.

Introduction: There is an increased prevalence of Nightmare disorder (ND) among patients with obstructive sleep apnea (OSA). A further investigation of objectively measured sleep parameters among patients with and without ND could inform on potential comorbidities. We hypothesize ND correlates with Epworth sleepiness scale (ESS), apnea hypopnea index (AHI), Trough 02% (Sp02 nadir), and periodic limb movement (PLM) index.

Methods: Data presented herein are interim results from an IRB approved study to determine correlates and sequelae of nightmares. A cohort of all patients with ND visiting James A Haley Veterans Hospital between 2007 and 2011 was defined along with a random cohort of control patients. Demographic and outpatient visit data between January 2006 and April 2016 was pulled from VISTA for both the ND and control cohorts, and patients who had undergone a sleep study were identified. To date, sleep summary data has been individually extracted for 111 ND patients and 835 control patients. Logistic regression (SAS 9.4) was used to compare ESS, AHI, Sp02 nadir, and PLM Index.

Results: Mean age for ND was significantly lower at 49.7 ± 14 , compared to 58.4 ± 12 for controls. Other demographic measures were similar including gender, race, and marital status. PLM index was significantly lower in ND compared to controls, however this relationship disappeared after adjusting for age. There were neither significant differences between other polysomnographic (PSG) variables, specifically AHI and Sp02 nadir, nor did OSA severity significantly different between ND and controls.

Conclusion: Among veterans undergoing a PSG, there were no significant differences between measured sleep parameters. Our results contradict our hypothesis that ND correlates with ESS, AHI, Sp02 nadir, and PLM index.

Support: This material is the result of work supported with resources and the use of facilities at the James A. Haley Veterans' Hospital.

0817

ABNORMAL REM SLEEP ATONIA CONTROL IN PATIENTS WITH CHRONIC POST-TRAUMATIC STRESS DISORDER

Feemster, J. Steele, T. Tao, Y. Rivera, S. Gossard, T. Teigen, L. Timm, P. McCarter, S. St. Louis, E. Mayo Clinic, Rochester, MN.

Introduction: Post-traumatic stress disorder (PTSD) is characterized by persistent mental and emotional stress following one or more significant physical or psychological traumatic incidents earlier in life. Vivid recall of the events, including traumatic nightmares, and prominent sleep disturbance are usual in PTSD. Previous studies have suggested that PTSD may share some clinical features with idiopathic REM sleep behavior disorder (iRBD) including altered REM sleep without atonia (RSWA) levels. Our group has previously found evidence for altered RSWA control in patients with psychiatric disease, including a pilot sample of PTSD patients with iRBD. We aimed to comparatively analyze RSWA levels between patients with PTSD, PTSD and RBD (PTSD+RBD), iRBD, and controls.

Methods: We selected 18 PTSD, 18 PTSD+RBD, 15 iRBD, and 51 healthy control patients matched for age and sex from the Mayo Clinic Center for Sleep Medicine's polysomnography database for RSWA quantification. RSWA amounts in the submentalis (SM) and anterior tibialis (AT) were quantitatively analyzed as a percentage of REM sleep duration, in accordance with previously published methods. Non-parametric analyses were performed to compare RSWA, patient demographics, and PSG data across groups. Significance was set at p < 0.016.

Results: Patients with PTSD had significantly higher RSWA than controls in all RSWA density measures (p < 0.016 for all). All measures of RSWA, excluding average SM duration, were significantly greater in PTSD+RBD patients compared with controls (p < 0.016 for all). Within the PTSD group, patients on antidepressants did not have significantly higher RSWA in any of the measures.

PTSD+RBD patients had significantly higher SM Phasic, AT Any, SM+AT Any, and Tonic RSWA measures than PTSD patients (p < 0.016 for all).

Conclusion: PTSD patients have significantly higher RSWA than controls, with PTSD+RBD patients having higher RSWA levels than PTSD patients. These data provide the first evidence for abnormal RSWA control in patients with chronic PTSD. This provides evidence of a unique biology in PTSD that could imply a future risk for neurodegenerative disease in PTSD similar to RBD patients. Further prospective studying will need to be performed on patients with PTSD to understand the unique biology. **Support:**

CYCLIC ALTERNATING PATTERN AS INDICATOR FOR SUBJECTIVE SLEEP QUALITY IN COMMUNITY-DWELLING OLDER MEN

Hartmann, S. Baumert, M.

The University of Adelaide, Adelaide, AUSTRALIA.

Introduction: The micro-architecture of NREM sleep displays a cyclic alternating pattern (CAP) comprising activation phases of slow high-amplitude waves (A1), fast low-amplitude brain activity rhythms (A3) or a mixture of both (A2). In this study, we investigated the relationship between CAP and subjective sleep quality parameters reported by community-dwelling older men from the Osteoporotic Fractures in Men Sleep Study.

Methods: CAP was scored in 2,811 overnight EEG recordings using a high performance automated CAP detection system. We quantified the ratio between CAP time and NREM sleep time (CAP rate), the number of A1-phases per hour of NREM sleep (A1 index), and the number of A2+A3-phases per hour of NREM sleep (A2+A3 index). Also, participants were asked to score the quality of their sleep on a Likert scale with five items from light to deep, from short to long, and from restless to restful. The relationship between CAP parameters and the subjective sleep quality measures was determined using ANCOVA with traditional sleep disturbance indices such as obstructive apneahypopnea index and arousal index as covariate.

Results: CAP rate decreased significantly with increasing quality of sleep for all three subjective measures (light vs. deep: $58.8\pm22.3\%$ vs. $54.6\pm20.5\%$, p < 0.001; short vs. long: $58.4\pm21.4\%$ vs. $55.1\pm20.5\%$, p < 0.001, restless vs. restful: $59.4\pm20.8\%$ vs. $55.6\pm21.0\%$, p = 0.002). The A1 index did not show any significant variations across all three sleep quality parameters. The A2+A3 index behaved similarly to the CAP rate with decreasing values for each subjective measure (all: p < 0.001).

Conclusion: CAP rate, especially A2+A3-phases, are reduced in older men who report good sleep quality, while A1 index did not show any significant relationship with subjective sleep quality measures. Hence, CAP is an indicator of sleep quality.

Support: The National Heart, Lung, and Blood Institute (NHLBI) provides funding for the MrOS Sleep ancillary study "Outcomes of Sleep Disorders in Older Men" under the following grant numbers: R01 HL071194, R01 HL070848, R01 HL070847, R01 HL070842, R01 HL070841, R01 HL070837, R01 HL070838, and R01 HL070839.

0819

OBJECTIVE SLEEP EFFICIENCY IS ASSOCIATED WITH LONGITUDINAL RISK OF HIGH DEPRESSIVE SYMPTOMS IN PREGNANT WOMEN

Tsai, S.¹ Lee, P.² Gordon, C.³ Cayanan, E.³ Lee, C.¹

¹National Taiwan University, Taipei, TAIWAN, ²National Taiwan University Hospital, Taipei, TAIWAN, ³University of Sydney, Sydney, AUSTRALIA.

Introduction: Sleep disturbances are one of the most frequent complaints identified during routine prenatal care visits. Sleep and mood disturbances are often intertwined, and depression in particular is a leading cause of disability and disease burden worldwide. The purpose of this study was to examine the predictive association of objective actigraphic and subjective sleep disturbances with depressive symptoms in pregnant women.

Methods: We recruited 204 first-trimester pregnant women from a large university-affiliated hospital. They provided baseline

socio-demographic and health information, wore a wrist actigraph for 7 days, and completed the Pittsburgh Sleep Quality Index and Center for Epidemiologic Studies - Depression Scale and repeated this again in the second and third trimesters. Each data collection was scheduled at least 8 weeks apart. Unadjusted and multivariable adjusted odds ratios with 95% confidence intervals were used to evaluate sleep disturbances at 1st trimester and risk of high depressive symptoms at follow-up.

Results: A total of 121 (59.3%) women had actigraphic sleep efficiency of < 85% and 92 (45.1%) had Pittsburgh Sleep Quality Index global scores > 5 indicative of poor sleep quality. In multivariable adjusted models, 1st trimester objectively measured sleep efficiency < 85% was associated with 2.65-, 3.86-, and 5.27-fold increased odds having risk of high depressive symptoms at 2nd trimester, 3rd trimester, and both 2nd and 3rd trimesters, respectively. No subjective sleep disturbance variables were significantly associated with risk of high depressive symptoms in multivariate adjusted models.

Conclusion: Objectively assessed poor sleep efficiency in the 1st trimester plays a crucial role in the development of both elevated and persistent high depressive symptoms in pregnancy. Future studies using objective sleep measurements and clinical diagnostic interviews are warranted to examine whether early interventions to improve sleep may help reduce high depressive symptom risk and lower depression rates in women during pregnancy.

Support: This study was funded by the Ministry of Science and Technology, Taiwan (MOST-101-2314-B-002-049-MY3).

0820

META-ANALYSIS OF THE ASSOCIATION OF AGE AND ACTIGRAPHY-ASSESSED SLEEP ACROSS THE LIFESPAN

Bowman, M. A. Buysse, D. J. Marsland, A. L. Wright, A. G. Foust, J. Mehra, R. Srinivasan, S. Kohli, N. Carroll, L. Jasper, A. Hall, M. H.

University of Pittsburgh, Pittsburgh, PA.

Introduction: Sleep quantity and continuity vary across the lifespan. Actigraphy is reliable, ecologically valid, and is the most widely-used behavioral measure of sleep in research and personal health monitoring. The extent to which age is associated with actigraphy-assessed sleep has not been evaluated across the lifespan. The aim of this meta-analysis was to evaluate the associations between age and actigraphy-assessed sleep in relatively healthy individuals.

Methods: A systematic search of PubMed, Embase.com, Cochrane CENTRAL, and PsycINFO using "actigraphy" and "sleep" terms provided 7,079 titles/abstracts, which were screened to exclude studies of only individuals with mental health disorders, medical conditions, sleep disorders, or shift workers. We evaluated 1,379 full-text articles for reports on the association between age and actigraphy-assessed sleep duration, efficiency, timing, and/or regularity. Overall, 88 articles met these criteria (182 effect sizes; N=18,443). Four meta-analyses were conducted, examining sleep duration (k=86), sleep efficiency (k=58), bedtime (k=27), and wake-up time (k=11). There were insufficient numbers of studies (less than 5) to evaluate sleep midpoint or sleep regularity. We tested continent of the study, study design, actigraphy device type, and number of nights of data collection as moderators of metaanalytic associations.

Results: With increasing age, sleep duration was shorter (r = -0.13) and sleep efficiency was lower (r = -0.06). Bedtime was later with age for ages 6-21 (r = 0.31) and earlier for ages 22 and up (r = -0.65).

Wake-up time was not associated with age for ages 6-21 (r = 0.20) but was earlier with increasing age for ages 22 and up (r = -0.71). The strength of these associations was modified by continent and study design, but not by type of actigraphy device or number of nights of data collection.

Conclusion: Weak associations between age and actigraphyassessed duration and efficiency suggest that inadequate sleep quantity or poor sleep continuity should not be dismissed as typical consequences of aging. Large associations between age and sleep timing, despite a small literature, highlights a promising area for further study, particularly to determine the age at which sleep timing shifts from delaying to advancing.

Support: MAB was supported by T32 HL07560.

0821

GENDER DIFFERENCES IN SAILOR WELL-BEING, SLEEP-RELATED BEHAVIORS, AND PSYCHOMOTOR VIGILANCE PERFORMANCE IN THE UNITED STATES NAVY

Shattuck, N. L. Matsangas, P.

Human Systems Integration Program, Operations Research Department, Naval Postgraduate School, Monterey, CA.

Introduction: Approximately 18% of US Navy sailors are females. Research has shown gender-related differences in the prevalence of sleep disorders in active duty personnel (Foster et al., 2017). Specifically, insomnia, depression, and anxiety are more prevalent in females, while obstructive sleep apnea is more prevalent in males. We have studied the sleep patterns and fatigue levels of crew members on more than 30 US Navy ships. The current study focuses on gender differences in well-being, sleep-related behaviors, and psychomotor vigilance performance of sailors in the US Navy.

Methods: Using a longitudinal, naturalistic observation paradigm, data were collected from crewmembers on nine USN ships while performing their normal underway duties. Participants (N=1,056) tended to be young (on average 27 years of age), predominantly male (80.6%), and enlisted (84.8%). We assessed average day-time alertness (Epworth Sleepiness Scale), insomnia symptoms (Insomnia Severity Index), mood (Profile of Mood States), and sleep quality (Pittsburgh Sleep Quality Index). Sleep was assessed with actigraphy and logbooks. Sailors performed a 3-minute version of the Psychomotor Vigilance Task (PVT), which was built into their wrist-worn actigraph.

Results: Compared to males, female sailors reported more depressive symptoms (p=0.042) and less vigor (p<0.001). Females slept more (daily sleep duration: p<0.001) but their sleep was split into more episodes than their male counterparts (p=0.029). Fewer females reported a regular exercise routine (p=0.033). In addition, females report consuming fewer energy drinks (p=0.007), and using fewer nicotine products (p=0.013). Lastly, consistent with findings from civilian populations, female sailors had slower reaction times on the PVT (p<0.001) and experienced more lapses combined with false starts (p<0.001) than their male counterparts.

Conclusion: Compared to their male peers, female sailors tend to report higher levels of depression and lower levels of vigor. They experience more pronounced split sleep, are less likely to report having an exercise routine, and have poorer performance on the PVT. Fewer females report using energy drinks and nicotine products.

Support: This research was supported by the Naval Medical Research Center's Advanced Medical Development Program, the US Navy 21st Century Sailor Office, and the US Navy OPNAV N1.

0822

FRAIL OLDER MEN WITH NOCTURIA ARE DISPROPORTIONATELY AFFECTED BY EXCESS NOCTURNAL URINE PRODUCTION

Monaghan, T. F.¹ Wagg, A. S.² Agudelo, C. W.¹ Rahman, S. N.¹ Michelson, K. P.¹ Epstein, M. R.³ Everaert, K.⁴ Lazar, J. M.¹ Weiss, J. P.¹ Bliwise, D. L.⁵

¹SUNY Downstate Health Sciences University, Brooklyn, NY, ²University of Alberta, Edmonton, AB, CANADA, ³Temple University Hospital, Philadelphia, PA, ⁴Ghent University Hospital, Ghent, BELGIUM, ⁵Emory University School of Medicine, Atlanta, GA.

Introduction: Nocturia is a risk factor for falls and hip fractures in older adults. We determined whether the Frailty Index (FI), incorporating comorbidities, functional performance, and physical signs, was associated with nocturia frequency and/or overnight urine production.

Methods: We examined nightly (24-hour) voiding diaries (men \geq 65 years) in an outpatient urologic clinic demonstrating \geq 2 nocturnal voids (n=158). FI calculations followed Rockwood (CMAJ 2005;173:489-95). A total of 39 conditions were assessed. Three FI groups were established: Low (\leq 0.077) (n=59), Intermediate (>0.077 and <0.179) (n=58), and High (\geq 0.179) (n=41). We compared number of nocturnal voids (NV), nocturnal urine volume (NUV) (in mL), and 24-hr total urine volume (24-hr TUV) (in mL) across groups.

Results: NV did not differ by group (p=0.333) (median for all groups=3). However, NUV (916 [671-1419] vs. 690 [505-942] vs. 630 [500-1050] mL) differentiated the High, Medium and Low FI groups (p<0.001 via Kruskal-Wallis with Bonferroni pairwise adjustments), respectively. Similarly, 24-hr TUV differentiated the 3 groups (2200 [1800-2550] vs. 1620 [1259-2119] vs. 1650 [1390-2517] mL, p=0.005). Differences in NUV remained significant (p=0.006) after eliminating Diabetes Mellitus cases (n=44). However, differences did not persist for 24-hr TUV (p=0.180).

Conclusion: Higher NUV, but not 24-hr TUV, was a robust correlate of frailty in these older men. Accounting for diabetes did not diminish the effect. Although undiagnosed sleep apnea remains a possible cause, recent chronobiologic data (Monaghan et al, Age Aging, 2020, in press) suggest that nocturia in the aged is characterized by excess free water clearance early in the sleep period. This argues against solute-driven urine production (as might be expected in sleep apnea) in accounting for the effect. Nocturia may represent a conspicuous and important change in circadian rhythm of urine production occurring in old age. **Support:** N/A

0823

AGE-RELATED CHANGES IN NOCTURNAL URINE COMPOSITION

Bliwise, D. L.¹ Monaghan, T. F.² Lazar, J. M.² Epstein, M. R.³ Agudelo, C. W.² Weiss, J. P.² Weedon, J.² Everaert, K.⁴ ¹Emory University School of Medicine, Atlanta, GA, ²SUNY Downstate Health Sciences University, Brooklyn, NY, ³Temple University Hospital, Philadelphia, PA, ⁴Ghent University Hospital, Ghent, BELGIUM.

Introduction: In humans sleeping nocturnally, nocturnal polyuria (NP) refers to high rate of overnight urine production. NP is a heterogeneous condition that may reflect both free water and/

or sodium diuresis, but the influence of age on differential fluid handling remains poorly understood. This study examined diuresis rate, sodium clearance, and free water clearance (FWC) by age, time of day (nighttime vs. daytime) and NP status (positive/negative) in subjects under entrained conditions sleeping nocturnally.

Methods: Convenience samples (age range 18-91; 82 men, 148 women) recruited from a urology ambulatory care unit (n=135) or continence clinic (n=95) collected 8 urine samples at 3-hour intervals over a single 24-hr period. Three separate mixed linear models were constructed for diuresis rate, sodium clearance, and FWC using four predictors: NP status (present [>90mL/h] vs. absent), time of day (night = 0100, 0400, 0700), age (as a continuous measure), and study source.

Results: Subjects with NP experienced both higher nighttime vs. daytime diuresis rate (1.89 vs. 1.44 mL/min, p<0.001), sodium clearance (0.91 vs. 0.74 mL/min, p<0.001), and FWC (-0.38 vs. -0.71 mL/min, p<0.001), whereas subjects without NP demonstrated lower nighttime vs. daytime diuresis rate (0.94 vs. 1.06, p=0.004) and no difference in sodium clearance (0.59 vs. 0.64, p=0.120) or FWC (-0.80 vs. -0.86, p=0.268). Regardless of NP status, FWC increased with age (p=0.039), and older age (>70) was accompanied by an increase in the ratio of nighttime/daytime diuresis rate and both nighttime and daytime sodium clearance.

Conclusion: Irrespective of NP, older adults experience proportionally greater nocturnal sodium clearance, as well as a complex surge in both daytime and nighttime FWC. The data imply that both nocturnal sodium clearance and FWC may reflect the relevant substrate underlying excess nocturnal urine production in elderly persons.

Support: N/A

0824

IMPROVING SUBJECTIVE SLEEP QUALITY MEASURES THROUGH MINDFULNESS TRAINING IN THE ELDERLY: PRELIMINARY DATA FROM THE MINDFULNESS SLEEP THERAPY (MIST) STUDY

Perini, F.¹ Foong Wong, K.¹ Teng, J.¹ Hassirim, Z.¹ Lin, J.¹ Leow, Z.¹ Lee Henderson, S.² Fan, Q.² Lo, J. C.³ Ong, J. C.⁴ Doshi, K.² Lim, J.¹

 ¹Centre for Cognitive Neuroscience, Duke NUS Medical School, Singapore, SINGAPORE, ²Department of Psychology, Singapore General Hospital, Singapore, Singapore, SINGAPORE,
 ³Department of Medicine, National University of Singapore, Singapore, SINGAPORE, ⁴Northwestern University, Feinberg School of Medicine, US, Chicago, IL.

Introduction: Poor sleep is a modifiable risk factor for multiple chronic disorders. Mindfulness-based therapies potentially improve sleep by enhancing awareness and acceptance of internal and external experiences, thus reducing pre-sleep hyper-arousal. In this pre-registered, randomized controlled trial, we tested the effect of mindfulness-based treatment for insomnia (MBTI) on subjective sleep quality measures (Pittsburgh Sleep Quality Questionnaire, PSQI) in the elderly.

Methods: Participants above 50 years old with sleep difficulties (PSQI \geq 5) (mean (sd) age = 62.0 (6.35), 44 female) attended either an 8-week MBTI (N = 34) or sleep hygiene education and exercise program (SHEEP; N = 35). Before and after the interventions, we collected PSQI, insomnia symptoms and features measures (Pre-Sleep Arousal Scale, PSAS; Insomnia Severity Index, ISI; Dysfunctional Beliefs and Attitudes about Sleep, DBAS-30), mindfulness (Five-Facets Mindfulness Questionnaire, FFMQ), and

mood and anxiety (Back Depression Inventory, BDI; State-Trait Anxiety Inventory, STAI). PSQI and PSAS (N = 26 to date) were collected at 6-month follow-up. Data were analysed with repeated-measures ANCOVA with group as a between-subject variable for the first 69 participants who completed the study.

Results: We observed significant improvement across both groups for sleep measures (PSQI: $F_{1,67}$ =36.442, p<.01; PSAS-Cognitive: $F_{1,67}$ =12.664, p<.01; ISI: $F_{1,67}$ =36.442, p<.0; DBAS: $F_{1,67}$ =28.749, p<.01) and mood (BDI: $F_{1,67}$ =26.393, p<.01; STAI-State: $F_{1,67}$ =4.608, p=.04; STAI-Trait: $F_{1,67}$ =7.687, p<.01), but not for Mindfulness ($F_{1,67}$ =2.256, p=.14) nor PSAS-somatic. No significant group by time interactions were found. We observed a correlation between PSQI decreases and FFMQ increases in MBTI (r=-.53, p<.01), but not in SHEEP (r=-.07, p=.70) participants. ANCOVA of 6-month PSQI data revealed a significant group by time interaction ($F_{1,24}$ =19.525, p=.03), with reduction from baseline in MBTI (t12=4.769, p<.01), but not in SHEEP group (t12=3.813, p=.08).

Conclusion: Preliminary results support MBTI as an accessible but effective behavioural intervention with potential long-term benefits for improving sleep and mood, and reducing cognitive-emotional arousal in the elderly.

Support: This study was supported by an award from the 7th grant call of the Singapore Millennium Foundation Research Grant Programme

0825

SLEEP DURING PREGNANCY THROUGH ONE YEAR POSTPARTUM: CORRESPONDENCE BETWEEN ACTIGRAPHY AND SELF-REPORT MEASURES

Beverly Hery, C. M.¹ Christian, L. M.²

¹Division of Epidemiology, College of Public Health, The Ohio State University, Columbus, OH, ²Department of Psychiatry & Behavioral Health, The Ohio State University Wexner Medical Center, Columbus, OH.

Introduction: Disrupted sleep and shorter sleep duration is common in pregnancy, due to hormonal changes and physical discomfort, and in postpartum due to new infants. Objective data in this population studied over more than one year are lacking. The current analysis focuses on actigraphy-based and self-reported sleep. We examined the level of correspondence between these two complementary measurement modalities.

Methods: Pregnant women were enrolled in the Stress and Health in Pregnancy and Postpartum (SHIPP) study. Study visits were conducted from 2016-2019 during the 3rd trimester, 4-6 weeks postpartum, and 4 months, 8 months, and 12 months postpartum. Participants completed the Pittsburgh Sleep Quality Index (PSQI) and provided wrist-actigraphy data (Actiwatch, Philips Respironics) for one week prior to each study visit. Actigraphybased time in bed (TIB), total sleep time (TST), sleep efficiency (SE), WASO and sleep latency (SL) were calculated. Correlations were conducted between actigraphy-based and self-reported PSQI sleep measures.

Results: 79 women (28.9 \pm 4.6 years) provided complete actigraphy data (\geq 3 valid days; 6.4 \pm 1.0 days) for at least one time point. Objective TST from the 3rd trimester to 12-months postpartum was 7.19, 6.87, 6.86, 6.89, and 6.78 hours, respectively. Actigraphy-based TIB was positively correlated with self-reported TIB at all five visits (r's: 0.37-0.62, p's<0.01). Actigraphy-based TST was positively correlated with self-reported TST (r's: 0.27-0.48, p's<0.05) at all visits. Actigraphy-based SE was negatively correlated with the

PSQI Global Score at 4, 8, and 12 months postpartum (r's: -0.29 to -0.39, p's<0.05). Of note, actigraphy-based WASO and SL were not consistently correlated with any self-reported PSQI sleep measures. **Conclusion:** Ensuring collection of accurate sleep data during pregnancy and postpartum is important, as poor sleep is associated with negative health outcomes for both mother and child. Self-reported data is common in large, epidemiologic studies yet actigraphy-based measures may capture different aspects of sleep than self-report.

Support: This study was supported by the National Institutes of Health (R01 NR01366).

0826

GENDER DIFFERENCES IN SLEEP KNOWLEDGE OF COMMUNITY-DWELLING OLDER ADULTS

Baldwin, C. M.¹ Link, D. G.¹ Coon, D. W.¹ Quan, S. F.^{2,3} ¹Arizona State University, Edson College of Nursing & Health Innovation, Center for Innovation in Healthy & Resilient Aging, Phoenix, AZ, ²University of Arizona College of Medicine, Tucson, AZ, ³Harvard Medical School, Division of Sleep Medicine, Boston, MA.

Introduction: This work compares sleep knowledge of community-dwelling older adult men and women.

Methods: Data were derived from a community-based sleep training program that assessed pre- and post-test knowledge of obstructive sleep apnea (OSA), Insomnia, short sleep duration (SSD), restless leg syndrome (RLS), circadian rhythm disorders (CRD), and drowsy driving (DD) on a 1 (none) to 5 (great deal of knowledge) Likert-like scale. Data were analyzed with frequencies for age, sex, and sources of sleep information, and ANOVA to determine gender differences using SPSS (V24) with significance set at p < .05.

Results: Participants (N=158; 68% women) were 56 years and older residing in a retirement community. Pre-test means±standard deviations showed women versus men had greater knowledge of Insomnia (3.5 ± 1.3 vs. 2.9 ± 1.0 , p=.004) whereas men showed more knowledge of DD (3.2 ± 1.1 vs. 2.6 ± 1.3 , p=.01). A trend was noted for women to have greater knowledge of SSD (3.6 ± 1.2 vs. 3.2 ± 1.0 , p=.05). Post-test ANOVA showed a further increase in Insomnia knowledge for women versus men (4.4 ± 0.8 vs. 4.1 ± 0.7 , p=.04); however, overall pre/post-test scores for each of the sleep disorders across men and women increased significantly at the p<.001 level. Notably, more women to men reported accessing various resources for sleep information: newspapers/magazines (46:7), friends/family (29:9), the internet (25:11), TV (37:7), and physicians/nurses (45:20).

Conclusion: Findings indicate, prior to sleep training, women have greater knowledge of insomnia and short sleep duration, while men have more knowledge of drowsy driving. Women's greater understanding of insomnia persists even after sleep training; however, pre- to post-test scores for both sexes across sleep disorders show significant learning outcomes. One possible reason for women's greater knowledge of insomnia and short sleep could be their greater likelihood to access information on health and healthy lifestyle factors, including sleep, as well as their greater health care utilization.

Support: N/A

0827

THE ASSOCIATION AMONG DIAGNOSED AND SUSPECTED SLEEP DISORDERS AND PAIN

Ravyts, S. Dzierzewski, J.

Virginia Commonwealth University, Richmond, VA.

Introduction: Poor sleep has increasingly been linked to adverse pain outcomes. Yet, the complex interplay between sleep apnea, insomnia, or comorbid sleep apnea and insomnia (COMISA), and pain are less well understood. The purpose of the present study was twofold: 1) assess pain intensity in individuals with diagnoses of insomnia, sleep apnea, and COMISA, and 2) examine pain intensity in individuals who are at a high risk for having either sleep apnea, insomnia, or both sleep disorders.

Methods: Participants included 3401 adults (mean age= 42.77, male= 45.6%) who participated in an online study investigating sleep across the lifespan. Sleep apnea and insomnia diagnoses were self-reported while participant risk profiles for these disorders were assessed via the Insomnia Severity Index and the STOP-BANG questionnaire respectively. Average pain intensity over the last two weeks was rated from 0 (*no pain*) to 100 (*very severe*).

Results: Participants with self-reported comorbid sleep disorders reported higher pain scores than individuals with one or no sleep disorder (F(2,3398) = 71.61, p < .001). Among individuals with no previously diagnosed sleep disorder, participants with more insomnia symptoms reported greater pain intensity (F(3,3100) = 201.64, p < .001), as did those with higher scores on the STOP-BANG (F(2,3125) = 46.79, p < .001). Participants with suspected comorbid sleep disorders reported higher pain scores than individuals with either sleep apnea or no sleep disorder, but not insomnia (F(3,2887) = 110.15, p < .001).

Conclusion: Results suggest that individuals with known sleep disorders report high levels of pain particularly in the context of comorbid sleep apnea and insomnia. Additionally, individuals with suspected, but untreated, sleep disorders also report increased pain. Future research should examine whether treating one of more sleep disorders can improve pain outcomes.

Support: This work was supported by the National Institute on Aging (K23AG049955, PI: Dzierzewski).

0828

DESCRIPTIVE MODEL OF SLEEP QUALITY IN THE OLDER ADULTS

Morelhao, P. K.¹ Fernandes, G. L.¹ Dokkedal-Silva, V.¹ Pires, G. N.¹ Tufik, S.¹ Andersen, M. L.¹

¹Universidade Federal de Sao Paulo, Sao Paulo, BRAZIL,

²Universidade Federal de Sao Paulo, Sao Paulo, BRAZIL.

Introduction: Poor sleep quality is a health condition that impacts the quality of life of the older population. In the literature, there are questions about which objective sleep parameters should be considered to describe precisely the definition of sleep quality. There is ongoing debate with this term usually being used in relation to subjective sleep perception. This study aimed to investigate which objective and subjective sleep parameters contribute to a measurement of sleep quality in older adults.

Methods: A cross-sectional study using a representative sample of adults from the city of São Paulo, Brazil was performed. We used a dataset from the 2015 Epidemiological Study of Sleep from the City of São Paulo (EPISONO), including only individuals aged 60 years or more. We used exploratory factor analysis and structural equation modelling to identify relevant variables to a descriptive model of sleep quality.

Results: A total of 152 older adults were included. The final model consists of two factors, objective sleep quality which comprises sleep efficiency, total sleep time and sleep latency, and poor sleep

perception, constituted by scores in the Pittsburgh Sleep Quality Index and Insomnia Severity Index.

Conclusion: The results suggested that sleep quality had both an objective (sleep efficiency, total sleep time, latency of sleep onset) and subjective dimensions (subjective questionnaires). These results may be useful in the clinical scenario, serving as leads for a better understanding of the sleep quality in aging patients. Future studies may also benefit from this descriptive model to further researches other associations, such as sleep and pain in this population.

Support: The study was supported by Associação Fundo de Incentivo à Pesquisa (AFIP) and Coordenação de Aperfeiçoamento de Nível Superior (CAPES). ST and MLA received support from Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq).

0829

SLEEP, CHRONIC PAIN, AND GLOBAL HEALTH IN ADULTS AGES 65 OR OLDER

Tran, L. Jeon, B. Chasens, E.

University of Pittsburgh School of Nursing, Pittsburgh, PA.

Introduction: Understanding the association of sleep and pain in older adults can help improve their global health. The purpose of the study was to describe the associations between sleep, chronic pain, and global health in adults ages 65 or older.

Methods: This study was a secondary analysis of data from adults over 65 years in the 2015 Sleep in America Poll - Sleep and Pain by the National Sleep Foundation (NSF). The survey included demographics (age, race, marital status, education), sleep (duration, quality, insomnia symptoms), and pain (type [none, chronic, fleeting], intensity, location). Global health derived from general health, physical health, mental health, and quality of life with a potential range of 4-20; higher score=better health. The survey also queried fatigue and stress.

Results: The sample (N=248) was 65-91 years (mean age= 72.8 ± 5.9), male (53.6%), White (82.7%), married (65.7%), and with posthighschool education (54.4%). Average sleep duration was 425±74 minutes. "No pain" was reported by 38.7% of the sample (n=96), "fleeting pain" by 32.7% (n= 81), and "chronic pain" by 28.6% (n=71). The most common locations for chronic pain were shoulder or neck (63.2%) and back (69.4%). Average global health score was 9.8±2.9. There was no significant difference in time in bed, sleep duration, bedtime, or wake-up time between groups. Persons with chronic pain had higher average pain intensity, worst pain intensity, and current pain; they reported significantly lower sleep quality with significantly more restlessness, trouble staying asleep, and worry about getting a good night sleep (all p-values<.02), there was no significant difference in difficulty falling asleep compared to persons with no pain. Persons with chronic pain had significantly worse general health, physical health, mental health, global health, fatigue, and stress (all *p*-values<.02); but no significant difference in quality of life compared to persons with no pain.

Conclusion: We conclude that chronic pain has a significant negative impact on sleep and global health in the sample of adults ages 65 or older from the 2015 Sleep in America Poll - Sleep and Pain by the NSF.

Support: Undergraduate Research Mentoring Program, University of Pittsburgh School of Nursing.

0830

HORMONAL CONTRACEPTIVE USE AND SLEEP: A SYSTEMATIC REVIEW AND META-ANALYSIS

Bezerra, A. G.¹ Pires, G.^{1,2} Andersen, M. L.¹ Tufik, S.¹ Hachul, H.^{1,3}

¹Universidade Federal de São Paulo, São Paulo, BRAZIL, ²Santa Casa de São Paulo School of Medical Sciences, Sao Paulo, BRAZIL, ³Casa de Saúde Hospital Santa Marcelina, Sao Paulo, BRAZIL.

Introduction: The effects of hormonal contraceptives on sleep has been matter of debate in current literature. While some articles observed a sleep promoting effect and reduced sleep disordered breathing, others have failed to detect any result or even detected a worse sleep pattern in women using hormonal contraception. As the literature has been growing on this field, a systematic review is necessary to gather and compare all the studies in a comprehensive way.

Methods: A bibliographic search was conducted in Pubmed, Scopus and Web of Science. Studies were selected first based on titles and abstracts, followed by full text analysis and data extraction. Only original studies evaluating women using hormonal contraception were considered eligible. Both objective and subjective sleep-related outcomes were extracted for analyzes. Individual effect size for each articles was calculated using regular or standardized mean differences and meta-analyses were conducted using a DerSimonian and Laird random effects model.

Results: After the bibliographic search, 1787 non-duplicated articles were included in our initial data screening. Articles sample was reduced to 114 records after abstract screening and to ten studies after full text analyses. The following sleep outcomes were eligible for meta-analysis: Pittsburgh Sleep Quality Index (PSQI - 3 studies), total time in bed (4), subjective total sleep time (4), objective total sleep time (3), sleep latency (6), sleep efficiency (6). None of them resulted in statistically significant effects of contraceptive use and the effect size \pm 95% interval of confidence overlapped the zero value.

Conclusion: Hormonal contraceptives is not associated to any alteration in sleep patterns in women. This conclusion should be restricted to a general framework, since our sample does not allowed stratified analyses. Future studies should consider the effect of specific hormonal composition (ex.: combined vs. progestogenonly contraceptives) and administration route (contraceptive pills vs. levonorgestrel intrauterine device).

Support: AFIP, CAPES, CNPq

0831

HOT FLASHES AND INSOMNIA THROUGHOUT THE LIFE SPAN OF WOMEN FROM THE EPISONO COHORT

Hachul, H. Castro, L. S. Bezerra, A. G. Poyares, D. Andersen, M. L. Bittencourt, L. Tufik, S. Universidadede Federal de Sao Paulo - Department of Psychobiology, Sao Paulo, BRAZIL.

Introduction: Hormonal changes may trigger sleep disturbances in women. Insomnia affects one in every three-to-four of them, most likely during *pre* to *post* menopause, and especially in association with hot flashes. Thus, the present study aimed to investigate the occurrence of hot flashes among women with and without insomnia and on different reproductive stages.

Methods: Sampling procedure was a three-stage clustering of the population of Sao Paulo, Brazil according to gender, age (20-80 years), and socio-economic status. A total of 574 women were interviewed, underwent polysomnographic recording (PSG), and had fasting-blood samples collected. Hormone levels and a gyne-cological questionnaire were used to classify reproductive stages. Premenopausal women were classified either in the follicular,

luteal, or periovulatory stage or as anovulatory or under hormonal contraceptives; whereas those menopausal were classified in perimenopause or in *early* or *late* stages. Individuals reporting frequent and persistent insomnia symptoms accompanied by relevant daytime impairment were classified with insomnia syndrome. Objective insomnia was defined by increased sleep onset latency and/or awake after sleep onset, decreasing sleep duration.

Results: The final sample included 550 women, representing 53% of the EPISONO cohort (n=1,042). Hot flashes were reported by 9% of the premenopausal women (n=339) and by 42% of the menopausal. Complaints were more frequent among women in perimenopause (67%) and those in use of hormonal therapy (60%), and it tended to decrease in later stages (33%); whereas before menopause, hot flashes were especially reported by anovulatory women (26%), while significantly less by those using contraceptives (6%). Hot flashes were associated with a 2-fold increase in insomnia symptoms and while it predicted objective sleep alterations among premenopausal women, they did not after menopause, when alterations in sleep were better explained by an effect of aging.

Conclusion: Our current findings suggest that hot flashes are associated with irregular menstrual cycles among premenopausal women, and particularly with early stages of menopause, predicting both subjective and objective sleep alterations.

Support: This research was supported by fellowships from Associação Fundo de Incentivo à Pesquisa (AFIP) and Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES) - Finance Code 001.

0832

PHYSICAL EXERCISE IMPROVES SLEEP AND MUSCLE FUNCTION IN SARCOPENIC PATIENTS: A RANDOMIZED CONTROLLED TRIAL

De Sá Souza, H.¹ Piovezan, R. D.¹ Chagas Miranda, R. E.¹ Silva, B. M.¹ Tufik, S.¹ Poyares, D.¹ D'Almeida, V.¹ ¹Universidade Federal de São Paulo, SAO PAULO, BRAZIL, ²Universidade Federal de São Paulo, SAO PAULO, BRAZIL.

Introduction: Sarcopenia is a multifactorial condition that, like sleep debt, affects the elderly and is related to metabolic, endocrine, inflammatory alterations and risk to mortality. Resistance training (RT), in turn, can improve both factors. **Aim:** investigate the effects of 12-week RT on sleep and muscle function in the sarcopenic elderly.

Methods: 28 sarcopenic elderly were equally distributed in 2 groups at random: CTL: who participated in weekly lifestyle change lectures or; RT: who did the progressive load RT. Sleep was assessed by polysomnography, actigraphy and questionnaires. Isokinetic and isometric of peak torque (PT) of skeletal muscle, anabolic and catabolic hormones, pro and anti-inflammatory cytokines concentrations were also evaluated. For intention to treat analysis (Δ) the generalized linear/non-linear for absolute variables or Wilcoxon rank-sum (Mann-Whitney) test. Data are expressed as mean±standard deviation or median, minimum and maximum values and difference witch p<0.05.

Results: The RT reduced the time to sleep onset (16.09 ± 15.21) compared to CTL (29.98±22.57) group after the intervention. The Δ shows that RT had more N3 sleep (median:0.90, min:-13.40, max: 25.00) than CTL (median:-3.35, min:-15.20, max:19.10). The RT increases TTS (median:57.55, min:-204.75, max:220.91 vs median:-9.63, min:-120.98, max:185.57) and improved self-reported sleep quality (median: -1.50, min: -9.00, max: 4.00 vs median: 0.50, min: -3.00, max: 6.00) and sleep efficiency (median: 9.50, min: -15.00, max: 34.00 vs 0.00, min: -28.00, max: 18.00). For all muscle function parameters (extension and flexion knee in isokinetic or isometric PT) RT had

higher values compared to CTL group after 12 weeks of intervention (p<0.05). IL-1ra concentrations were higher in RT (median: 0.04, min: -0.02, max: 0.36) vs CTL (median:-0.01, min:-0.12, max:0.07).

Conclusion: Progressive load resistance training improves sleep parameters associated to muscle recovery in elderly people with sarcopenia, along with positive changes in physical performance.

Support: Associação Fundo de Incentivo à Pesquisa (AFIP), Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES), Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) and Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP).

0833

MIDLIFE SLEEP HEALTH IS ASSOCIATED WITH LATER-LIFE DEPRESSION AND ANXIETY

Hagen, E. W.¹ Barnet, J. H.¹ Sprecher, K. E.¹ Peppard, P. E.¹ ¹University of Wisconsin, Madison, Madison, WI, ²University of Wisconsin, Madison, Madison, WI.

Introduction: Several aspects of sleep - collectively conceptualized as 'sleep health' - are associated with anxiety and depression. This study investigated whether specific components of sleep health experienced during midlife are associated with depression and anxiety symptoms in later life.

Methods: A subset of Wisconsin Sleep Cohort participants (n=616; 45% female; mean [SD] baseline age=55 [8] years) completed 4 study visits at 4-year intervals. Visits included polysomnography and questionnaires about sleep, mood, and health. Outcomes (Zung depression score, State and Trait Anxiety) were regressed on sleep health characteristics (AHI, %N3 sleep, %REM sleep, sleep efficiency, sleep latency, sleep duration, sleep debt, nap duration, insomnia symptoms, circadian preference, excessive daytime sleepiness [EDS], Epworth Sleepiness Scale [ESS]) using 2 types of linear models adjusting for age, sex, BMI, education, exercise, smoking, and caffeine consumption: 1) longitudinal models in which baseline sleep health predicted mood outcomes 12 years later (adjusting for baseline levels of the outcome variable), and 2) models in which 12-year change in sleep health predicted 12-year change in outcomes.

Results: Longer nap duration, evening circadian preference, and EDS during midlife were associated with worse depression scores in later life. 12-year increases in nap duration, EDS, and ESS were associated with 12-year worsening of depression. Longer sleep duration and greater EDS during midlife were associated with worse trait anxiety in later life. 12-year increases in sleep duration, nap duration, insomnia symptoms, EDS and ESS were associated with 12-year worsening of trait anxiety. Greater AHI and EDS during midlife were associated with worse state anxiety in later life. 12-year increases in ESS were associated with worse state anxiety in later life. 12-year increases in ESS were associated with worse state anxiety in later life. 12-year increases in ESS were associated with worsening state anxiety. (P<0.05 for all reported results.)

Conclusion: Multiple aspects of sleep health experienced during midlife are associated with greater depression and anxiety in later life.

Support: NIH grants: National Institutes of Aging (R01AG058680; R01AG036838); National Heart, Lung, and Blood Institute (R01HL62252); National Center for Research Resources (1UL1RR025011)

0834

DIFFERENT NEURAL CIRCUITS WERE ASSOCIATED WITH DIFFERENT PARTS OF RELATIONSHIPS AMONG SLEEP, PAIN AND ANXIETY IN WOMEN WITH PRIMARY DYSMENORRHOEA

Chein, K.¹ Wei, S.¹ Hung, C.³ Tu, C.⁴ Hsieh, J.⁵

¹National Cheng Kung University Hospital, Tainan city, TAIWAN, ²National Cheng Kung University Hospital, Tainan city, TAIWAN, ³Taipei City Hospital, Taipei City, TAIWAN, ⁴China Medical University, Taichung City, TAIWAN, ⁵National Yang-Ming University, Taipei City, TAIWAN.

Introduction: Although the relationships amongst sleep, pain and anxiety have been evidenced, the underlying neurological mechanisms remain elusive. Primary dysmenorrhea is a good model of spontaneous pain with clear painful (menstruation, *state*) and pain free (e.g., peri-ovulatory phase, *trait*) conditions. We sought to investigate the *state-* and *trait*-related neural signatures that link sleep and anxiety in primary dysmenorrhea.

Methods: Thirty female subjects with primary dysmenorrhea and 30 healthy female controls completed resting-state functional magnetic resonance imaging, the State-Trait Anxiety Inventory and sleep quality assessments during menstruation and peri-ovulatory phases. As we have reported that there was altered default mode network in the primary dysmenorrhea, posterior parietal cortex (PPC) was therefore chosen as the seed to elucidate the functional connectivity that may correlate with sleep and anxiety.

Results: The primary dysmenorrheic subjects exhibited sleep disturbances throughout the menstrual cycle with increased *state* anxiety. The primary dysmenorrheic subjects demonstrated significantly higher correlations between the sleep quality and the PPC-insula and -putamen functional connectivity during the peri-ovulatory phase. During menstruation, the primary dysmenorrheic subjects demonstrated significantly lower correlations between their *state*-anxiety scores and the PPC-occipital cortex functional connectivity.

Conclusion: After regressing out the effects of *trait* anxiety, the correlation between sleep quality and the PPC-putamen functional connectivity was not significant, indicating anxiety-mediated sleeppain relationship. The PPC-insula functional connectivity, by contrast, was remained significant. Furthermore, *state* anxiety was associated with the PPC-occipital cortex functional connectivity that was significantly impaired in primary dysmenorrheic subjects. These findings hinted pain and anxiety mediating sleep quality in different neurological circuits.

Support: Taipei City Goverment(10401-62-046 and 10501-62-046)

0835

ELUCIDATING THE EFFECT OF SLEEP APNEA ON COGNITIVE HEALTH: A PRELIMINARY REPORT

Ramos, A. R.¹ Alperin, N.² Junco, B.³ Lee, S.²

Hernandez-Cardenache, R.⁴

¹University of Miami, Miller School of Medicine, Miami, FL, ²Department of Radiology, Miller School of Medicine, University of Miami, FL, ³Department of Neurology, Miller School of Medicine, University of Miami, FL, ⁴Department of Psychiatry, Miller School of Medicine, University of Miami, FL.

Introduction: We aim to determine the cognitive domains associated with obstructive sleep apnea (OSA) age-related brain atrophy in a sample of middle-aged to older males.

Methods: We evaluated consecutive treatment naïve male OSA patients (AHI \geq 15) without dementia, stroke or heart disease (infarction, heart failure), from March to November of 2019. We obtained demographic variables, vascular risk factors, the Epworth sleepiness scale (ESS) and the Pittsburgh sleep quality index (PSQI). We also obtained computerized neurocognitive testing with the Go-NoGo Response Inhibition Test, Stroop Interference Test, Catch Game Test, Staged Information Processing Speed Test,

Verbal Memory Test and Non-Verbal Memory Test. We derived domain-specific Z-scores age and education adjusted for global cognition, memory, attention, processing speed and executive function. Pearson correlation was used to evaluate bivariate associations between the sleep exposures and neurocognitive outcomes. Linear regression was used to evaluate associations between AHI and neurocognitive domains, adjusting for the ESS.

Results: A total of 15 participants 40 to 76 years of age, 73% of Hispanic/Latino background, completed neurocognitive testing. The average ESS was 8.2 ± 6.0 , PSOI= 5.7 ± 4.9 , and AHI= 48.9 ± 25.5 . Hypertension was seen in 66% and diabetes in 27%. The AHI was correlated with global cognition (r= -0.66; p=0.008), memory (r= -0.73; p=0.002) and attention (r= -0.67; p =0.007), but not executive function or processing speed. In addition, the AHI correlated with verbal memory (r = -0.76; p = 0.001), but not with non-verbal memory. In adjusted models, the AHI was associated with global cognition (β = -0.60; p=0.05) and decreased memory (β = -0.85; p=0.006). However, the association with attention was explained by the ESS. The PSQI was not correlate with the cognitive domains. Conclusion: In this pilot-study, the AHI was associated with decreased global cognition, and verbal memory accounting for sleepiness. Findings suggest the left-hippocampus as a region vulnerable to early age-related brain loss in OSA.

Support: Scientific Advisory Committee, Pilot grant, Miller School of Medicine; R21AG056952; R21HL140437.

0836

SELF-REPORTED DIFFICULTY INITIATING SLEEP AND EARLY MORNING AWAKENINGS ARE ASSOCIATED WITH NOCTURNAL DIASTOLIC NON-DIPPING IN OLDER MEN

Tan, X. Benedict, C.

Uppsala University, Uppsala, SWEDEN.

Introduction: Aging increases the risk of insomnia and elevated blood pressure (BP). Here, we examined in older men whether reports of difficulty falling asleep (DIS) and early morning awakenings (EMA) are associated with 24-h BP and heart rate.

Methods: We utilized variables from 995 men (mean age: 71 years) who participated in the Uppsala Longitudinal Study of Adult Men (ULSAM). BP and heart rate were measured over 24 hours.

Results: Non-dippers (night-to-day BP ratio > 0.90) had a higher risk of hypertension than dippers (systolic non-dippers vs. systolic dippers, OR [95%CI]: 1.64 [1.21, 2.21], P=0.001; diastolic nondippers vs. diastolic dippers, 1.50 [1.10, 2.04], P=0.01). Compared to men without DIS, men who reported DIS (10% of the cohort) had a higher risk of diastolic non-dipping (1.85 [1.19, 2.87], P=0.006). Similarly, men who reported EMA (19% of the cohort) had a higher risk of diastolic non-dipping than those without EMA (1.59 [1.12, 2.24], P=0.009). Despite a slightly higher nocturnal diastolic BP among men with EMA vs. those without EMA (+1.4 mmHg, P=0.035), no other differences in BP and heart rate were found between men with and those without insomnia complaints.

Conclusion: Our findings uncover a link between disruption in nocturnal dipping of diastolic BP and insomnia symptoms related to difficulty initiating sleep and early morning awakening in older men.

Support: Authors' work is funded by the Novo Nordisk Foundation (C.B., NNF19OC0056777), Swedish Brain Research Foundation (C.B., FO2019-0028), Swedish Research Council (C.B., 2015-03100), Åke Wiberg Foundation (X.T., M18-0169, M19-0266), Fredrik and Ingrid Thuring Foundation (X.T., 2018-00365), and the Swedish Medical Research Society (X.T., P18-0084).

SLEEP, Volume 43, Abstract Supplement, 2020

WELL-BEING PREDICTS SLEEP DISTURBANCE IN A PROSPECTIVE COHORT OF OLDER ADULTS

Hershner, S. D.¹ Swanson, L. M.² Meng, A.³ Jansen, E. C.⁴ Burke, J. F.⁵ Braley, T. J.⁵ Dunietz, G. L.¹

¹Department of Neurology, Division of Sleep Medicine, Ann Arbor, MI, ²Department of Psychiatry, University of Michigan, Ann Arbor, MI, ³Department of Statistics, University of Michigan, Ann Arbor, MI, ⁴Department of Nutritional Sciences, University of Michigan, Ann Arbor, MS, ⁵Department of Neurology, University of Michigan, Ann Arbor, MI.

Introduction: Lower well-being negatively impacts health among older adults. Optimal sleep - a determinant of health - has been associated with higher well-being. Several domains of well-being, e.g., mindfulness and purpose in life have been shown to improve sleep. But, whether well-being impacts sleep remains unclear. This study examined associations between well-being and sleep duration, sleep quality, and incident insomnia symptoms among a nationally representative sample of older US adults.

Methods: This study analyzed data from the 2011-2013 National Health and Aging Trends Study (NHATS), a longitudinal, annual survey of community-dwelling Medicare beneficiaries. The exposure, a validated scale of well-being used questions on purpose, emotion, and self-satisfaction and divided responses into quartiles. Sleep outcomes included sleep duration, sleep quality, and insomnia symptoms. Unadjusted and adjusted linear and logistic regression models examined relationships between the health characteristics and well-being score in 2012 and sleep outcomes in 2013. Covariates included demographics and health characteristics.

Results: Half of study participants (n=2,000) were women. The mean sleep duration was 7.2 and 7.3 (standard error(SE) ± 0.1) for men and women. Poor sleep quality was reported by 30% of subjects and more frequently among Hispanic subjects, older adults, and those with less education. The mean well-being score was 17.2 (SE ± 0.07). Higher well-being scores correlated with male gender, younger age, higher education, marriage, and increased physical activity. Well-being scores in the 2nd - 4th quartile had lower odds of poor sleep quality (4th quartile adjusted odd ratio 0.24 (95% CI 0.15, 0.38). The highest well-being quartile had a 4-fold lower incidence of insomnia symptoms. Well-being scores were not associated with sleep duration

Conclusion: Higher well-being may protect older adults against the development of insomnia and poor sleep quality. Strategies to improve well-being could offer an innovative way to improve the health of older Americans though better sleep. **Support:** none

0838

SEX SPECIFIC CHANGES IN SLEEP MACRO-STRUCTURE WITH OBSTRUCTIVE SLEEP APNEA IN A LARGE CLINICAL POPULATION OF OLDER ADULTS

Mullins, A. E. Bagchi, N. Parekh, A. Kam, K. Wang, J. Williams, M. K. Rapoport, D. M. Ayappa, I. Burschtin, O. E. Varga, A. W.

Icahn School of Medicine at Mount Sinai, New York, NY.

Introduction: Sleep architecture is influenced by age and sex and is disrupted by obstructive sleep apnea (OSA) and periodic limb movements (PLM) of sleep. Although increasing OSA severity is thought to decrease both REM and slow wave sleep (SWS), it may do so in non-linear ways. Here, we aim to 1) compare sleep

macrostructure between older men and women, 2) compare metrics of total and REM-specific OSA severity between older men and women, and 3) examine associations between metrics of OSA severity and REM sleep and SWS in a clinical sample.

Methods: Clinical in-lab diagnostic polysomnography (PSG) in adults ≥64 years of age from the greater New York area recorded between 2006- 2016 were collated including demographic and traditional sleep scoring metrics. Studies where TST < 4 hours were removed. Demographic, sleep macrostructure, OSA (AHI4% & AHI3A criteria), pulse oximetry (SpO2) nadir and PLM measures were compared according to sex.

Results: PSGs from 1282 older adults (average age 70 years in both sexes, 41% female) were included in the analyses. Women had a significantly greater SWS% (14.5 vs 7.9, p<0.001) and less N1% (18.2 vs 24.4, p<0.001), without significant differences in TST, N2%, REM%, sleep efficiency or SpO2 nadir. Men had significantly higher all-sleep OSA (median AHI4% 8.8 vs 11.1, p=0.0004; median AHI3A 24.4 vs 27.9, p=0.003) and PLM's (4.0 vs 7.6/hour, p=0.008) but women had significantly more OSA during REM sleep (median REM AHI4% 16.7 vs 14.0, p=0.01; median REM AHI3A 32.6 vs 27.4, p=0.0002). Inverse non-linear associations were observed between OSA severity and %SWS and %REM with a unique pattern for each sleep stage. The pattern between men and women within each stage appeared similar.

Conclusion: In this clinical sample of older adults, women exhibit a greater proportion of SWS and worse REM-related OSA then men. Increasing OSA severity is associated with non-linear reductions in %SWS and %REM, and we plan to further investigate these relationships and sexual dimorphism by using quantitative analysis of PSG signals for more precise measures of slow wave activity and breathing physiology than traditional sleep scoring metrics.

Support: R01AG056682

0839

NAPPING STIGMA AMONG FRAIL OLDER ADULTS: REFLECTIONS FROM A QUALITATIVE STUDY

Berkley, A. S.¹ Carter, P. A.²

¹University of Kansas School of Nursing, Kansas City, KS, ²Capstone College of Nursing, The University of Alabama, Tuscaloosa, AL.

Introduction: Napping and other daytime sleep is often overlooked in insomnia research and poorly defined in many studies. Research has shown some correlations between older adults' napping habits and increased medical co-morbidities and risks of dementia, but it has also shown that napping enhances memory consolidation and broader aspects of cognition in younger adults. Where along the aging spectrum this line between beneficial napping and potentially risky napping falls is not clear.

Methods: This study employed a qualitative descriptive approach in which semi-structured interviews (N=18) were supplemented by the widely used self-report instruments and anxiety scales.

Results: Insomnia in these older adults directly resulted in reduced energy and stamina, poor mood, and reduced functional capacity. Indirect effects included reduced social interaction and increased isolation. Several participants reported napping in qualitative interviews but denied daytime sleep on standard sleep assessments, and associated napping with anxiety and dread of functional and cognitive decline. Planned or intentional napping was viewed with guilt and denial, while dozing off accidentally was considered an acceptable coping strategy.

Conclusion: While research about the relationships between disordered sleep and cognitive impairment is still at an early stage, it seems ironic that the participants in this study stigmatized planned napping, which could potentially benefit their cognitive functioning, but seemed accepting of accidental napping, which may well indicate some more serious cognitive issues. More education about sleep needs for older adults is needed.

Support: I am grateful to the Longhorn Village chapter of Texas Exes for their Gerontology Nursing Scholarship, which helped to fund this project.

0840

SLEEP QUALITY IN CLINICALLY INDICATED IN-LABORATORY POLYSOMNOGRAPHY

Roth, R. H.¹ Harrison, E.² Kang, H.³ Lobo, J.⁴ Logan, J.⁵ Sohn, M.⁶ Kwon, Y.¹

¹University of Virginia School of Medicine, Charlottesville, VA, ²UPMC Children's Hospital of Pittsburgh, Pittsburgh, PA, ³University of Illinois College of Applied Health Sciences, Champaign, IL, ⁴University of Virginia Public Health Sciences, Charlottesville, VA, ⁵University of Virginia School of Nursing, Charlottesville, VA, ⁶University of Kentucky College of Public Health, Lexington, KY.

Introduction: Few studies have explored how patients sleep or what characteristics might be predictive of poor sleep during clinically-indicated polysomnography (PSG) in an in-laboratory setting.

Methods: We reviewed clinically indicated diagnostic PSG studies completed over a 10-year period in a single academic sleep center. Total sleep time (TST) and sleep efficiency (SE) were used as proxies for sleep quality. Patients were categorized as normal or poor sleepers based on TST <4 hours or SE <50%. Multivariate linear and logistic regression analyses were performed to determine factors associated with sleep quality while controlling for demographics, medications, comorbidities and measures of sleep.

Results: We included 4957 patients, who were mostly female (58.9%), middle-aged (52.9 y), Caucasian (69.3%), and overweight or obese (91.3%). 3682 patients (74.2%) were diagnosed with sleep apnea (Apnea Hypopnea Index(AHI)>5/hr).

Average TST was 5.75 ± 1.43 hours (Interquartile range [IQR] = 4.94 - 6.73) and average SE was $75.1\%\pm16.1\%$ (IQR=66.9 - 87.2). TST and SE were lower for males compared to females (5.48 vs 5.93 hr, p<0.001; 73% vs 77%, p<0.001). In multivariable analysis, older age (TST: OR = 1.04, 95% CI:[1.03,1.05]; SE: OR = 1.04, 95% CI:[1.04,1.05]), male sex (TST: 1.38,[1.14,1.68]; SE: 1.34,[1.07,1.68]), normal body habitus (TST: 1.47,[1.02,2.08]; SE: 1.51,[1.01,2.27]) and a higher AHI (TST: 1.02,[1.02,1.03]; SE: 1.02,[1.003,1.03]) were significantly associated with being a poor sleeper for both TST and SE. Antidepressant use was associated with poor sleep for TST (0.77, [0.59,1]), but not for SE (0.98, [0.73,1.3]).

Conclusion: Sleep quality during the in-laboratory PSG differed by sex, age and presence of sleep apnea. Sleep quality during in-lab PSG is thought to be compromised by obtrusive monitoring and an unfamiliar environment, but average sleep quality may be higher than expected for patients in the laboratory. Future studies should consider examining in-lab sleep quality in different patient populations.

Support: N/A

0841

TRAJECTORIES OF SLEEP QUALITY DURING THE PERINATAL PERIOD IN THE "LIFE-ON" STUDY

Garbazza, C.¹ Castronovo, V.² Sforza, M.² Manconi, M.¹ ¹Sleep and Epilepsy Center, Neurocenter of Southern Switzerland (EOC), Lugano, SWITZERLAND, ²IRCCS San Raffaele Scientific Institute, Department of Clinical Neurosciences, Neurology - Sleep Disorders Center, Milan, ITALY.

Introduction: The perinatal period is characterized by poor sleep quality, with a worsening trend across pregnancy, peaking in the first month after delivery and often persisting during the postpartum. Consequences of perinatal sleep problems range from negative obstetric outcomes, to mood disturbances in the mothers and neurobehavioral deficits in the infants. Therefore, an early identification of at-risk women is highly relevant.

Methods: As part of the "Life-ON" project, a multicenter study on sleep and mood changes during the perinatal period, 120 women (aged 36.31±4.21 years) were repeatedly evaluated with the Pittsburgh Sleep Quality Index (PSQI), Insomnia Severity Index (ISI), Epworth Sleepiness Scale (ESS), Edinburgh Postnatal Depression Scale (EPDS) and Montgomery-Åsberg Depression Rating Scale (MADRS), from the first gestational trimester (baseline) to 12-months postpartum. Using latent class analysis (LCA) we identified different subtypes according to baseline evaluation and analyzed their distinct trajectories of sleep quality based on PSQI.

Results: We chose 3 latent classes that were statistically different in all scales at baseline (p<0.05), with lowest values in the stable-low class, followed closely by a mild class and highest values in the stable-high class (mean \pm SD for each class, respectively: EPDS 2.58 \pm 2.34 vs. 3.54 \pm 2.87 vs. 7.22 \pm 3.1, MADRS 1.75 \pm 1.83 vs. 4.08 \pm 2.67 vs. 7.17 \pm 3.74, PSQI 2.50 \pm 1.26 vs. 4.89 \pm 1.73 vs. 8.00 \pm 3.26, ISI 1.40 \pm 1.58 vs. 6.46 \pm 2.90 vs. 11.13 \pm 4.28). This trend remained significant for the whole observation period. Focusing on PSQI, the mild (n=37) and stable-low class (n=60) maintained a mean score below 5, only peaking above 5 around delivery. In contrast, the stable-high class (n=23) showed a constant mean PSQI>5 throughout the perinatal period (first trimester=8.00 \pm 3.26; second trimester=8.26 \pm 3.41; third trimester=8.39 \pm 3.90; 20-days postpartum=7.35 \pm 3.79; 12-months postpartum=6.83 \pm 3.35).

Conclusion: We identified a latent class of women with a stablehigh poor sleep quality (PSQI>5) across the perinatal period. Determining baseline sleep and mood characteristics in early pregnancy can help predicting distinct sleep trajectory subgroups in the perinatal period and detecting at-risk women, who should be targeted by early preventive and therapeutic interventions.

Support: The "Life-ON" project is supported by the Swiss National Science Foundation (grant: 320030_160250/1) and the Italian Ministry of Health and Emilia-Romagna Region (grant: PE-2011-02348727).

0842

THE MODERATING EFFECTS OF SLEEP AND SEX ON PHYSICAL ACTIVITY AND COGNITIVE FUNCTIONING IN AFRICAN AMERICANS

Beard, B.¹ Ramirez-Ruiz, M.¹ Mwendwa, D. T.¹ Sims Wright, R.² ¹Howard University, Washington, DC, ²University of Delaware, University of Delaware, DE. **Introduction:** Research suggests modifiable lifestyle behaviors are associated with delayed cognitive decline. Identifying modifiable lifestyle behaviors in African Americans is critical because they are more likely to be diagnosed with Alzheimer's disease and other related dementias. Evidence shows the positive effects of physical activity (PA) on cognitive functioning. Sleep quality also impacts cognitive functioning. Therefore, the purpose of this study examined how sleep quality and sex moderated the relationship between PA and cognitive functioning in African Americans.

Methods: A sample of 147 African Americans (mean age 59) completed the Pittsburgh Sleep Quality Inventory (PSQI) and a battery of cognitive tests (The Delis-Kaplan Executive Function System (DKEFS) Verbal Fluency Test, The Stroop Color Word Test and Trail Making Test). Participants completed a participant screener to determine PA frequency and intensity. Anthropomorphic measures were also obtained. A moderated moderation analysis was conducted.

Results: Poor sleep quality was associated with poorer performance on a verbal fluency task after controlling for age, BMI and years of education (B=-1.37, p=.012). There was a significant interaction between PA frequency and sleep quality on verbal fluency performance (B=0.42, p=.047). Additionally, there was a significant interaction between sleep quality and sex on verbal fluency performance (B=0.80, p=.016). Further adjustments revealed significant conditional effects such that more frequent PA per week improved verbal fluency performance among women, who reported better sleep quality (B=0.20, p=0.03).

Conclusion: The current study suggests sleep quality strengthens the relationship between PA and cognitive performance, specifically in African-American women. Consistent with previous studies, our findings support the need to target sleep quality and PA in women as modifiable lifestyle factors that may delay cognitive decline. **Support:** This research was funded by the Office of the Provost at Howard University.

0843

PROSPECTIVE ASSOCIATIONS OF INSOMNIA SYMPTOMS WITH HEALTH SERVICES USE IN COMMUNITY-DWELLING OLDER ADULTS

Tzuang, M.¹ Owusu, J. T.² Huang, J.³ Sheehan, O. C.^{3,4} Rebok, G. W.^{1,3,5} Kasper, J.⁶ Spira, A. P.^{1,3,5}

¹Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ²Independent Researcher, Oakland, CA, ³Center on Aging and Health, Johns Hopkins University, Baltimore, MD, ⁴Division of Geriatric Medicine and Gerontology, Johns Hopkins School of Medicine, Baltimore, MD, ⁵Department of Psychiatry & Behavioral Sciences, Johns Hopkins School of Medicine, Baltimore, MD, ⁶Department of Health Policy and Management, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD.

Introduction: Limited research has examined links of insomnia with health services use, particularly using claims-based data. We investigated the association of insomnia symptoms with costly health services use, measured by Medicare claims, in a nationally representative sample of U.S. older adults.

Methods: Participants were 4,302 community-dwelling Medicare beneficiaries aged ≥65 years from Round 1 (2011) of the National Health and Aging Trends Study who had continuous fee-forservice Medicare coverage 1 year before and after the Round 1 interview. Participants reported past-month insomnia symptoms (i.e., sleep onset latency >30 minutes, difficulty returning to sleep after early awakening) which we categorized as 0, 1, or 2 symptoms. Outcomes were linked Medicare claims occurring after Round 1 interviews: emergency department (ED) visits, all-cause hospitalizations, preventable hospitalizations, all-cause 30-day readmissions, home health care (all measured as yes/no); and number of hospitalizations and ED visits.

Results: Overall, 18.9% of participants were hospitalized, 29.3% visited the ED, 3.1% had a preventable hospitalization, 2.6% had a readmission, and 11.7% used home health care. After adjustment for demographics, compared to participants with no insomnia symptoms, those with 2 symptoms had a higher odds of ED visits (odds ratio (OR)=1.42, p<0.001), all-cause hospitalizations (OR=1.30, p<0.01), preventable hospitalizations (OR=1.83, p<0.05), 30-day readmissions (OR=1.73, p<0.05), and home health care use (OR=1.27, p<0.05). These associations did not hold, however, upon further adjustment for health characteristics (i.e., depressive/anxiety symptoms, medical comorbidities and BMI). After full adjustment, reporting 2 insomnia symptoms, versus no insomnia symptoms, was associated with a greater number of ED visits (Incidence Rate Ratio=1.16, p<0.05).

Conclusion: Among older adults, a greater number of insomnia symptoms is associated with greater health services use. Insomnia symptoms may be a marker of, or exacerbate, health conditions. Targeting insomnia may lower health services use.

Support: National Institute on Aging: R01AG050507 & R01AG050507-02S (PI: Spira); F31-AG058389; U01AG032947 (PI: Kasper) for the National Health and Aging Trends Study. Johns Hopkins Center on Aging and Health Data Use Agreement (PI: Roth, Co-I: Sheehan) with Centers for Medicare & Medicaid Services titled, "Potentially modifiable factors influencing outcomes in NHATS."

0844

LINKS OF NAPPING WITH SUBSEQUENT ALL-CAUSE HOSPITALIZATIONS AND EMERGENCY DEPARTMENT VISITS IN OLDER ADULTS

Tzuang, M.¹ Owusu, J. T.² Huang, J.³ Sheehan, O. C.^{3,4} Rebok, G. W.^{1,3,5} Kasper, J.⁶ Spira, A. P.^{1,3,5}

¹Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ²Independent Researcher, Oakland, CA, ³Center on Aging and Health, Johns Hopkins University, Baltimore, MD, ⁴Division of Geriatric Medicine and Gerontology, Johns Hopkins School of Medicine, Baltimore, MD, ⁵Department of Psychiatry & Behavioral Sciences, Johns Hopkins School of Medicine, Baltimore, MD, ⁶Department of Health Policy and Management, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD.

Introduction: Few studies have examined whether napping is associated with objective measures of health services use. We investigated links of napping characteristics with all-cause hospitalizations and emergency department (ED) visits in Medicare claims from a nationally representative sample of older adults.

Methods: Participants were 869 community-dwelling Medicare beneficiaries aged ≥65 years from Round 3 (2013) and Round 4 (2014) of the National Health and Aging Trends Study (NHATS) who had continuous fee-for-service coverage 1 year before and after the NHATS interview. Participants reported past-month napping frequency (re-categorized as non-nappers, infrequent nappers, frequent nappers), napping type (intentional, unintentional), and nap duration. Outcomes were from linked Medicare claims measured

after napping assessment: all-cause ED visits and hospitalizations (yes vs. no), and number of hospitalizations and ED visits.

Results: Overall, 45.4% of participants were nappers, and 55.2% of the nappers reported taking unintentional naps. After adjustment for demographics, depressive/anxiety symptoms, medical comorbidities and BMI, compared with non-nappers, infrequent and frequent nappers had a higher odds of hospitalization (odds ratio (OR)=1.65 and 1.73, respectively, both p<0.05), as did unintentional nappers (OR=1.85, p<0.05). We found no significant adjusted associations of napping frequency with ED visits. However, compared with non-nappers, unintentional nappers had a higher odds of visiting the ED (OR=1.94, p<0.01). Additionally, compared to nappers taking short naps (\leq 30 minutes), those with naps >60 minutes had a greater number of ED visits (Incidence Rate Ratio=1.99, p<0.05).

Conclusion: Among older adults, napping—and particularly unintentional napping—may be a modifiable risk factor for health services use. More studies that consider multiple napping characteristics (e.g., duration, frequency), and using objective measures (e.g., actigraphy), are needed to advance understanding of how napping might influence health services use.

Support: National Institute on Aging: R01AG050507 & R01AG050507-02S (PI: Spira); F31-AG058389; U01AG032947 (PI: Kasper) for the National Health and Aging Trends Study. Johns Hopkins Center on Aging and Health Data Use Agreement (PI: Roth, Co-I: Sheehan) with Centers for Medicare & Medicaid Services titled, "Potentially modifiable factors influencing outcomes in NHATS."

0845

THE DIFFERENCE IN SLEEP CHARACTERISTICS OF CHRONIC INSOMNIA DISORDER ACCORDING TO GENDER AND AGE

Hong, J. Lee, H. Yoon, I. Seoul National University Bundang Hospital, Seongnam, KOREA, REPUBLIC OF.

Introduction: Impacts of age and gender on sleep have been reported in normal population, but rarely in chronic insomnia disorder (CID). This study aimed to investigate difference in sleep characteristics of CID according to gender and age.

Methods: The participants with drug-naïve CID and aged between 40 and 79 years were recruited. We compared subjective and objective sleep parameters between the middle-aged (40-64 years, N=86) and the elderly (65-79 years, N=50), and between men (N=45) and women (N=91). The subjective sleep quality and habitual sleep time were measured by Pittsburgh Sleep Quality Index (PSQI). The participants were asked to wear an actigraph for 4 days to obtain objective sleep parameters.

Results: In the PSQI, the elderly reported earlier bedtime and wake-up time (p=0.018; p=0.026), reduced total sleep time (TST) and sleep efficiency (p=0.003; p=0.011), and low sleep quality (p=0.034) compared to the middle-aged. However, according to the actigraphy, differences were observed only in the bedtime (p=0.016) and the wake-up time (p=0.002) between the two age groups. Between genders, the actigraphy showed that the male patients woke up earlier than the female group (p=0.015); except for this finding, there was no significant gender effect. Meanwhile, regarding gender and age interactions, the elderly women with CID showed longer time in bed (TIB) with increase in both TST and wake after sleep onset (WASO) compared to the middle-aged women. The elderly men showed decreased TIB and TST, and slightly decreased WASO than the middle-aged men.

Conclusion: The elderly with CID show more subjective sleep complaints than the middle-aged CID despite little difference in objective sleep characteristics, which suggests that the elderly CID may seek medical help more than the middle aged. As women with CID get older, they increase time spent in bed to maintain sleep time, but with resultant increase in wake.

Support: None

0846

REPORTS OF SLEEP SYMPTOMS IN YOUNG ADULTS OF COLLEGE AGE

Thatipelli, S.¹ Abbasi, A. A.²

¹College of Life Sciences, University of California Los Angeles, Los Angeles, CA, ²Southern Arizona VA Health Care Systems, Tucson, AZ.

Introduction: Young adults of college age in the United States often report inadequate sleep. The objectives of this study were to identify a) how often young adults of college age report symptoms of insomnia, fatigue and mood disorder and b) identify differences in symptom reporting based on their sleep duration.

Methods: This is a cross-sectional study, using data from 2013 through 2018 of the National Health Interview Survey (NHIS). 11,028 subjects, ages 18-22 years, for whom information was available on duration of sleep were included. We analyzed reports of trouble falling sleep, trouble staying sleep, non-restorative sleep, fatigue and symptoms of mood disorder. Subjects were divided into 2 groups: \leq 7 hours and > 7 hours of sleep. Data was analyzed using Complex Sample Analysis of IBM SPSS version 26.

Results: Out of the 11,028 subjects 50.7% were male and 49.3% were female. Mean sleep duration for all subjects was 7.44 \pm 0.02 hours (mean \pm SE). 13.5% subjects reported trouble falling asleep while 10.6% reported trouble staying asleep for \geq 4 nights per week. Non-restorative sleep and frequent fatigue were reported by 33.8% and 12.4% respectively. Daily/weekly symptoms of anxiety and depression were reported by 23.9% and 8.6% respectively. There were 48.4% subjects with \leq 7 hours and 51.6% with >7 hours of sleep. When compared to subjects with \leq 7 hours to >7 hours of sleep, trouble falling asleep, trouble staying asleep and non-restorative sleep were reported by 19.9% vs. 7.5%, 14.8% vs. 6.6% and 45.9% vs. 22.4% respectively. Symptoms of fatigue, anxiety and depression reported by subjects with \leq 7 hours vs. >7 hours of sleep were: 18.6% vs. 6.6%, 26.9% vs. 21% and 10.2% vs.7% respectively.

Conclusion: Young adults with \leq 7 hours of sleep are more likely to report trouble falling sleep, trouble staying sleep, non-restorative sleep, fatigue and symptoms of mood disorder. **Support:** None

0847

ASSOCIATION BETWEEN CANNABIS USE FREQUENCY AND SLEEP DURATION AMONG A REPRESENTATIVE SAMPLE OF US ADULTS

Bowles, N. P. Shea, S. A.

Oregon Health and Science University, Portland, OR.

Introduction: Cannabis use is on the rise in the US, and while the cannabis plant and related compounds are considered to have low toxicity, the impact on physiology including sleep remains unclear. Further, the bulk of cannabis research has focused on adolescents and young adults despite growing use among the elderly. Thus, this analysis sought to determine the impact of the frequency of cannabis use on sleep duration and determine if this relationship varies by age.

SLEEP, Volume 43, Abstract Supplement, 2020

Methods: This cross-sectional study used data from the 2016-2018 Behavioral Risk Factor Surveillance System surveys. Multinomial logistic regression was used to evaluate the association between the frequency of cannabis use, 0-30 times over the prior 30 days; and average sleep duration, short (<7 hours); recommended (7-9 hours); and long (>9 hours) sleep.

Results: The study sample included 235,667 participants (48% male, 43% 18-44 years old and 35% 44-64 years old) of which 14,122 consumed cannabis. The weighted proportion who reported using cannabis 1-4x, 5-15x, or more than 16x/month were 3.1% (95% CI, 3.0%-3.3%), 2.1% (2.0%-2.2%), and 4.4% (4.2%-4.6%) respectively. In an age stratified model adjusted for sociodemographic and clinical characteristics, there were no associations between sleep and cannabis use up to 15x/month. Among those adults who consumed cannabis more than 16x/month: (1) 18-44 year olds were more likely to report either short sleep (risk ratio (RR), 1.21; 95% CI, 1.05-1.39) or long sleep (RR, 1.57; 95% CI, 1.11-2.22) as opposed to the recommended amount of sleep; whereas (2) 44-64 year olds demonstrated an increased risk for long sleep (RR, 1.77; 95% CI, 1.09-2.89); and (3) participants 65 years and older demonstrated an increased risk for short sleep (RR, 1.61; 95% CI, 1.04-2.49).

Conclusion: We demonstrate that modest cannabis use was not associated with sleep, whereas daily cannabis use is associated with age-dependent sleep duration above and below the recommended amount.

Support: KL2TR002370, 2R25HL105444-09.

0848

THE RELATIONSHIP BETWEEN SLEEP DISTURBANCE & SLEEP HYGIENE IN JAPANESE PREGNANT WOMEN

ASAKA, y.¹ Morioka, A.²

¹Hokkaido University, Sapporo, JAPAN, ²Faculty of Health Sciences, Hokkaido University, Sapporo, JAPAN.

Introduction: Pregnant women are more likely to develop sleep disorders due to poor sleep quality. Sleep hygiene has been reported as a method for dealing with sleep disorders. However, studies on the relationship between sleep hygiene and sleep disorders in Japanese pregnant women remain insufficient. This study aimed to clarify the actual state of sleep disorders during pregnancy and the relationship between sleep disorders and sleep hygiene.

Methods: A cross-sectional study was conducted on 147 Japanese pregnant women. We used the Japanese versions of the Pittsburgh Sleep Quality Index (PSQI) and the Epworth Sleepiness Scale (JESS) for evaluation of sleep. We used 32 items of habitual behavior important for sleep improvement for evaluation of sleep hygiene; these items were self-selected.

Results: The average PSQIG scores of women in their first, second, and third trimesters were 5.6 ± 4.3 , 5.4 ± 2.4 , and 6.6 ± 2.8 , respectively; 22.2%, 46.4%, and 62.2% of women in their first, second, and third trimesters, respectively, exceeded the cut-off score. The average JESS scores were 12.2 ± 4.5 , 9.9 ± 4.1 , and 10.0 ± 4.6 for first, second, and third trimester women, respectively; 55.6%, 35.7%, and 41.9% of first, second, and third trimester women, respectively, slept excessively during the day (JESS score>11). Following factor analysis, sleep hygiene was categorized into four factors: "thinking positively," "adding rhythm to the day," "avoiding going out and caffeine before bedtime," and "avoiding eye irritation." Pregnant women who "added rhythm to their day" had lower PSQI scores, and sleep hygiene was not related to JESS scores in the multiple regression analysis of sleep hygiene and sleep disorders.

Conclusion: Sleep disorders in pregnancy are related to habitual behavior such as being active during the day, eating and sleeping regularly, and basking in the sunlight every morning. **Support:**

0849

ASSOCIATION OF SLEEP DISTURBANCE WITH NECK CIRCUMFERENCE, BODY MASS INDEX & BLOOD PRESSURE IN PREGNANT JAPANESE WOMEN

Morioka, A.¹ ASAKA, y.²

¹Faculty of Health Sciences, Hokkaido University, Sapporo, JAPAN, ²Faculty of Health Sciences, Hokkaido University, Sapporo, JAPAN.

Introduction: Snoring is one of the symptoms of sleep-disordered breathing. Sleep-disordered breathing is associated with blood pressure in pregnant women. However, studies reporting this association have been conducted overseas, and there is a lack of research in the Japanese context, where women have different life-styles and physical attributes. The aim of this study is to clarify the association of sleep disturbance with physical factors in pregnant Japanese women.

Methods: A cross-sectional study was conducted with 80 pregnant Japanese women. The assessments for statistical analysis included the Japanese versions of the Pittsburgh Sleep Quality Index (J-PSQI) and Epworth Sleepiness Scale (JESS), as well as snoring frequency, blood pressure, and body mass index (BMI) before and during pregnancy.

Results: Participants' average gestational age was 26.23 ± 7.56 weeks, and the J-PSQI and JESS scores were 5.5 ± 2.6 and 9.7 ± 4.2 , respectively. The average neck circumference was 33.4 ± 2.6 cm, and BMI before and during pregnancy was 21.0 ± 2.9 cm and 23.1 ± 3.1 cm, respectively. Among the participants, 42.5% displayed habitual snoring. These women had significantly higher BMI and weight before and during pregnancy than those who did not snore habitually. Participants with lower diastolic blood pressure and pre-pregnancy weight had significantly higher JESS scores. Participants with thick necks (neck circumference ≥ 33.4 cm) had significantly higher BMI and weight before and during pregnancy, as well as lower J-PSQI scores, than those with thin necks.

Conclusion: In pregnant Japanese women, neck circumference and BMI before and during pregnancy were lower than among pregnant women from other countries. However, Japanese women displayed a greater tendency toward snoring during pregnancy compared to women from other countries. Snoring was associated with obesity before and during pregnancy. However, the results suggest that thinness of physique prior to pregnancy is a risk factor for sleep disturbance during pregnancy. **Support:**

0850

SELF-REPORT AND POLYSOMNOGRAPHY SLEEP AND MORTALITY IN ADULTS: A MACHINE LEARNING REPLICATION ANALYSIS

Wallace, M. L.¹ Peppard, P.² Coleman, T. L.¹ Mentch, L.¹ Buysse, D. J.¹ Hall, M. H.¹ Redline, S.⁴ Hagen, E. H.² ¹University of Pittsburgh, Pittsburgh, PA, ²University of Wisconsin-Madison, Madison, WI, ³University of Pittsburgh, Pittsburgh, PA, ⁴Harvard Medical School, Boston, MA.

Introduction: Individual sleep health characteristics (e.g. efficiency, timing, duration, architecture) and signs and symptoms of sleep

disorders (e.g., difficulty falling and staying asleep, apnea hypopnea index, measures of oxygen desaturation) predict mortality in adults using traditional regression methods. However, it is important to examine and compare their predictive abilities in context of other established non-sleep predictors using high-dimensional methods that better reflect the complexity of the data. Therefore, we applied a novel random forest machine learning (RFML) hypothesis-testing framework to data from the Sleep Heart Health Study (SHHS) and the Wisconsin Sleep Cohort (WSC) to determine which risk factor domains (sleep, physical health, sociodemographic factors, medications, health behaviors, mental health) and sleep subdomains (self-report and polysomnography sleep health characteristics and signs and symptoms of sleep disorders) predict time to mortality in adults.

Methods: We harmonized 82 predictors across SHHS and WSC (32 sleep, 24 physical health, 8 sociodemographic, 9 medications, 4 mental health, 5 health behaviors) and fit sociodemographicadjusted and fully-adjusted RFML models in each cohort to test the overall predictive importance of each domain and sleep subdomain. Permutation-based p-values and unbiased variable importance metrics (change in Harrell's C *100, Δ C) were computed and summarized with medians across 20 independent subsampled testing sets in each cohort.

Results: In the fully-adjusted SHHS and WSC models, the most predictive domains were physical health (SHHS p<0.001, ΔC =1.48; WSC p=0.002, ΔC =2.68) and sleep (SHHS p=0.008, ΔC =0.71; WSC p=0.044, ΔC =1.65). Sleep subdomains were not significant in the fully adjusted model. However, the sociodemographic-adjusted models indicated that the predictive importance of sleep may be driven by polysomnography sleep health characteristics in SHHS (p=0.026, ΔC =0.77) and polysomnography signs of sleep apnea in WSC (p<0.001, ΔC =3.20).

Conclusion: Sleep is a strong predictor of mortality in adults that should be considered among other more routinely used predictors. Future research should examine differences and similarities between SHHS and WSC that may explain the finding that different aspects of sleep were important in each cohort.

Support: NIA grant AG056331, NHLBI grant HL114473, NHLBI grant R01HL62252, NIA grant R01AG036838, NIA grant R01AG058680.

0851

SLEEP DISTURBANCE BUT NOT SLEEP DURATION IS ASSOCIATED WITH PERCEIVED STRESS AND SELF-ESTEEM IN POSTPARTUM WOMEN

*Wang, X.*¹ *Kishma, E. E.*¹ *Sparks, J. R.*¹ *Liu, J.*¹ *Castleberry, L. A.*¹ *Cook, J. W.*¹ *Youngstedt, S. D.*²

¹University of South Carolina, Columbia, SC, ²Arizona State University, Phoenix, AZ.

Introduction: Healthy sleep is known to contribute to psychosocial well-being. Pregnancy and postpartum could have profound influences on women's psychosocial well-being related to physiological changes and interrupted sleep due to caring for the infant. The purpose of this study was to determine the associations between self-reported sleep characteristics and psychosocial well-being.

Methods: Forty-seven women who delivered a singleton infant after \ge 37 weeks of gestation were interviewed at 6-8 weeks, 4 months, and 6 months after delivery. The Pittsburgh Sleep Quality Index (PSQI) was used to obtain sleep duration, sleep disturbance, and the global PSQI score, and the Epworth Sleepiness Scale total score was used to assess daytime sleepiness. The Edinburgh

Postnatal Depression Scale, Perceived Stress Scale, and Rosenberg Self-Esteem Scale were used to assess psychosocial well-being, and a summary score of each instrument was calculated.

Results: The self-reported nightly sleep duration decreased over time (535 \pm 95, 505 \pm 78, 488 \pm 66 minutes, respectively at each timepoint, mean \pm SD, p = 0.007). Other sleep characteristics did not change. There were also no significant changes over time in the scores of depressive symptoms, stress, or self-esteem. At 6-8 weeks postpartum, sleep disturbance was associated with stress (r = 0.32, p = 0.026) and self-esteem (r = -0.38, p = 0.008) so that women who had greater sleep disturbances perceived greater stress and lower self-esteem. These associations did not exist 4 months or 6 months after delivery. Sleep duration, global PSQI score, and sleepiness were not associated with any of the psychosocial measures.

Conclusion: Sleep disturbance is an important correlate of psychosocial well-being in early postpartum. The decreased sleep duration likely indicates recovering from pregnancy and delivery, and adapting to routine lifestyle.

Support: National Institute On Minority Health And Health Disparities of the National Institutes of Health under Award Number R21MD012740

0852

GERIATRIC HEALTH CONDITIONS AND THE COMBINED OUTCOME OF POOR SLEEP QUALITY WITH OBJECTIVE SHORT SLEEP DURATION

Miner, B.¹ Vaz Fragoso, C. A.¹ Han, L.¹ Yaggi, H. K.¹ Redeker, N. S.² Stone, K. L.³

¹Yale University School of Medicine, New Haven, CT, ²Yale School of Nursing, Orange, CT, ³California Pacific Medical Center Research Institute, San Francisco, CA.

Introduction: Poor sleep quality with objective short sleep duration (≤ 6 hours) is a high-risk phenotype. The associations of geriatric health conditions with this sleep phenotype have not been described.

Methods: Using data on 3,127 older women from the Study of Osteoporotic Fractures (SOF), mean age 84 years, and 3,058 older men from the Osteoporotic Fractures in Men Sleep Study (MrOS), mean age 76 years, we evaluated cross-sectional associations between geriatric health conditions and the combined outcome of poor sleep quality with actigraphic short sleep duration. Geriatric health conditions included cognitive impairment (modified MMSE score 1.5SD below the cohort mean value), physical impairment (inability to do a chair stand), falls (≥ 2 in past year), and vision impairment (acuity ≤20/40). Poor sleep quality was defined by Pittsburgh Sleep Quality Index (PSQI) score >5 and short sleep duration by average total sleep time ≤ 6 hours from wrist actigraphy (averaged over ~5 days). Women (SOF) and men (MrOS) were evaluated separately and multivariate logistic regression models also included age, race, education, comorbidities (medical, psychiatric, and primary sleep disorders), and medication use.

Results: Poor sleep quality with actigraphic short sleep duration was present in 475 (15.6%) men and 400 (13.1%) women. In men, the unadjusted odds of having combined poor sleep quality with actigraphic short sleep duration were statistically higher with cognitive impairment (OR=1.45 [CI 1.05, 1.98]), physical impairment (2.90 [1.87, 4.51]), and falls (1.97 [1.48, 2.62]). In women, the unadjusted odds of having combined poor sleep quality with actigraphic short sleep duration were statistically higher with physical impairment (1.54 [1.16, 2.04]) and falls (1.63 [1.21, 2.20]).

However, these associations were no longer statistically significant in adjusted models (men and women).

Conclusion: Older persons with geriatric health conditions are more likely to have the combined phenotype of poor sleep quality with actigraphic short sleep duration, but this association is likely explained by comorbidity and medication use.

Support: Dr. Miner is supported by the Yale Claude D. Pepper Older Americans Independence Center (P30AG021342), the American Academy of Sleep Medicine Foundation, a foundation of the American Academy of Sleep Medicine, and the National Institute on Aging T32AG019134.

0853

FETAL HEART RATE VARIABILITY INCREASES DURING MATERNAL SLEEP APNEA EVENTS

Pien, G.¹ Bei, J.² Watson, H.¹ Sgambati, F.¹ Raghunathan, R.² Henderson, J.¹ DiPietro, J. M.²

¹Johns Hopkins University School of Medicine, Baltimore, MD, ²Johns Hopkins University School of Public Health, Baltimore, MD.

Introduction: While studies have established that SDB during pregnancy increases the risk of adverse maternal outcomes, fetal effects are less well studied. Evidence suggests that fetal heart rate decelerations, an indicator of fetal distress, may be elicited by SDB. We examined the relationship between maternal SDB events and fetal heart rate (FHR) and fetal heart rate variability (FHRV).

Methods: Obese (BMI≥30kg/m2) non-smoking women carrying singleton fetuses underwent overnight polysomnography (34-37 weeks gestational age), with simultaneous fetal heart rate monitoring. Standard methods were used to score sleep, SDB events (apneas/hypopneas) and to analyze fetal heart rate parameters. Using linear mixed effect models, we examined changes in mean FHR and FHR variability (expressed by FHR SD) between the 10-second period immediately before individual SDB events, during events to the end of the associated oxygen desaturation period, and the 10-second period immediately following the SDB event.

Results: Valid PSG and FHR data were obtained from 85 third trimester maternal-fetal dyads. Across all participants, there were 2779 maternal SDB events (apneas or hypopneas). Mean AHI for individual subjects was 9.04 (SD 13.75). 39 women had OSA (AHI \geq 5), which was mostly mild. Mean FHR did not change significantly during and after episodes of SDB episodes compared to pre-event FHR, and did not change afterwards compared to during events, in unadjusted or adjusted (sleep stage, apnea type, degree of desaturation, age) analyses. In unadjusted analyses, FHRV significantly increased during SDB episodes compared to pre-SDB FHRV. After SDB events, FHRV was significantly lower than during SDB events. In fully adjusted models, these findings remained highly significant. FHRV was not significantly different after SDB events compared to pre-SDB events models.

Conclusion: We observed consistent changes in FHR variability during and after maternal SDB events. Mean FHRV significantly increased during maternal SDB episodes compared to baseline FHRV, and decreased after SDB episodes. In contrast, mean FHR did not change significantly before, during and after SDB episodes. These data demonstrate that the fetus reacts to maternal SDB events, and raise questions about persistent effects of maternal SDB on the developing fetus. **Support:** NIH HD079411

0854

EXPLORATION OF SLEEP HEALTH AMONG MIDDLE-AGED AND OLDER ADULTS

Lorenz, R. A. Auerbach, S. L. Li, C. Chang, Y. University at Buffalo School of Nursing, Buffalo, NY.

Introduction: Sleep health, a construct introduced to characterize the multidimensional attributes of sleep, has been explored in a variety of populations; however has not been adequately examined for middle-aged and older adults. As attributes of sleep may change with age, the dimensional structure of sleep health may differ in this population. This study aimed to validate a composite measure of sleep health among middle-aged and older adults using data from the Health and Retirement Survey (HRS).

Methods: Data from the 2014 Core survey of the HRS was used to create a composite measure of Sleep Health including sleep efficiency, duration, timing, satisfaction, and alertness. We standardized and averaged the original variables before transforming to T scores. Sleep Health T scores (ranging 0-100, higher scores indicating better sleep health) were examined using exploratory and confirmatory factor analysis (EFA; CFA).

Results: Our sample included 6,095 adults with mean age of 68 years (SD=10.1; range 50-99 years). The majority were female (59.7%), white (77%), with high school education (53.9%). Sleep Health T scores ranged from 27-61 (mean=50; SD=6.7). EFA identified one factor. Timing was removed due to low factor loading (<0.4). The revised four-dimension composite Sleep Health measure had acceptable reliability (Cronbach's alpha 0.6). CFA showed a well-adjusted model (REMSA=0.097; NFI=0.964; RMR=0.035; GFI=0.990; AGFI=0.951).

Conclusion: These results suggest that the composite measure was valid for assessing sleep health among middle-aged and older adults. Limitations include the use of secondary data, as sleep health dimensions were based on variables not created specifically for our research question. Future research should further examine the role of sleep timing in overall sleep health among middle-aged and older adults.

Support: This study was supported by the University at Buffalo Clinical and Translational Science Institute (CTSI) funded by the National Institutes of Health (Lorenz, PI).

0855

PRIMARY SLEEP DISORDERS AND THE COMBINED OUTCOME OF POOR SLEEP QUALITY WITH OBJECTIVE SHORT SLEEP DURATION IN OLDER PERSONS

Miner, B.¹ Vaz Fragoso, C. A.¹ Han, L.¹ Stone, K. L.² Redeker, N. S.³ Yaggi, H. K.¹

¹Yale University School of Medicine, New Haven, CT, ²California

Pacific Medical Center Research Institute, San Francisco, CA, ³Yale School of Nursing, Orange, CT.

Introduction: Poor sleep quality with short sleep duration (SSD) is a high-risk phenotype that is likely to be associated with primary sleep disorders (obstructive sleep apnea [OSA], periodic limb movements of sleep [PLMS], and restless legs syndrome [RLS]) in older persons. We evaluated the associations among primary sleep disorders and this high-risk phenotype in older persons.

Methods: Using data on 3,058 men from the Osteoporotic Fractures in Men Sleep Study and 3,127 women from the Study of Osteoporotic Fractures, mean ages 76 and 84 years, respectively, we evaluated cross-sectional associations between primary sleep disorders and the combined outcome of poor sleep quality

with actigraphic SSD. In women, OSA and RLS were evaluated by self-report. In men, OSA and PLMS were evaluated by polysomnography and RLS by self-report. Poor sleep quality was defined by Pittsburgh Sleep Quality Index score >5 and SSD by average total sleep time ≤ 6 hours from wrist actigraphy (averaged over ~ 5 days). Men and women were evaluated separately. Multivariate logistic regression models also included demographics, self-reported chronic conditions, anxiety, depression, and medication use.

Results: Poor sleep quality with actigraphic SSD was more prevalent in men (475 [15.6%]) than women (400 [13.1%]). In unadjusted models in men, odds of poor sleep quality with actigraphic SSD were significantly higher with OSA, PLMS, and RLS (ORs [95% Cis] = 1.99 [1.57, 2.52], 2.11 [1.41, 3.18], and 5.58 [2.51, 12.43], respectively). In multivariable models in men, odds of poor sleep quality with actigraphic SSD were significantly higher with OSA (1.59 [1.18, 2.14]) but not with PLMS or RLS. In unadjusted models in women, odds of poor sleep quality with actigraphic SSD were significantly higher SSD were significantly higher with OSA (1.59 [1.18, 2.14]) but not with PLMS or RLS. In unadjusted models in women, odds of poor sleep quality with actigraphic SSD were significantly higher with OSA (3.57 [0.40, 31.88]) and RLS (5.60 [3.04, 10.32]), but results were not significant in multivariable models in women.

Conclusion: Older persons with primary sleep disorders have higher odds of poor sleep quality with actigraphic SSD. However, the predominant mechanisms underlying this high-risk phenotype may be driven more by medical and psychiatric comorbidity than by primary sleep disorders.

Support: The American Academy of Sleep Medicine Foundation and the Yale Claude D. Pepper Older Americans Independence Center

0856

RELATIONSHIP BETWEEN A COMPOSITE MEASURE OF SLEEP HEALTH AND BONE MINERAL DENSITY IN A SAMPLE OF OLDER WOMEN FROM THE STUDY OF OSTEOPOROTIC FRACTURES

Kubala, A. G.¹ Sullivan, K. J.² Kline, C. E.¹ Cauley, J. A.³ ¹Department of Health and Physical Activity, Pittsburgh, PA, ²Department of Medicine, Jackson, MS, ³Department of Epidemiology, Pittsburgh, PA.

Introduction: Observational studies suggest poor sleep is related to lower bone mineral density (BMD) and increased osteoporosis risk. Yet, many studies focus on sleep duration and lack inclusion of other sleep characteristics. The sleep health construct simultaneously recognizes multiple dimensions of sleep and is operationalized as a composite score. Thus, we examined whether a composite measure of sleep health was related to BMD in a sample of older women.

Methods: The sample included 1968 older women (mean age: 83.6 \pm 3.1 years) from the Study of Osteoporotic Fractures. Six sleep health domains (regularity, duration, satisfaction, timing, efficiency, sleepiness/alertness) were dichotomized into either "good" or "poor" categories. The number of "good" characteristics were summed into a score ranging from 0 (poor) to 6 (good). BMD (g/cm²) was measured at the femoral neck, total hip, and trochanter sites with dual energy x-ray absorptiometry. Multiple linear regression was used to explore the association between sleep health (composite score and the individual domains) with BMD (cross-sectional) and annualized percent change in BMD (longitudinal). All models were adjusted for age, body mass index, alcohol consumption, smoking, physical activity, education, diabetes, hyperthyroidism, fracture history, and cardiovascular disease.

Results: Average sleep health score was 3.8 ± 1.2 . Cross-sectionally, better sleep health was associated with higher BMD at the femoral neck (β =.04, p=.04) and trochanter (β =.05, p=.02). Sleep health was not cross-sectionally associated with BMD at the total hip (β =.03, p=.09) or with change in BMD at any region (Each p >.13). The individual domain of sleep regularity was cross-sectionally related to BMD at the total hip and trochanter, respectively (β =.04, P=.04; β =.05, P=.02).

Conclusion: A multi-dimensional measure of sleep health was related to greater BMD cross-sectionally at the femoral neck and trochanter regions in a sample of older women. Future studies should focus on associations between sleep health and osteoporoticrelated fractures.

Support: The Study of Osteoporotic Fractures (SOF) is supported by National Institutes of Health funding. The National Institute on Aging (NIA) provides support under the following grant numbers: R01 AG005407, R01 AR35582, R01 AR35583, R01 AR35584, R01 AG005394, R01 AG027574, and R01 AG027576

0857

FACTORS ASSOCIATED WITH SLEEP HEALTH AMONG MIDDLE-AGED AND OLDER ADULTS

Lorenz, R. A. Auerbach, S. L. Li, C. Chang, Y. University at Buffalo School of Nursing, Buffalo, NY.

Introduction: The concept of Sleep Health (SH) was developed to provide a multidimensional framework consisting of characteristics of sleep that have been closely associated with physical and mental well-being. Identification of key factors associated with SH can identify targets for intervention for its improvement. This study aimed to identify factors related to SH among middle-aged and older adults.

Methods: A valid Sleep Health Composite Measure was recently developed from the data of the 2014 Core Survey of the Health and Retirement Survey and was used to assess overall SH in middle-aged and older adults. Spearman's correlations, T-test, and Kruskal-Wallis tests were used to explore the association of SH with age, gender, self-rated health, chronic disease burden, disability, pain, and modifiable lifestyle behaviors (physical activity, smoking, alcohol, sleep medication).

Results: Our sample included 6,095 adults with mean age of 68 years (SD=10.1; range 50-99 years). The majority were female (59.7%) and white (77%). Correlations showed significant moderate associations between SH and self-rated health (r=.404), sleep medications (r=.390), pain (r=-.315). There were significant but small associations between vigorous activity (r=-.193), alcohol (r=.112), currently smoking (r=-.089) and SH. SH was significantly worse among women compared to men (t(6093)=6.996, p<0.001). Furthermore, SH scores were significantly different between middle-aged versus older adults (p<0.001); varying levels of self-rated health (p<0.001), number of chronic diseases (p<0.001), and degree of physical disability (p<0.001).

Conclusion: These findings provide evidence of factors related to SH among middle-aged and older adults, providing targets for intervention to improve health in this population. Age-related differences in relation to SH indicate intervention tailored for middle-aged adults may improve health outcomes as they age. Future studies are warranted to extend these findings using multivariable statistical approaches.

Support: This study was supported by the University at Buffalo Clinical and Translational Science Institute (CTSI) funded by the National Institutes of Health (Lorenz PI).

0858

IMPACT OF ACTIGRAPHIC SLEEP MEASURES ON AMBULATORY COGNITIVE PERFORMANCE IN A COMMUNITY-BASED SAMPLE OF OLDER ADULTS

Buxton, O. M.^{1,2,3} Zhaoyang, R.^{4,5} Jiao, J. L.¹ Sliwinski, M. J.^{4,5} Derby, C. A.⁶

¹Department of Biobehavioral Health, College of Health and Human Development, The Pennsylvania State University, University Park, PA, ²Division of Sleep Medicine, Harvard Medical School, Boston, MA, ³Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, Boston, MA, ⁴Center for Healthy Aging, Pennsylvania State University, University Park, PA, ⁵Department of Human Development and Family Studies, College of Health and Human Development, The Pennsylvania State University, University Park, PA, ⁶Departments of Neurology and Epidemiology & Population Health, Albert Einstein College of Medicine, Bronx, NY.

Introduction: Few longitudinal studies link objectively assessed sleep and cognition, especially day to day differences in sleep as they relate to daily cognitive performance in ecologically-valid, natural environments. We examine the associations of sleep (actigraphy) with ambulatory ecological momentary assessments (EMA) of cognitive performance.

Methods: Analyses involved 225 participants enrolled in The Einstein Aging Study, a community based longitudinal cohort of older adults free of dementia at enrollment (Mage=77.27 years; 33% males; 47% Caucasian, 39% African American, 13% Hispanic). We examined between-person associations between actigraphic sleep duration and wake after sleep onset (WASO) with mean and variability across the day in cognitive performance. Cognitive performance was assessed via validated, smartphone-based EMA over a mean of 18 days. Six assessments/day included Symbol Match (processing speed), Color Dot (working memory), and Color Shape (memory binding). Models controlled for age, gender, ethnicity, education (years), clinically assessed mild cognitive impairment, and learning effects. Actigraphy data was collected throughout the study period.

Results: Sleep duration had a significant effect on within-person variability on ambulatory cognition: Color Dot, Symbol Match, and Color Shape (all p's <0.001). Older adults with longer mean nightly sleep duration exhibited more stable cognitive performance over time versus those with shorter sleep duration; sleep duration did not predict mean levels of daily cognitive performance. Person-level means of WASO (0.99±0.45 hours/night) predicted mean levels on cognitive tests, independent of sleep duration. Older adults with less WASO/night exhibited better cognitive performance. One half hour less nightly WASO predicted 175ms shorter Symbol Match response time (p=0.004), 1.5% lower Color Dot error proportion (p=0.048), 0.07 points higher Color Shape accuracy (p<0.001). Older adults with less WASO/night also had less within-person variations in Color Dot (p<0.001), Symbol Match (p<0.001) and Color Shape performance (p=0.01).

Conclusion: Ambulatory cognitive performance assessed using EMA was related to actigraphic sleep. Poor sleep health may be a target for prevention of early cognitive changes that may precede onset of cognitive impairment and AD.

Support: Research was supported by the National Institute on Aging (NIA) award numbers P01AG003949 and R01AG056538.

0859

TYPES OF CHILDHOOD MALTREATMENT AND SLEEP REGULATION DURING PREGNANCY

Touchette, E.¹ Servot, S.² Lemieux, R.³ Berthelot, N.⁴ ¹Department of Psychoeducation, Université du Québec à Trois-Rivières, Quebec, QC, CANADA, ²Department of Psychoeducation, Université du Québec à Trois-Rivières, QC, CANADA, ³Department of Nursing, Université du Québec à Trois-Rivières, QC, CANADA, ⁴Department of Nursing, Université du Québec à Trois-Rivières, QC, CANADA.

Introduction: Pregnant women with history of childhood maltreatment would have around 2-fold increased odds of poor subjective sleep in comparison to pregnant women without history of trauma (Gelaye et al., 2015). Our aim was to evaluate whether different types of childhood maltreatment were associated with poorer subjective and objective sleep regulation during the second trimester of pregnancy.

Methods: Sleep regulation between 18-20 weeks of gestation was assessed in a sample of 55 expectant mothers, including 31 women exposed to childhood maltreatment. Three measures of sleep were administered: 7-day actigraph measures (Mini-Mitter/Respironics), 7-day sleep diary and the completion of Pittsburgh Sleep Quality Index. Childhood maltreatment was assessed using the Chilhood Trauma Questionnaire. Generalized linear regression models were used to examine the associations between sleep measures and types of childhood maltreatment after adjusting for confounding variables (e.g., maternal age, maternal wellbeing, education attainment and family income).

Results: Among the 31 participants with history of childhood maltreatment, 71% (n=22) reported emotional abuse, 26% (n=8) physical abuse, 39% (n=12) sexual abuse, 42% (n=13) emotional neglect and 65% (n=20) physical neglect. Pregnant women with childhood emotional abuse had around 2.8 higher score on PSQI in comparison to pregnant women without childhood emotional abuse (P<0.003). For objective sleep measures, pregnant women with childhood sexual abuse had around 1 hour less of nocturnal sleep (P<0.004), 30 minutes more nocturnal awakenings (P<0.03) and 6% less of sleep efficiency (P<0.01) compared with pregnant women without childhood sexual abuse.

Conclusion: Emotional abuse during childhood was associated with poorer perceived sleep quality during the 2nd trimester of pregnancy while childhood sexual abuse was particularly associated with objective measures of sleep regulation. Future larger studies are needed to confirm the impact of the different types of childhood maltreatment on maternal sleep quality during pregnancy.

Support: Social Sciences and Humanities Research Council (SSHRC, 2018-2020, Canada)

0860

SLEEP DISORDERS IN FEMALE MILITARY PERSONNEL

*Villarreal, B.*¹ *Foster, S.*¹ *Hansen, S.*¹ *Brock, M.*¹ *Sanchez, H.*¹ *Gerwell, K.*³ *Carrizales, F.*³ *Peterson, A.*³ *Pruiksma, K.*³ *Mysliwiec, V.*¹

¹San Antonio Military Healthcare System, Lackland AFB, TX, ²San Antonio Military Healthcare System, Lackland AFB, TX, ³University of Texas Health Science Center at San Antonio, San Antonio, TX, ⁴University of Texas Health Science Center at San Antonio, San Antonio, TX. **Introduction:** Sleep is an essential biological function and the disruption of sleep has deleterious consequences. Military personnel experience unique stressors related to their service, elevating the risk of developing sleep disorders. The etiologies and impact of sleep disorders on military women's health is poorly understood. This study is the first to prospectively assess whether military women with insomnia, obstructive sleep apnea (OSA), or comorbid insomnia and OSA (COMISA) have different gender roles, military service-associated factors, and biological characteristics than military men with the same disorders.

Methods: This is a prospective observational study of military personnel with sleep disturbances. The study will evaluate women and men matched for sleep disorder. Participants will complete an evaluation to include polysomnography (PSG), sleep questionnaires and validated clinical assessments of associated disorders of interest. The baseline demographics, questionnaire, and PSG results will be analyzed to assess for commonalities or differences between genders.

Results: We have enrolled 45 patients (24% female). Males had a higher BMI (29.1) than females (26). Males were also older (38) than females (35). The leading diagnosis in males was OSA (44%) and insomnia in females (64%). In males, the apnea-hypopnea index (AHI) was 11.3/hr, arousal index (ARI) was 20/hr, and sleep efficiency (SE) was 86.5%. Total sleep time (TST), wake after sleep onset (WASO), and sleep onset latency (SOL) were 364.6, 40.9, and 12.6 minutes, respectively. In females, the average AHI was 6.6/hr, ARI was 15/hr, and SE was 87.2%. Their TST, WASO, and SOL were 359, 44, and 12.6 minutes, respectively.

Conclusion: Military personnel are at increased risk of sleep disorders. Literature comparing male and female characteristics and sleep disorders is scarce. In this study, baseline demographics were similar in both groups but insomnia was the leading diagnosis for women. This emphasizes the importance of adequate recognition and treatment of insomnia in this group.

Support: This study is supported by the Defense Health Agency, Defense Medical Research and Development Program, Clinical Research Intramural Initiative for Military Women's Health.

0861

AGE, RACE, AND CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP) CONFIDENCE SCORE AT 1-WEEK PREDICT 3-MONTH CPAP ADHERENCE IN OLDER ADULTS WITH AMNESTIC MILD COGNITIVE IMPAIRMENT AND MODERATE TO SEVERE OBSTRUCTIVE SLEEP APNEA

Richards, K. C.¹ Vallabhaneni, V.^{2,3} Moelter, S.⁴ Davis, E. M.⁵ Morrison, J.¹ Lozano, A.⁶ Hanlon, A.⁶ Wang, Y.¹ Wolk, D.⁷ Gooneratne, N.⁷

¹University of Texas at Austin, Austin, TX, ²Sleep 360 Diagnostic Center, Austin, TX, ³Texas A&M University, College Station, TX, ⁴University of the Sciences, Philadelphia, PA, ⁵University of Virginia, Charlottesville, VA, ⁶Virginia Tech, Blacksburg, VA, ⁷University of Pennsylvania, Philadelphia, PA.

Introduction: Adherence to continuous positive airway pressure (CPAP) may delay cognitive decline in older adults with obstructive sleep apnea (OSA) and amnestic mild cognitive impairment (MCI), defined as deficits in memory that do not significantly impact daily functioning. The aim of this analysis was to identify predictors of CPAP adherence in this population.

Methods: Data are from Memories 2, an ongoing multisite clinical trial on the effect of treatment of moderate to severe OSA on cognitive decline in older adults 65-85 years of age who have amnestic MCI. Unadjusted and adjusted linear models were used to examine predictors of mean hours of CPAP use at 3 months. Predictors were age, sex (male/female), race (White/Non-White), education (more than high school, less than high school), Apneahypopnea index (AHI), Epworth Sleepiness Scale (ESS), and CPAP Comfort and Confidence scores at 7 days. Collinearity in the adjusted model for CPAP use at 3 months was examined using the variance inflation factor.

Results: Of 57 participants, most were male (54%), White (72%), with a mean age of 66.3 years (SD: 6.1). Mean AHI in this sample was 35.1 (SD: 19.9), with mean daily hours of CPAP use at 3 months 5.3 hours (SD: 2.3). Adjusted linear model results demonstrated that younger age (β =-0.13, SE=0.04, p=0.0032), White race (β =2.56, SE=0.58, p<0.0001), and higher 7-day CPAP Confidence score (β =0.48, SE=0.17, p=0.0086) were significantly associated with CPAP use at 3 months. Sex, education, AHI, ESS, and CPAP comfort were not statistically significant predictors of adherence.

Conclusion: Tailored interventions to increase self-efficacy during the first 7 days of CPAP treatment, especially in Non-Whites and those older than 74 years, may improve long-term CPAP adherence in older adults with amnestic MCI. **Support:** R01AG054435

0862

ADEQUATE SLEEP DURATION ENHANCES CARDIOVASCULAR BENEFITS OF A PHYSICAL ACTIVITY INTERVENTION IN OLDER AFRICAN AMERICANS

Hoddy, K. K. Singh, P. Beyl, R. A. Kirwan, J. P. Carmichael, O. T. Newton, R. L.

Pennington Biomedical Research Center, Baton Rouge, LA.

Introduction: African Americans are at a greater risk for cardiovascular disease and inadequate sleep than are corresponding whites. Age-associated declines in sleep duration, cardiovascular health, and physical activity highlight the need to understand the relationship among these variables in this population. While physical activity is thought to be beneficial for promoting sleep quality, it remains unknown how habitual short sleep during a physical activity intervention influences the intervention response in this population.

Methods: Sedentary older African Americans (n=27; 65-85 years old; 74% female) participating in the intervention arm of a 12-week randomized controlled physical activity trial (NCT03474302) were categorized as short (n= 15) or adequate (n=12) sleepers, defined as sleeping <6 hours/night or >6 hours/night on average during the intervention. Participants wore validated activity monitors at baseline and 12 weeks, and commercially available sleep monitors were worn daily. Differences in cardiovascular outcomes at baseline and 12 weeks were assessed between sleep categories using sex-adjusted linear mixed models.

Results: The intervention increased accelerometer derived steps (p=0.04) with no between group differences (p=0.78). Moderate to vigorous activity (MVA) duration increased (p<0.001), but change was greater in adequate sleepers (9 minutes; p<0.05). Body weight did not significantly change (-0.71 kg; p=0.11) and changes were similar between groups (p=0.55). Total (p=0.046) and LDL cholesterol (p=0.04) decreased over time. Adequate sleepers experienced improvements in systolic blood pressure (-10 \pm 4.5 mmHg; p=0.03), total cholesterol (-30 \pm 11 mg/dL; p=0.01), and LDL cholesterol

 $(-23 \pm 9 \text{ mg/dL}; p=0.01)$ from baseline while short sleepers did not (all p>0.05). Significant differences or trends between adequate and poor sleepers were observed (-10.5 mmHg; p=0.096; -30 mg/ dL p=0.044; -21 mg/dL; p=0.095, respectively).

Conclusion: Adequate sleep during a physical activity intervention may be important to elicit cardiovascular benefits. Thus, research evaluating sleep extension complementary to increased physical activity is warranted in short sleepers.

Support: BrightFocus (A20175472); National Institute of General Medical Sciences of the National Institutes of Health (U54-GM104940)

0863

AGE-CATEGORIZED TRENDS IN SELF-REPORTED SLEEP DURATION FOR THE NON-INSTITUTIONALIZED U.S. CIVILIAN POPULATION FROM 2004-2013: CONSIDERATIONS OF RACIAL/ETHNIC VARIATIONS

Christina, M.¹ Bubu, O. M.¹ Donley, T.¹ Blanc, J.¹ Oji, E.¹

Turner, A. D.¹ Mbah, A. K.² Williams, N. J.¹ Youngstedt, S.³
Shochat, T.⁴ Azizi, S. A.¹ Osorio, R. S.¹ Jean-Louis, G.¹
¹NYU School of Medicine, New York, NY, ²University of South Florida, Tampa, FL, ³Arizona State University, Phoenix, AZ,

⁴University of Haifa, Haifa, ISRAEL.

Introduction: We examined age-categorized trends in self-reported sleep duration using data from the National Health Interview Survey (NHIS) 2004-2013 and explored how these trends may vary based on individuals' race/ethnicity.

Methods: Study participants were aged 18-85 (N=258,158). Sleep duration within a 24-hour period on average was categorized as \leq 6hrs (short-sleep), 7-8 hours (adequate-sleep), and \geq 9hrs (long-sleep). Age was categorized as 18 - <26, 26 - <65 and 65 - 85. Racial categories included non-Hispanic Whites (NHW), Blacks/African Americans (AAs) and Hispanics. Adjusted multinomial logistic regression models examined trends in self-reported sleep duration across age-categories and assessed race/ethnic differences in these trends.

Results: Mean sleep duration (hrs.) across all years was 7.4, 7.0, and 7.5, for ages 18 - <26, 26 - <65 and 65 - 85, respectively and was relatively stable from 2004-2013. However, compared to individuals ages 18 - <26, those categorized as ages 26 - <65 were 55% more likely to be short sleepers while those ages 65 - 85 were 20% less likely to be short sleepers (P < .001 for all). Mean sleep duration was 7.2hrs, for NHW and 7.1hrs for AAs and Hispanics, and showed increasing trend toward short sleep beginning in 2007 through 2013 (P < .01 for trend). In the age 18 - <26 category, compared to whites, blacks and Hispanics were 35% and 29% more likely to be short sleepers, respectively. In the age 65 - 85 category, compared to whites, blacks and Hispanics were 35% and 21% more likely to be short sleepers, respectively. In the age 65 - 85 category, compared to whites, blacks were 19% more likely to be short sleepers (P < .001 for all).

Conclusion: Continued surveillance of population-level sleep trends among minority populations is essential as growing race/ ethnic (age specific) disparities in self-reported sleep duration may have consequences for racial/ethnic health disparities.

Support: NIH/NIA/NHLBI (L30-AG064670, CIRAD P30AG059303Pilot,T32HL129953,R01AG056531,R25HL105444, R25NS094093, K07AG05268503, R01HL142066, K23HL125939)

0864

RACE/ETHNICITY AND SEX-DEPENDENT EFFECTS OF METABOLIC BURDEN ACROSS DIFFERENT AGE-CATEGORIES ON TRENDS IN SELF-REPORTED SLEEP DURATION: FINDINGS FROM THE NATIONAL HEALTH INTERVIEW SURVEY, 2004-2013

Eirene, O.¹ Bubu, O. M.¹ Donley, T.¹ Blanc, J.¹ Madera, C.¹ Turner, A.¹ Mbah, A. K.² Williams, N. J.¹ Youngstedt, S.³ Shochat, T.⁴ Seixas, A. A.¹ Osorio, R. S.¹ Jean-Louis, G.¹ ¹NYU School of Medicine, New York, NY, ²University of South Florida, Tampa, FL, ³Arizona State University, Phoenix, AZ, ⁴University of Haifa, Haifa, ISRAEL.

Introduction: We examined race and sex-dependent effects of metabolic burden across different age-categories on trends in self-reported sleep duration for the U.S. non-institutionalized civilian population.

Methods: We analyzed data from the National Health Interview Survey (NHIS) adults aged 18-85 from 2004 to 2013 (N=258,158). Metabolic burden was characterized by obesity (BMI>30), dyslipidemia, diabetes, and hypertension morbidity burden levels. Racial/ethnic categories included non-Hispanic Whites (NHW), Blacks/African Americans (AAs) and Hispanics. Sleep duration within a 24-hour period on average was categorized as short sleep (\leq 6hrs), adequate sleep (7-8 hrs.), and long sleep (\geq 9hrs). Age was categorized as 18 - <26, 26 - <65 and 65 - 85. Adjusted multinomial logistic regression models stratified by race, sex and age-categories examined effects of metabolic burden on trends in self-reported sleep duration.

Results: The prevalence of short sleep duration was relatively stable from 2004-2012 for NHW and all females. However, AA and Hispanic males showed consistent increase in the rates of short sleepers beginning in 2007 through 2013 especially for ages 18 - <26, and 26 - <65 (*P* <.001 for trend). For all racial/ethnic categories, compared to individuals aged 18 - <26, individuals aged 26 - <65 were more likely to report short sleep (aOR: 1.55, 95% CI: 1.50-1.61) and individuals aged 65 - 85 were more likely to be long sleepers (aOR: 1.95, 95% CI: 1.86-2.05). Interestingly, the rate of short sleep increased as the metabolic burden increased (*P* <.001 for trend). This trend was more pronounced among AA and Hispanic males aged 65 - 85 with ≥ 2 metabolic conditions who were more likely to report short sleep (aOR: 1.77, 95% CI: 1.44-2.19 and aOR: 1.45, 95% CI: 1.17-1.93 respectively), compared to NHW males.

Conclusion: Increased metabolic burden among minority populations and especially in the elderly male, affect sleep and may have consequences for treating these populations.

Support: NIH/NIA/NHLBI (L30-AG064670, CIRAD P30AG059303 Pilot, T32HL129953, R01AG056531, R25HL105444, R25NS094093, K07AG05268503, R01HL142066, K23HL125939)

0865

MENSTRUAL REGULARITY AND BLEEDING ASSOCIATED WITH SLEEP DURATION, SLEEP QUALITY, AND DAYTIME SLEEPINESS IN A COMMUNITY SAMPLE

Onyeonwu, C.¹ Nowakowski, S.² Hale, L.³ Branas, C.⁴ Barrett, M.⁵ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹

¹University of Arizona, Tucson, AZ, ²Baylor College of Medicine, Houston, TX, ³Stony Brook University, Stony Brook, NY, ⁴Columbia University, New York, NY, ⁵University of Pennsylvania, Philadelphia, PA.

Introduction: Female menstrual health and its relationship to sleep duration, quality, fatigue and other factors is an understudied subject in the field of sleep and women's health. Few studies examined sleep in relation to menstrual regularity and bleeding.

Methods: Data were obtained from N=579 women who have had a menstrual period in the past 12 months who participated in the Sleep and Health Activity, Diet, Environment, and Socialization (SHADES) study, a community-based sample of adults age 22-60 living in southeastern Pennsylvania. Participants were asked, "How regular is your period?" with response choices of "Very Regular," "Mostly Regular," "Fairly Regular," and "Not Regular." They were also asked, "How much bleeding do you usually experience during your period?" Responses were "Very Heavy," "Medium," "Light," or "Very Light." These were evaluated as ordinal outcomes. Sleep-related predictors included sleep duration (<=6h [Short], 7-8 [Normal], and >=9 [Long]), Insomnia Severity Index (ISI) score, Pittsburgh Sleep Quality Index (PSQI) score, Epworth Sleepiness Scale (ESS) score, and Fatigue Severity Scale (FSS) score. Covariates included age, education, income, race/ethnicity, and body mass index.

Results: Short sleep duration was associated with a greater likelihood of heavier bleeding (OR=1.46, p=0.026) and greater irregularity (OR=1.44, p=0.031), compared to Normal sleep. Higher PSQI score was associated with more irregularity (OR=1.05, p=0.022). FSS score was associated with both heavier bleeding (OR=1.02, p=0.003) and more irregularity (OR=1.02, p=0.008). Long sleep, ISI, and ESS were not associated with either outcome. A sleep duration by FSS score interaction was found for irregularity (p=0.1). Among Normal sleepers, FSS was associated with greater irregularity, but not among Short sleepers.

Conclusion: There is a relationship between short sleep and heavier and irregular menses. These findings have implications for treating sleep problems among women. Also, mechanisms of these associations should be explored further.

Support: Dr. Grandner is supported by R01MD011600 The SHADES study was funded by R21ES022931

0866

A COMPARISON OF RESTING CEREBRAL BLOOD FLOW AND SLEEP QUALITY IN OLDER ADULTS

Hays, C. C.¹ Almklov, E. A.² Orff, H. J.^{1,3} Wierenga, C. E.^{3,2} ¹VA San Diego Center of Excellence for Stress and Mental Health (CESAMH), San Diego, CA, ²VA San Diego Healthcare System, San Diego, CA, ³UCSD Department of Psychiatry, San Diego, CA.

Introduction: Sleep disturbances have been linked to a variety of health-related consequences, including clinically significant cognitive alterations. Older adults represent a particularly vulnerable population given that advanced age is associated with an increased risk for both sleep disorders, such as insomnia, and cognitive decline. Examining the relationship between resting cerebral blood flow (rCBF) and sleep quality in older adults will better our understanding of the neurophysiologic implications of poor sleep in aging adults.

Methods: Thirty-three cognitively normal older adults (15 males) between the ages of 65-85 (mean age=73) were administered the

Pittsburg Sleep Quality Index (PSQI) and underwent assessment of rCBF using arterial spin labeling (ASL). Those who scored above 5 on the PSQI were defined as poor sleepers (n=17) and those who scored 5 or below were defined as good sleepers (n=16). Groups were then compared on voxel-wise whole-brain rCBF using independent samples t-tests statistically adjusting for age, sex, and the time interval between neuroimaging and sleep assessment.

Results: Compared to good sleepers, poor sleepers exhibited higher rCBF within bilateral thalamus and the left precuneus and lower rCBF within the left putamen (all *ps*<.01, uncorrected).

Conclusion: In this preliminary investigation, poor sleepers exhibited a differential pattern of rCBF in several brain regions, including those involved in consciousness and other important cognitive abilities such as attention. Future research is needed to determine the short- and long-term implications of poor sleep on the aging brain.

Support: U.S. Department of Veterans Affairs Clinical Sciences Research and Development Service Merit Award 5I01CX000565 (CEW) & VA Rehabilitation Research & Development - Career Development Award - RX001512-01A2 (HJO)

0867

GENDER DIFFERENCES IN THE RELATIONSHIP BETWEEN EXERCISE AND SLEEP IN YOUNG ADULTS *Glavin, E. Spaeth, A. M.*

Rutgers University, New Brunswick, NJ.

Introduction: Exercise is widely prescribed as a behavioral treatment for sleep health, but generalizations about the benefits of exercise for sleep do not typically consider gender differences. The present study examined relationships between exercise frequency and self-reported sleep outcomes in undergraduate men and women.

Methods: Students were recruited from campus fitness facilities immediately after they finished a workout (N=829, 19.5 \pm 1.4 y, 38.5% Female) and completed demographic, Pittsburgh Sleep Quality Index (PSQI) and exercise questionnaires. Department of Health and Human Services Physical Activity Guidelines for Americans were used to dichotomize students into those who met recommendations for weekly aerobic and strength training exercise and those who did not. Multiple regression was used to examine the relationships between exercise frequency and PSQI outcomes and analysis of variance was used to compare PSQI outcomes by gender and exercise guideline groups.

Results: Compared to women, men reported more days of exercise, fewer days of aerobic exercise, more days of strength training, better sleep efficiency and later bedtimes (ps<0.05). Among all participants, exercise frequency associated with an earlier bedtime, higher sleep efficiency, less daytime dysfunction and better sleep quality (ps<0.05). When divided by gender, exercise frequency associated with an earlier bedtime and better sleep quality in women (ps<0.05) but not men and associated with less daytime dysfunction in men (p=0.012) but not women. Approximately 47% of participants met exercise guidelines for both aerobic and strength training. Participants who met the guidelines exhibited an earlier bedtime (p=0.01) and higher sleep efficiency (p=0.08) than those who did not. There was also a significant interaction effect for sleep latency (p=0.042) such that meeting the guidelines associated with a shorter sleep latency in men (p=0.006) but not women.

Conclusion: These findings highlight the importance of taking an individual's gender and specific sleep issue into account when implementing an exercise prescription to improve sleep. **Support:** Rutgers University Aresty Foundation

SLEEP, Volume 43, Abstract Supplement, 2020

0868

HOW DO SLEEP MORBIDITIES DIFFER AMONGST PREGNANT WOMEN, WOMEN WHO ARE INTENDING TO CONCEIVE, AND WOMEN WHO ARE NOT INTENDING TO CONCEIVE?

Hartman, A. R.¹ Geller, P. A.¹ Morales, K.² Lee, K.³ Kloss, J.⁴ Perlis, M. L.⁴

¹Drexel University, Department of Psychology, Philadelphia, PA, ²University of Pennsylvania, Department of Biostatistics, Epidemiology, and Informatics, Philadelphia, PA, ³University of California, San Francisco, Department of Family Health Care Nursing, San Francisco, CA, ⁴Behavioral Sleep Medicine Program, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA.

Introduction: Maternal sleep disturbance is common during pregnancy and is associated with adverse maternal and child outcomes, such as postpartum depression and preterm birth. The extent to which sleep disorder symptoms are normative among women of reproductive age, however, is largely unknown. The present study's primary aim was to explore cumulative sleep morbidity and the incidence of disorder-specific symptoms among reproductive-aged women of different childbearing statuses.

Methods: Sleep morbidity variables were examined crosssectionally among three groups of reproductive-aged nulliparous women: those 1) currently pregnant (n=148), 2) currently intending to conceive (n=233), and 3) not currently intending to conceive (n=379). All subjects self-reported sleep disorder symptoms at baseline using the Sleep Disorders Symptom Checklist-25 (SDS-CL-25). This instrument measures symptoms related to 13 sleep disorders scaled 0 (never) to 4 (> 5 days per week). Average scores were calculated for each item, each of 13 sleep disorders, and for the whole instrument (0-100).

Results: Initial results indicated that pregnant women (M=22.80, SD=11.49) had a higher rate of cumulative sleep morbidity than women who were intending to conceive (M=20.33, SD=11.14) and women who were not intending to conceive (M=20.15, SD=12.03) (p=.05). Pregnant women also had increased rates of insomnia (M pregnant=8.38, SD=3.77; M intending=6.86, SD=3.60; M not intending=6.53, SD=3.47; p<.001) and restless legs syndrome/ periodic limb movement disorder (M pregnant=2.77, SD=3.05; M intending=2.02, SD=2.28; M not intending=1.99, SD=2.43; p=.004) as compared to non-pregnant women.

Conclusion: These data suggest, as is widely held, that pregnant women have greater levels of sleep disturbance than women of a common reproductive age who are currently intending to conceive or who are not currently intending to conceive. The observed sleep disturbance appears to be limited to sleep initiation and maintenance and RLS/PLMs symptomatology. Additional analyses are ongoing.

Support: Perlis & Kloss: R21HD083628; Perlis K24AG055602

0869

SLOW WAVE SLEEP IS ASSOCIATED WITH DECREASED RISK OF GESTATIONAL DIABETES MELLITUS

Izci Balserak, B. Bronas, U. Prasad, B. Shah, K. Steffen, A. Carley, D.

University of Illinois at Chicago, Chicago, IL.

Introduction: Pregnancy is associated with disrupted slow-wave sleep (SWS) and a high prevalence of sleep disordered breathing (SDB), which may further exacerbate the decrease of deep sleep.

Reduced slow wave sleep may impair glucose homeostasis, contributing to Gestational Diabetes Mellitus (GDM). Studies investigating EEG markers of deep and light sleep, and their associations with SDB and GDM are lacking. In this study, we measured associations of EEG Delta-power with objective SDB measures assessed in late-pregnancy to determine if changes in these bands are associated with GDM risk.

Methods: 74 women (24-36 weeks pregnancy) underwent overnight polysomnography. Spectral profiles for Delta relative power were created for NREM and REM sleep after removing epochs with movements or muscle artifacts. The association of Delta power with SDB, assessed by the Apnea Hypopnea-Index (AHI) and AHI-based SDB severity (none, mild, moderate, severe) was tested by multivariate linear regression including demographic variables with bivariate correlations (p<0.2) versus Delta-power. Conditional-regression was used to explore relationships between Delta-power and GDM, controlling for covariates.

Results: Obstructive Sleep Apnea (OSA, AHI>5) was present in 14% of subjects (8 GDM-cases and 3 controls). In bivariate analyses, AHI, AHI-severity categories and OSA were associated with Delta-power in NREM (all p<0.2) and AHI was associated with Delta relative-power in REM (p=0.18). However, these associations did not remain significant after adjusting for covariates. Delta relative-power in NREM was significantly associated with decreased risk of GDM (OR:0.50, 95%CI-0.25,0.91), but, in REM sleep, was not associated with GDM risk (OR:1.25, 95%CI-0.79,1.97).

Conclusion: These analyses failed to demonstrate an association between OSA or OSA severity and EEG Delta power. However, lower levels of SWS, characterized by low Delta power were associated with increased GDM risk.

Support: NIH-R00-NR013187

0870

MEDICARE 4% DESATURATION CRITERIA LEAD TO SIGNIFICANT UNDERDIAGNOSIS OF GESTATIONAL OBSTRUCTIVE SLEEP APNEA

Bazalakova, M. Rice, L. Wiedmer, A. Mourey, H. Antony, K. University of Wisconsin Madison, Madison, WI.

Introduction: Use of 4% criteria in non-pregnant populations significantly underestimates obstructive sleep apnea (OSA) prevalence and severity (Budhiraja et al, JCSM 2019). Women are significantly more likely than men to not meet Medicare 4% diagnostic criteria for OSA (Khalid et al, Sleep Vol 42 supplement, 2019). We investigated how use of 4% desaturation criteria affects the diagnosis of gestational sleep apnea (gOSA) in pregnancies complicated by obesity.

Methods: We retrospectively reviewed respiratory event index (REI) and pregnancy outcomes in 56 pregnancies with prepregnancy BMI>30 kg/m2, where home sleep apnea tests (HSATs, Alice PDx) were scored using both 4% and 3% criteria.

Results: 33 out of 56 HSATs met 4% diagnostic criteria for OSA (REI4% > 5/hr). In 4 out of 33 cases (12%), use of 3% rather than 4% criteria changed the REI from 5-15/hr to the 15-30/hr range. An additional 11 studies were negative for gOSA by 4% criteria (REI4% < 5/hr), but met diagnostic criteria for gOSA by 3% criteria (REI4% > 5/hr). All 11 cases were in the "mild" category of REI 5-15/hr. Thus prevalence of gOSA was 58.9% by 4% but 78.6% by 3% diagnostic criteria. In an ongoing analysis of pregnancy outcomes in the two groups, the gestational age at delivery was lowest in the 4%gOSA group (35.9 weeks), but similar and no longer in the preterm range in the 3%gOSA and no gOSA (REI3%<5/hr)

groups (38.7 and 38.3 weeks respectively). However the average baby weight at delivery was lower in the 3%gOSA compared to no gOSA group (3061g and 3580g respectively), suggestive of a potential pathophysiological significance of gOSA defined by <4% desaturation.

Conclusion: The use of 4% desaturation criteria leads to underestimation and missed diagnoses of gOSA complicating pregnancy. As the pathophysiological mechanisms of gOSA have not yet been elucidated, and are likely to differ from those of non-pregnant women and, certainly, men, examination of associated pregnancy morbidity is merited in gOSA defined by 4% versus 3% criteria. **Support:** None.

0871

AIR POLLUTION EXPOSURE AND ADVERSE SLEEP HEALTH ACROSS THE LIFE COURSE: A SYSTEMATIC REVIEW

Liu, J.¹ Wu, T.¹ Chen, J.²

¹University of Pennsaylvania, Philadelphia, PA, ²University of Southern California Keck School of Medicine, 1975 Zonal Ave, Los Angeles, CA 90033, los angeles, CA.

Introduction: An increasing number of epidemiologic studies have examined air pollution as a possible contributor to adverse sleep health, but the results were mixed. The aims of this systematic review are to investigate and summarize the associations between exposures to air pollutants and various sleep measures across the lifespan.

Methods: PubMed, CINAHL, Cochrane, Scopus, Web of Science, and PsycInfo were searched through October 2019 to identify original data-based research examining the direct epidemiological associations between air pollution exposures, both ambient and indoor, and various sleep health measures, including sleep quality, sleep duration, sleep disturbances, and daytime sleepiness.

Results: Twenty-two articles from 2010-2019 spanning a wide range of study populations (from early childhood to elderly) and locations (10 Asian, 4 North American, 3 European, 5 other) were selected for inclusion. Due to variation in both exposure and outcome assessments, conducting a meta-analysis was not plausible. Twenty-one reported a generally positive association between exposure and poor sleep quality. While most studies focused on ambient air pollutants, five assessed the specific effect of indoor exposure. Increased exposure to both ambient and indoor pollutants is associated with increased respiratory sleep problems and a variety of additional adverse sleep outcomes in children and adolescents. In adults, air pollution exposure was most notably related to sleep disordered breathing severity.

Conclusion: Existing literature generally show negative relationships between exposure to air pollution and sleep health across different ages, countries, and measures. While many associations between air pollution and sleep outcomes have been investigated, the mixed study methodologies and use of subjective air pollution and sleep measures result in a wide range of specific associations. Plausible toxicological mechanisms remain inconclusive. Future studies utilizing objective sleep measures and controlling for all air pollution exposures an individual encounters may help ameliorate variability in the results reported by current published literature.

Support: yes

0872

CORRELATES OF RESTLESS SLEEP AMONG FEMALE SEX WORKERS IN BALTIMORE, MD

*Urquhart, G. J.*¹ *Spira, A. P.*² *Park, J. N.*¹ *Sherman, S. G.*¹ ¹Department of Health, Behavior and Society, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ²Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD.

Introduction: Sleep quality is understudied among street-based female sex workers (FSW), a population characterized by high rates of structural vulnerability. FSW may be at higher risk for poor sleep due to food and housing insecurity, violence, substance use and irregular work hours. We studied correlates of restless sleep in an urban, street-based risk environment.

Methods: Data are from a year-long observational prospective cohort study of street-based FSW (n=250) in Baltimore, MD. Baseline survey data, collected April 2016 to February 2017, included topics such as structural vulnerability, mental and physical health, substance use and lifetime violence. Sleep was measured with item 7 on the CES-D-10 Scale; respondents indicated how many days in the past week their sleep was restless (< 1 day, 1-2 days, 3-4 days, 5-7 days). We conducted bivariate and multivariable logistic analysis to identify correlates of experiencing 5-7 days of restless sleep vs. fewer.

Results: Median age was 35 years. Most (66%) were Non-Hispanic White, 62% were homeless, 61% reported food insecurity, and 65% injected heroin. We documented a high prevalence of trauma; 81% reported lifetime sexual or physical violence by family members, intimate partners, paying clients, or police officers, and 62% screened positive for PTSD on the PCL-5 Checklist. 54% reported 5-7 days of restless sleep in the past week. Independent associations with frequent restless sleep included older age (age 35+ aOR: 2.67 [1.64-4.36]), food insecurity (aOR: 2.02 [1.42-2.88]), self-reported poor health (aOR: 3.98 [1.99-7.96]), and lifetime violence: a greater number of violent experiences corresponded with higher odds of restless sleep (1-2 vs. 0 experiences aOR: 2.38 [1.43-3.99], 3-4 vs. 0 experiences aOR: 3.67 [2.22-6.05]).

Conclusion: These data demonstrate high prevalence of restless sleep among street-based FSW with higher risk among those who experience intersecting vulnerabilities and multiple exposures to violence. Trauma-informed structural interventions may improve sleep quality among this population.

Support: This work was supported by the National Institute of Drug Research (R01DA038499-01).

0873

OBSTRUCTIVE SLEEP APNEA IMPACTS BRAIN DEVELOPMENT IN OBESE CHILDREN AND ADOLESCENTS: AN MRI STUDY

Sarzetto, F.¹ Naik, T.² Narang, I.^{2,1} Kassner, A.^{2,1} ¹University of Toronto, Toronto, ON, CANADA, ²The Hospital for Sick Children, Toronto, ON, CANADA.

Introduction: Obstructive sleep apnea (OSA) is a breathing disorder characterized by episodes of nocturnal hypoxia and chronic systemic inflammation, affecting more than 50% of obese youths. Both obesity and OSA independently have a negative impact on brain structure and function, but their combined effect on the developing brain is unknown. The purpose of this study was to assess MRI measurements of cortical thickness (CT) in obese youths with various degrees of OSA severity. We hypothesized that CT is abnormal in obese adolescents with OSA.

Methods: 55 obese subjects (26 females, 29 males, mean 14.3 \pm 2.4 years) were included in the analysis. All subjects were assessed with polysomnography (PSG) to evaluate presence and severity of OSA. T1-weighted MPRAGE images were acquired using a 3T MRI scanner following PSG. CT was extracted using the CIVET 2.1.1 pipeline, and statistical analysis was performed on SurfStat to examine global and regional CT in relation to age using a general linear model.

Results: Based on PSG outcome, subjects were divided into 3 groups, no OSA (OAHI < 1.5 events/hr., n = 15), mild OSA (OAHI < 5, n = 14), and moderate/severe OSA (OAHI ≥ 5, n = 26). Cortical thickness analysis revealed a negative-trending correlation between global CT and age in no OSA (T = -0.49, P > 0.6), as seen in typical development. This correlation weakened in the presence of mild OSA (T = -0.20, P > 0.8) and became significantly positive in moderate/severe OSA (T = 3.87, P = 0.001), affecting several cortical areas.

Conclusion: These results indicate that brain development in obese adolescents with moderate/severe OSA significantly deviates from the typical trajectory of cortical thinning. This thickening could be due to exacerbated inflammation from the combined effect of both diseases, or a neurotrophic effect of leptin. More data is needed to validate these findings.

Support: None

0874

PATIENTS WITH CONGENITAL CENTRAL HYPOVENTILATION SYNDROME DO NOT WAKE UP TO VENTILATOR ALARMS

Mathur, S. K. Kun, S. Keens, T. G. Perez, I. A. Children's Hospital Los Angeles, Los Angeles, CA.

Introduction: Congenital Central Hypoventilation Syndrome (CCHS) requires lifelong ventilatory support during sleep. CCHS patients are vulnerable to sleep disturbances associated with treatments, monitoring alarms, and care they receive. We hypothesized that sleep would be disrupted in CCHS patient's due to ventilatory support and other treatments at night.

Methods: An anonymous survey of CCHS patients aged 0-17 years was conducted through REDCAP. Patients were recruited in person, by flyer, email, and social media. Data collected included demographics, PHOX2B genotype, ventilatory support, treatments, nursing, and sleep parameters.

Results: We received 22 responses (27% Female, 8.1 years \pm 5.7). PHOX2B genotypes were 20/27 PARM (8), 20/26 PARM (2), 20/24

PARM (2), 20/25 PARM (4), ≥ 20/28 PARM (2), and 2 NPARM (2). Two respondents did not indicate the PHOX2B genotype. 13/22 were ventilated by PPV via tracheostomy, 7/22 by BPAP, and 2/22 by diaphragm pacing. Additional treatments received at night included suctioning (8), aerosol (1), G-tube feeding (2), and none (11). Only 9 received nursing at night. 13 used pulse oximetry for monitoring, and 9 used both pulse oximetry and end tidal CO2 monitor. 16/22 rarely woke up due to ventilator or monitor alarms. 15/22 slept within 20 minutes after going to bed. Sleep latency was not affected by mode of ventilation. 11/22 reported night waking \geq 2 nights/week and 10/22 returned to sleep without help after night waking. 6/7 BPAP dependent patients reported low frequency of night waking (0-1 time/week). Of the PPV + trach group, 7/13 reported high frequency of night wakings, mostly 5-7 times/week. Conclusion: Most CCHS patients do not awaken in response to ventilator alarms. Sleep is rarely disrupted by nursing or feeding intervention. We speculate that CCHS patients contemplating to live independently should be tested to see if they awaken in response to ventilator alarms.

Support: None

0875

INTERRELATIONS AMONG RACE, SOCIOECONOMIC STATUS, SLEEP DURATION VARIABILITY, AND NEUROBEHAVIOR IN CHILDREN WITH OBSTRUCTIVE SLEEP APNEA SYNDROME

Williamson, A. A.^{1,2} *Fan, J.*² *Xiao, R.*^{1,2} *Tapia, I. E.*^{1,2} ¹Children's Hospital of Philadelphia, Philadelphia, PA, ²Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA.

Introduction: There are racial and socioeconomic status disparities in child obstructive sleep apnea syndrome (OSAS) and in sleep patterns, but research on sleep patterns in children with OSAS is limited. This project examined interrelations among race, socioeconomic status (SES), sleep duration variability, and neurobehavior in children with OSAS.

Methods: Baseline data were drawn from 464 children with OSAS (M age 7 years, SD 1.4 years; 49% male; 34% Black) participating in the Childhood Adenotonsillectomy Trial. Sleep duration variability was calculated as the coefficient of variation for 5-day sleep diaries. Linear regression was used to examine whether sleep duration variability was associated with child race (Black vs. non-black), SES (family income; maternal education; neighborhood distress index based on US Census data), z-scored body mass index, asthma, prematurity, and secondhand smoke exposure. We then examined whether sleep duration variability was associated with parent-and teacher-reported child neurobehavior including executive functioning (Behavior Rating Inventory of Executive Functioning) and inattention (Conners Rating Scale), adjusting for the variables included in the first regression analysis.

Results: Black race was correlated with increased sleep duration variability (p = .05), but this association was not significant in the adjusted regression model. Secondhand smoke exposure was significantly associated with sleep duration variability (p < .001). Greater sleep duration variability was significantly associated with increased parent-reported neurobehavioral impairments (p=.004 for executive functioning and for inattention), adjusting for race, SES, secondhand smoke exposure, and the other covariates. Sleep duration variability was not associated with teacher-rated child neurobehavior.

Conclusion: In children with OSAS, sleep duration variability is greater in those exposed to secondhand smoke and is linked to increased parent-rated child neurobehavioral impairments. Findings suggest that clinicians should screen for secondhand smoke exposure and that treatment of pediatric OSAS should include a focus on promoting healthy sleep patterns.

Support: Sleep Research Society Foundation and K23HD094905 (AAW).

0876

OBSTRUCTIVE SLEEP APNEA, LOW PAP USE, AND DAYTIME SLEEPINESS ARE ASSOCIATED WITH WEIGHT REGAIN FOLLOWING BARIATRIC SURGERY IN ADOLESCENTS WITH SEVERE OBESITY

Kaar, J. L.¹ Patten, L.¹ Kaizer, A.¹ Hawkins, S. M.² Moore, J. M.¹ Alioa, M. S.³ Inge, T. H.² Simon, S. L.²

¹UNIVERSITY OF COLORADO ANSCHUTZ MEDICAL

CAMPUS, Aurora, CO, ²Children's Hospital Colorado, Aurora, CO, ³National Jewish Health, Denver, CO.

Introduction: Significant weight loss is seen following bariatric surgery, leading to an ameliorative effect on obesity-related comorbidities such as OSA. Weight loss maintenance is a priority, and identifying factors that may be associated with weight loss outcomes following bariatric surgery is of high importance. The current study examined whether OSA symptoms and PAP therapy were associated with weight outcomes following bariatric surgery in adolescents.

Methods: Participants from the Teen-LABS Study, which follows adolescents undergoing bariatric surgery were examined. Demographic and anthropometric data, OSA diagnosis, and PAP prescription and self-reported usage information were assessed 6 months before surgery. Pediatric Sleep Questionnaire (PSQ) responses were utilized from baseline to 48 months post-surgery. All analyses were adjusted for time, age, sex, surgery type, and ethnicity. Results: 242 adolescents (76% female, 72% White, age at surgery = 16.6 [1.6] years) were included. 57% had a diagnosis of OSA at pre-surgical baseline, and 56% of adolescents with OSA reported PAP use at pre-surgery. BMI increase over time from year 1-4 postsurgery was 11% more for those with high PSQ severity compared to those with low PSQ severity (p = 0.01). Those with pre-surgical OSA that reported using PAP "often" or "always" at baseline had an 8% lower increase in BMI from year 1-4 post-surgery compared to those that reported using PAP "rarely" or "sometimes" at baseline (p = 0.004). Finally, endorsing daytime sleepiness on the PSQ was associated with a 11% greater increase in BMI during years 1-4 postsurgery (p = 0.01).

Conclusion: OSA and daytime sleepiness may be associated with greater weight regain following bariatric surgery in adolescents. Adherence to PAP therapy pre-surgery may be a protective factor in preventing or reducing weight regain following surgery. Daytime sleepiness may be an effect of OSA, or due to the insufficient sleep that is prevalent among adolescents. Research is needed to examine the impact of additional aspects of sleep health such as duration, timing, and quality on health outcomes, as well as the impact of PAP adherence and sleep interventions on weight regain following bariatric surgery in adolescents with severe obesity. **Support:** None.

0877

IMPACT OF SLEEP-DISORDERED BREATHING ON QUALITY OF LIFE IN PEDIATRIC CYSTIC FIBROSIS Shakkottai, A. Nasr, S. Z. O'Brien, L. M. Chervin, R. D.

University of Michigan, Ann Arbor, MI.

Introduction: The frequency of sleep-disordered breathing (SDB) may be high among children with cystic fibrosis (CF), a life-shortening, genetic disease that affects 1/3400 Caucasian live-births. Yet, the potential impact of SDB on their quality of life has not been well-studied.

Methods: The Pediatric Sleep Questionnaire Sleep-Related Breathing Disorders (PSQ-SRBD) Scale, a well-validated SDB screening tool, and two validated quality-of-life questionnaires, the Pediatric Quality of Life Inventory (PedsQL[™]) and the Cystic Fibrosis Questionnaire-Revised (CFQ-R), were administered to children 6-17 years of age with CF consecutively during a 2-week period during routine pulmonary clinic visits.

Results: Twenty-two children with CF and their parents completed the questionnaires. Mean age of the participants was $11.6\pm3.8(sd)$ years. Mean body mass index (BMI) percentile was 54.8±27.1%. Mean forced expiratory volume in 1 second percent predicted (FEV1 PPD) was 86.6±22.5%. Five subjects (23%) showed high risk for SDB (PSQ-SRBD Scores > 0.33). Seven additional subjects had PSQ-SRBD Scores of 0.3. Mean BMI percentile was higher among subjects with vs. without positive PSQ-SRBD Scores (66%) vs. 42% respectively, p=0.03). The groups did not differ with regards to FEV1. Pearson/Spearman correlation identified negative associations between PSQ-SRBD Scores and functioning in multiple parent PedsQL domains: physical (R=-0.45, p=0.03), social (R=-0.53, p=0.01), school (R=-0.56, p=0.008); two child PedsQL domains: physical (R=-0.41, p=0.06), school (R=-0.41, p=0.06); multiple parent CFQ-R domains: physical (R=-0.44, p=0.08), respiratory (R=-0.45, p=0.07), emotional (R=-0.54, p=0.02), school (R=-0.70, p=0.002); and one child CFQ-R domain: physical (R=-0.51, p=0.01). None of these domains were associated with FEV1. Conclusion: Children with CF may have SDB symptoms independent of the extent of lung dysfunction. Although the current cross-sectional data cannot prove a causal effect, they raise the possibility that the SDB itself may have adverse impact on physical, emotional, social, and school functioning.

Support: NIH Training Grant (F32HL145915)

0878

ASSOCIATION OF OBSTRUCTIVE SLEEP APNEA WITH INTERNALIZING SYMPTOMS VS. EXTERNALIZING BEHAVIORS IN ADOLESCENTS WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER

Puzino, K.¹ Calhoun, S. L.¹ He, F.¹ Toth, S.¹ Vgontzas, A. N.¹ Liao, D.¹ Bixler, E. O.¹ Fernandez-Mendoza, J.¹ ¹Penn State College of Medicine, Hershey, PA, ²Penn State College of Medicine, Hershey, PA.

Introduction: Attention deficit hyperactivity disorder (ADHD) in children has been associated with insomnia, obstructive sleep apnea (OSA), and abnormal periodic limb movements (PLMS). However, there is lack of data examining the contribution of OSA to ADHD-related internalizing symptoms and externalizing behaviors in adolescents.

Methods: We studied the Penn State Child Cohort, a random general population sample of 700 children ($8.7\pm1.7y$), of whom 421 were followed-up 8.3 years later during adolescence ($17.0\pm2.3y$, 53.9% male). All adolescents underwent a 9-hour PSG, clinical history and physical examination. ADHD was ascertained by a parent- or self-report of having been diagnosed with ADHD. OSA was defined as an apnea hypopnea index (AHI) of ≥ 2 events per hour of sleep, while a periodic limb movement index (PLMI) ≥ 5

events per hour of sleep was indicative of PLMS. Controls, OSAalone, ADHD-alone and ADHD+OSA were identified. The Child or Adult Behavior Checklist were used to ascertain internalizing and externalizing behaviors. Multivariable-adjusted models controlled for sex, race, age, and body mass index (BMI) percentile.

Results: As compared to controls, adolescents with ADHD-alone or ADHD+OSA had significantly greater externalizing behaviors (p<0.001), inattention (p<0.001) and thought problems (p<0.001). While adolescents with ADHD-alone had higher internalizing symptoms (p=0.021), specifically withdrawn-depression (p<0.01), adolescents with ADHD+OSA had more somatic problems than controls (p=0.048). There were no statistically significant differences in behavioral outcomes between controls and adolescents with OSA-alone or between adolescents with ADHD-alone and ADHD+OSA.

Conclusion: Adolescents with comorbid ADHD and OSA do not present with worse behavioral outcomes than those with ADHD alone. Future studies should examine whether the progression of these adolescents into young adulthood differs in terms of their behavioral outcomes and development of mental health disorders. **Support:** National Institutes of Health (R01HL136587, R01HL97165, R01HL63772, UL1TR000127)

0879

OUTCOMES OF ADENOTONSILLECTOMY IN CHILDREN WITH DOWN SYNDROME AND OBSTRUCTIVE SLEEP APNEA

Morello Gearhart, A.¹ Gunaratnam, B.² Senthilvel, E.³ ¹University of Louisville - Department of Pulmonary, Critical Care and Sleep Medicine, Louisville, KY, ²University of Louisville - Department of Bioinformatics & Biostatistics, Louisville, KY, ³University of Louisville - Department of Pediatrics, Louisville, KY.

Introduction: Obstructive sleep apnea (OSA) is highly prevalent in children with Down Syndrome (DS). The aim of this study was to assess the effectiveness of adenotonsillectomy (T&A) on polysomnographic parameters of children with DS.

Methods: Retrospective chart review of children with DS who underwent T&A between 2012-2019 was performed. Preoperative OSA severity was categorized by obstructive apnea-hypopnea index (OAHI): mild = 1-4.9 events/h; moderate = 5-9.9 events/h; severe ≥ 10 events/h.

Results: We identified 43 DS children with pre and post T&A polysomnographic data in a population of 162 DS patients. A total of 25 were male, mean age 5.1 years (± 3.8 years) and 56% Caucasians. Preoperative data showed 19% mild OSA, 30% moderate and 51% severe. Postoperatively, apnea-hypopnea index (AHI) normalized in 9.3%, 37.2% had mild OSA, 18.6% moderate and 34.9% severe. Overall, T&A resulted in significant improvement (p-value <0.05) in mean AHI, (18.51 ± 28.05 vs 11.72 ± 16.43), SaO2 nadir (80.00 ± 14.82 vs 85.51 \pm 5.94), sleep efficiency (81.97 \pm 11.15 vs 85.9 \pm 8.28), arousal index (16.14 \pm 10.23 vs 14.45 \pm 12.34), and wake after sleep onset (67.19 \pm 46.89 vs 50.55 \pm 40.83) and no statistical difference (p-value >0.05) in end-tidal carbon dioxide (43.86 ± 9.56 vs 44.17 \pm 3.78), Rapid Eye Movement (REM)% (15.86 \pm 7.75 vs 15.92 ± 7.41), sleep latency (24.03 \pm 34.39 vs 22.55 \pm 21.11), and central apnea index (0.86 ± 1.38 vs 0.66 ± 0.82) in pre and post T&A data. There was no statistically significant difference in pre and post T&A polysomnographic parameters between 17 DS and 17 age and gender-matched non-DS control subjects.

Conclusion: Adenotonsillectomy resulted in improvement in AHI, oxygen desaturation nadir, sleep efficiency, arousal index and wake after sleep onset. However, a significant portion of children with DS continued to have moderate to severe OSA after T&A. **Support:** None.

0880

A PILOT STUDY EVALUATING THE EFFECT OF HEAD ELEVATION DURING SLEEP ON OBSTRUCTIVE SLEEP APNEA IN PRE-PUBERTAL CHILDREN WITH DOWN SYNDROME

Kaplan, K.^{1,2} *Spielberg, D.*² *Petitto, L.*² *Musso, M.*² *Glaze, D.*² ¹Baylor College of Medicine, Houston, TX, ²Texas Children's Hospital, Houston, TX.

Introduction: Children with down syndrome are at high risk for developing obstructive sleep apnea when compared to typically developing children. Treatment of obstructive sleep apnea is complicated as these children often struggle with traditional therapies such as positive airway pressure. In adult populations it has been shown that head elevation is successful in reducing the severity of OSA (AHI). The hypothesis of this study is that head elevation (30°) would improve OSA in a cohort of pre-pubertal children with down syndrome.

Methods: Children with down syndrome, aged 4-13, presenting to the sleep clinic at Texas Children's Hospital were screened for enrollment into the study (n=21; 11 male). Subjects were randomized to begin a diagnostic polysomnogram with either the head of the bed flat (0°) or elevated (30°). Head position was alternated every 2 hours during the study. Studies were performed in an AASM pediatric sleep center by a registered PSG technologist. Studies were scored using AASM pediatric scoring rules. Data was analyzed using paired student t-tests. Each subject served as their own control.

Results: There was no significant difference in AHI (p=0.71), RDI (p=0.7), O2 nadir (p=0.17), total sleep time (p=0.34), sleep efficiency (p=0.28), time in REM sleep (p=0.94) or arousal index (p=0.14) when the head of the bed was flat (0°) versus elevated (30°). The study shows that head elevation is not successful in significantly reducing obstructive sleep apnea in a pre-pubertal pediatric population of children with down syndrome.

Conclusion: In children with down syndrome, aged 4-13, referred for a diagnostic sleep study, there is no improvement in OSA due to head elevation (30°) when compared to sleeping flat (0°) . These findings were independent of if the subject started with the head of the bed flat or elevated. Other cofounders were eliminated as each subject served as their own control.

Support: No external funding was utilized for this study.

0881

CLINICAL EVALUATION OF ADENO-TONSILLAR HYPERPLASIA IN NON-SYNDROMIC CHILDREN AND ADOLESCENTS DURING GROWTH HORMONE TREATMENT

Nasser, R.¹ Vervloet, T.¹ Eckley, C. A.¹ Amade, S.¹ Dokkedal-Silva, V.² Pires, G. N.² Andersen, M. L.² Tufik, S.² Longui, C.¹ Xavier, S. D.¹

¹Santa Casa de Sao Paulo School of Medical Sciences, Sao Paulo, BRAZIL, ²Universidade Federal de Sao Paulo, Sao Paulo, BRAZIL.

Introduction: Recombinant human Growth Hormone (rhGH) is an important pharmacological agent for linear growth stimulation and body composition in children with growth hormone deficiency (GHD). However, reports indicate that treatment with rhGH can be associated with adeno-tonsillar hyperplasia. This condition can lead to occurrence of obstructive sleep apnea (OSA) and worsening of sleep and life quality. Nonetheless, studies assessing this outcome in non-syndromic children are scarce.

Methods: We evaluated the difference in size of pharyngeal and palatine tonsils in patients aged between 5 and 17 being treated with recombinant human Growth Hormone (rhGH). We conducted a prospective longitudinal observational study. Twelve patients in treatment with rhGH were evaluated by otorhinolaryngological physical examination, nasofibrolaryngoscopy and obstructive sleep apnea (OSA)-18 questionnaire in two different time-points: when selected (T0) and after 6 months (T1).

Results: No significant associations were found regarding palatine and pharyngeal tonsil size with rhGH treatment. In relation to OSA 18 questionnaires, there was no statistically significant result in the absence of covariables for the general score as well as for the five domains that it comprises. When covariables were included in the analyses, controlled by the patients age, we observed statistically significant increases in the general score and in the domains relating to sleep disorders, emotional suffering, diurnal problems and the caretaker's concerns.

Conclusion: In the present study, pharyngeal or palatine tonsils hyperplasia were infrequent during treatment with rhGH in the non-syndromic children and adolescents. However, the impact on sleep and quality of life that may arise warrant careful monitoring during therapy.

Support: Associação Fundo de Incentivo à Pesquisa (AFIP).

0882

LOCAL DEFORMATION ANALYSIS OF LATERAL CEPHALOGRAM FOR CHILDHOOD OSA CLASSIFICATION

Yuen, H. M.¹ Chan, H. L.² Au, C. T.¹ Chan, K. C.¹ Lui, L. M.² Li, A. M.¹

¹Department of Paediatrics, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, HONG KONG, ²Department of Mathematics, The Chinese University of Hong Kong, Shatin, HONG KONG.

Introduction: Craniofacial profile is one of the anatomical causes of obstructive sleep apnea (OSA). Cephalometry provides information on patients' skeletal structures and soft tissues. Traditional cephalometric analysis focuses on linear distances, angles, ratios and area of specific variables. Its classification power is often disappointed. In this study, a novel approach to cephalometric analysis using local deformation information was carried out to assess its efficacy in OSA classification.

Methods: This study was a retrospective analysis based on 60 casecontrol pairs who were Chinese children recruited for sleep studies in the Prince of Wales Hospital, with accessible lateral cephalometry and polysomnography (PSG) data. Local deformation technique was adopted to derive 1215 deformations from 15 manual landmarking on each cephalogram. In addition, three linear distances (hyoid bone to mandibular plane, hyoid bone to posterior pharyngeal wall, and minimal distance between tongue base and posterior pharyngeal wall) were measured from each cephalogram. A total of 1218 information features were obtained per subject. Classification models were built with an equal ratio between OSA and non-OSA groups (defined by OAHI≥1 and OAHI<1 respectively). Forty pairs were used as training data and twenty pairs were used as testing data.

Results: Three model settings which used all 1218 cephalometric features, 800 features, and 500 features were tested. The accuracy for the three settings were 67.5% (sensitivity: 70%, specificity: 65%), 87.5% (sensitivity: 90%, specificity: 85%), and 92.5% (sensitivity: 95%, specificity: 90%) respectively. Apart from the three distances, the 500 topmost discriminative features were predominantly land-marks around the nasal cavity.

Conclusion: A new approach to cephalometric analysis using local deformation information can provide additional details on each cephalogram, hence, achieving better classification. The classification models using 500 features yielded the highest accuracy among the three settings. This setting could benefit most from the comprehensive comparison while avoiding overfitting.

Support: -

0883

IMPACT OF IMPLEMENTATION OF A COMPREHENSIVE POSITIVE AIRWAY PRESSURE PROGRAM

Oppy, K. Huffman, B. Kalra, M. Dayton Children's Hospital, Dayton, OH.

Introduction: The American Academy of Sleep Medicine (AASM) guidelines for treatment of Obstructive Sleep Apnea (OSA) with Positive Airway Pressure (PAP) state good practice standards involve adequate follow up with a clinician tele-monitoring efficacy through objective usage data to ensure acceptable treatment and compliance is met, and provide education, behavioral and/ or troubleshooting interventions. In 2016, Dayton Children's Hospital's pap compliance was 29% due to limited staff support.

Methods: To efficiently implement the AASM guidelines, one dedicated Respiratory Therapist (RT) was assigned to help manage OSA patients at Dayton Children's Sleep Medicine. The RT responsibilities include, PAP therapy education, arranging home PAP system, and a follow-up call within 7 days of setup. Through the tele-monitoring system, the RT assesses compliance and addresses equipment issues and mask fitting at the 4 to 6 week clinic visit. To enhance compliance, a welcome postcard and gift card were implemented. Monthly clinic visits occur until compliance is met, wearing device greater than 4 hours 60% of the time, then appointments are scheduled every 6 months to 1 year.

Results: Since 2016, compliance rate increased from 29% to 58%. There was a year over year growth of number of patients starting therapy from 2017 to 2019, 86 patients were added to the PAP program. In 2019, 60% of 6 to 12 years old met compliance and 51% of 13 to 18 years old.

Conclusion: A comprehensive PAP program resulted in improved compliance and substantial growth. Referring providers and families are more likely to accept PAP therapy when made aware of extensive education and follow up by RT staff. To further improve compliance, especially in the 13 to 18 age range, a desensitization program has recently been implemented.

Support: No support provided.

0884

THE EFFECT OF BARIATRIC SURGERY ON SLEEP DISORDERED BREATHING IN ADOLESCENTS AND YOUNG ADULTS

Koirala, A.

Cincinnati Children's Hospital Medical Center, cincinnati, OH.

Introduction: Childhood obesity is the major risk factor sleep disordered breathing (SDB). Severely obese young people may require bariatric surgery for correction of obesity, if other methods of weight loss have failed. We aim to assess the effect of bariatric surgery on SDB, in adolescents and young adults.

Methods: We conducted a retrospective chart review study of patients who underwent bariatric surgery between January 1, 2006 and August 31, 2019 at Cincinnati Children's Hospital. Only patients who had pre-operative and post-operative polysomnograms were included in the study.

Results: Twenty-seven children and young adults (female: 59.2%) met the criteria for entry into analysis. The mean age of subjects at the time of weight loss surgery was 17.9 years (range: 12.9 to 32.5). Majority of the patients underwent laparoscopic partial gastrectomy (85.2%) and the remaining underwent laparoscopic gastric bypass surgery (14.8%). The average duration of follow up for post study measurements after the surgery was 10.4 months (Range: 0.4 to 57.5). The median Body Mass Index (BMI) was significantly lower at post-surgery (49.9 kg/m²[IOR: 45.4-55.9][pre] vs 39.3 kg/m2[IQR: 33.9-46][post], P<0.001). The median obstructive AHI was significantly reduced at post-surgery (6.7/hr[IQR: 3.1-16.4][pre] vs 2.6/hr[IQR: 1.6-6][post], P= 0.03). Median heart rate (HR) during REM (79 bpm[IQR:67-90][pre] vs 67 bpm[IQR: 59.7-72][post], P<0.0001) and NREM (81 bpm[IQR:65-91][pre] vs 65 bpm[IQR: 58-73][post], P<0.0001) sleep were significantly lower at post-surgery. There was no statistically significant difference in sleep architecture (sleep latency, arousal index and percentage of REM, N1 and N3 sleep, P> 0.05) except N2 sleep (53.1% [IQR:47.7-57.3] [pre] vs 55.7% [IQR:51.4-63.7] [post], P= 0.03) which was significantly increased at post-surgery.

Conclusion: There was significant improvement in BMI and SDB after weight loss surgery in children and young adults. Interestingly, there was a decrease in heart rate during both REM and NREM sleep after surgery which may suggest a decrease in sympathetic activation due to improvement if SDB. The sleep architecture remained unchanged after surgery, except the percentage of N2 sleep. **Support:** Cincinnati Children's Hospital Research Foundation

0885

REDUCTION IN EMERGENCY DEPARTMENT AND INPATIENT HOSPITALIZATION VISITS AND LENGTH OF STAY IN A COHORT OF PEDIATRIC PATIENTS INITIATED ON POSITIVE AIRWAY PRESSURE FOR OBSTRUCTIVE SLEEP APNEA SYNDROME

Xanthopoulos, M. S.¹ Williamson, A. A.¹ Tapia, I. E.¹ Cielo, C. M.¹ Ku, H.¹ Smith, J.¹ Matthews, E.¹ Beck, S. E.¹ ¹Children's Hospital of Philadelphia, Philadelphia, PA,

²Children's Hospital of Philadelphia, Philadelphia, PA.

Introduction: Positive Airway Pressure (PAP) is an efficacious treatment of pediatric obstructive sleep apnea syndrome (OSAS). However, it is unknown whether PAP initiation is associated with reduced healthcare utilization, an important metric of care management. We hypothesized that healthcare utilization would be reduced after initiation of PAP in a cohort of pediatric patients prescribed PAP for OSAS.

Methods: Data were extracted from electronic medical records of 475 patients (Mean±SD age at PAP initiation=7.7±5.7 years; 58.7% male; 40.6% White; 38.3% Black; 18.1% multiracial/other; 12.1% Hispanic/Latinx) prescribed PAP for OSAS and followed in our Sleep Center quality improvement program. We extracted the total number of emergency department (ED) visits and hospitalizations

and computed the related average length of stay (LOS) in hours for these visits in the 18 months prior to and 18 months following PAP initiation.

Results: Paired samples t-tests showed that number of ED visits and hospitalizations, and the related visit LOS, were significantly reduced following PAP initiation. The average number of visits reduced from 2.20 pre-PAP to 1.77 post-PAP initiation [t(474) = 3.48,p<.001, effect size = 0.16], while average LOS reduced from 185.14 hours pre-PAP to 42.94 hours post-PAP initiation [t(474) = 4.81,p<.001, effect size = 0.29]. Findings for the significant reduction in LOS held after adjusting for the number of pre and post-PAP ED visits and hospitalizations, average pre-PAP LOS, and patient demographics (age at the time of initiation; sex; race/ethnicity) using multiple linear regression.

Conclusion: PAP initiation was associated with fewer and shorter ED visits and hospitalizations in a large sample of pediatric patients. We speculate that PAP initiation could help reduce morbidity associated with pediatric OSAS, as well as improve healthcare utilization, capacity management and care in this population. **Support:** K23HD094905 and Sleep Research Foundation (AAW)

0886

CHANGES IN PARENT KNOWLEDGE AND SELF-EFFICACY AND ASSOCIATION WITH ADHERENCE FOLLOWING THE STEPS TOWARDS ACHIEVING RESTFUL SLEEP (STARS) PEDIATRIC PAP THERAPY DESENSITIZATION PROGRAM

Crane, S. C.¹ Taylor, A.¹ Wesley, K. L.² Simon, S. L.² ¹Children's Hospital Colorado, Aurora, CO, ²University of Colorado Anschutz Medical Campus, Aurora, CO.

Introduction: Obstructive sleep apnea (OSA) presents in 2-5 % of youth and has been consistently linked to sleepiness, cognitive deficits, behavior difficulties, and cardiovascular morbidity. PAP effectively treats OSA, however, nonadherence is the most common cause of PAP treatment failure in children. Few adherence interventions have been empirically studied for youth with OSA. The STARS (Steps Towards Achieving Restful Sleep) Clinic is a behavioral program to optimize child tolerance to PAP therapy through a parent class, and in vivo practice. The goal of the study was to examine parent knowledge and self-efficacy following participation in STARS, and associations with subsequent PAP adherence.

Methods: A retrospective chart review was conducted for patients in the STARS program. Items queried included patient demographics, diagnostic and treatment characteristics, pre- and post- STARS responses to a parent self-efficacy and knowledge questionnaire, and polysomnography and PAP adherence data. Paired-samples t-tests examined changes in parent self-efficacy and knowledge pre- to post STARS, and regression analyses examined associations between self-efficacy and knowledge with demographics, treatment-related characteristics, and PAP adherence.

Results: 130 patients completed the STARS program from October 2016 to February 2019. Participants were 8.3 years old ± 6.3 , 63% male, 57% white, 33% Hispanic, with severe OSA (OAHI =22 ± 33). Most participants (87%) had at least one medical comorbidity (e.g., Down Syndrome, 41%). Both parent knowledge and self-efficacy increased significantly from pre- to post- STARS. Approximately 60% of patients were adherent following STARS (defined as ≥ 4 h use and $\geq 70\%$ of days used). Higher post- knowledge, but not efficacy score, was significantly associated with better PAP adherence. **Conclusion:** Parent knowledge and self-efficacy for PAP improved following the STARS program, and greater knowledge was

VII. Pediatrics

associated with better adherence. Future research evaluating the efficacy and effectiveness of the STARS program is needed but preliminary evidence suggests it may be a promising model for improving youth PAP adherence.

Support: None

0887

DIAPHRAGM PACER MALFUNCTIONS REQUIRING SURGICAL REPAIR IN CCHS PATIENTS

Kiang, J. B. Kun, S. S. Shin, C. McComb, G. J. Keens, T. G. Perez, I. A.

Children's Hospital Los Angeles, Los Angeles, CA.

Introduction: Congenital Central Hypoventilation Syndrome (CCHS) is a genetic disorder that results in the loss of autonomic ventilatory control, and patients require ventilatory support during sleep or both sleep and wakefulness. One method of ventilatory support is diaphragm pacing (DP), where electrodes surgically placed on the phrenic nerve are connected to subcutaneously implanted receivers that communicate with external antennas and transmitter. There are limited data on the frequency of DP malfunctions that require surgical revision.

Methods: We reviewed the records of 24 CCHS patients ventilated by DP followed at CHLA from 1990-2019. Records were examined for demographics, PHOX2B mutation, pacing duration/day, date and type of malfunctions, age and time since implantation at malfunction occurrence, and repair success rate.

Results: All 24 patients had thoracoscopic electrode placement. 17/24 (71%) of patients used DP while asleep; 3/24 (13%) during wakefulness only. 4/24 (17%) were not currently using their pacers. 10/24 (42%) patients required at least one surgical intervention (Age at implantation 9 ± 4.6 (SD) years; age at malfunction 12.5 ± 7.4 years). The average time from pacer implantation to malfunction was 3.8 ± 3.5 years. Malfunctions included defective receivers (6), insulation leaks (1), defective electrodes (4), and hardware infection (1). Of 12 unique component repairs, 6/12 (50%) involved changing receivers, 1/12 (8%) involved repairing an insulation leak, 4/12 (33%) involved replacing the electrodes and receivers, and 1/12 (8%) involved hardware extraction. Of the 12 malfunctions, 10 (83%) had successful surgical revision, 2/12 (17%) repairs were not attempted. While awaiting surgical revision, patients were successfully ventilated by unilateral DP.

Conclusion: Nearly half of CCHS patients on DP experienced malfunctions within 11 years of implantation. The most common DP repair was receiver replacement. Patients who are waiting for repair often successfully ventilate while pacing unilaterally. **Support:** None

0888

A PEER-BASED SOCIAL MEDIA INTERVENTION TO PROMOTE CONTINUOUS POSITIVE AIRWAY PRESSURE ADHERENCE IN ADOLESCENTS WITH OBSTRUCTIVE SLEEP APNEA

*Watach, A. J.*¹ *Bishop-Gilyard, C. T.*² *Ku, H.*³ *Afolabi-Brown, O.*³ *Parks Prout, E.*⁴ *Xanthopoulos, M.*³

¹University of Pennsylvania, Center for Sleep and Circadian Neurobiology, Philadelphia, PA, ²University of Pennsylvania, Psychiatry and Center for Weight and Eating Disorders, Philadelphia, PA, ³Children's Hospital of Philadelphia, Sleep Center, Philadelphia, PA, ⁴University of Pennsylvania, Department of Pediatrics, Perelman School of Medicine, Philadelphia, PA.

at communicate intervention than adolescents, viewing content 65%, 75%, 85%, 90%, and 100% (n=2) of days versus adolescents who viewed content 0%, 20% (n=2), 40%, 75% and 100%. Semi-structured intervention that adolescents with the large structure intervention of the structure intervention of the structure intervention.

post-intervention.

views revealed the intervention was perceived positively; learning/ expanded knowledge, gaining a sense of community, and enjoyment in the opportunity to help others were commonly identified. Interview feedback revealed utilizing a different social media platform may be more beneficial for adolescent engagement. Participants noted the intervention promoted conversations between the adolescent and caregiver, and 4/6 teens cited increased motivation to use CPAP. Average CPAP use increased in 50% of participants (n=3); 33% (n=2) sustained their use, and one decreased use.

Introduction: Continuous positive airway pressure (CPAP) ad-

herence in adolescents with obstructive sleep apnea (OSA) is

suboptimal. This study evaluated an innovative CPAP adherence

intervention for adolescents and their caregivers delivered via pri-

vate Facebook groups. Study aims: (1) determine feasibility and acceptability of the intervention and (2) assess CPAP use pre- and

Methods: A pilot cohort study design was employed (N=6 dyads).

Intervention included psychoeducation, CPAP use downloads/

feedback, promoting peer-engagement through posts, videos, and

polls. Adolescent and caregiver groups ran separately and simul-

taneously over 4-weeks. Measures: demographics, engagement/

participation data, CPAP use, semi-structured interviews. Analysis:

Results: Adolescents were Black/African American (100%), males

(100%) with a median age of 16 years (range 13-17). Caregivers

(n=6) were mothers (67%) and fathers (33%) with a median age

of 47 years (range 38-55). Caregivers were more engaged with the

descriptive statistics and thematic content analysis.

Conclusion: Participants consistently noted appreciation for knowledge gained and sense of community derived from the intervention. This study supports the potential utilization of social media platforms to not only provide reliable OSA/CPAP education but to also promote peer-engagement. Given the acceptability and increased CPAP use in this small sample, a larger trial is indicated. **Support:** Lead author receives support from NIH/NHLBI Award T32 HL07953. Videos included in intervention supported by The Children's Hospital of Philadelphia Metabolism, Nutrition and Development Research Affinity Group Pilot and Feasibility Grant.

0889

UTILITY OF PULSE WAVE AMPLITUDE IN DETECTING AROUSALS IN CHILDREN

Al-Shawwa, B. Cruz, J. Ehsan, Z. Ingram, D. G. Children's Mercy Hospital, Kansas City, KS.

Introduction: Identifying and scoring arousals are crucial to score hypopneas and respiratory efforts related arousals (RERAs). At the same time, children's sleep architecture appears to be more stable compared to adults which makes it more difficult to score arousals. Pulse wave amplitude (PWA) is a signal obtained from finger photoplethysmography which is directly and positively correlated to finger blood flow. It is also used as a marker for finger vasoconstriction reflected by decreased signal amplitude and as a surrogate for autonomic and cortical arousals. Our aim was to study the effect of scoring arousals based on PWA on scoring of respiratory events in pediatric patients.

Methods: Ten polysomnograms for patients between the ages of 5-15 years who had apnea-hypopnea indices between 1 and 3 events/hour were identified. Patients with syndromes were excluded. A drop in PWA signal of at least 30% that lasted for 3

SLEEP, Volume 43, Abstract Supplement, 2020

seconds was needed to identify subcortical/autonomic arousals. Arousals were rescored based on this criteria and subsequently respiratory events were rescored. Paired t-tests were employed to compare PSG indices scored with or without PWA incorporation. Results: The sample of 10 children included 2 females, and the average age was 9.8 +/-3.1 years. Overall, polysomnography revealed an average total sleep time of 464.1 +/-25 mins, sleep efficiency of 92% +/-4.2, sleep latency of 19.6 +/-17.0 mins, rapid eve movement (REM) latency 143 +/-66 mins, N1 3.9% +/-2.0, N2 50.3% +/-12.0, N3 28.2% +/-9.1, REM 16.7% +/-4.0, and wakefulness after sleep onset (WASO) 18.1 +/-7.5 mins. Including arousals from PWA changes, respiratory indices significantly increased including total AHI (2.3 +/-0.7 vs 5.7 +/-2.1, p<0.001), obstructive AHI (1.45 +/-0.7 vs 4.8 +/-1.8, p<0.001), and RDI (2.36 +/-0.7 vs 7.6 +/-2.0, p<0.001). Likewise, total arousal index was significantly higher (8.7 +/-2.3 vs 29.4 +/-6.5, p<0.001).

Conclusion: Pulse wave amplitude is a useful marker for arousals that are not otherwise easily identified in pediatric polysomnography. This in turn would lead to a more accurate scoring of respiratory events and severity assessment of sleep disordered breathing. This likely will have clinical and therapeutic implications. **Support:** None

0890

SLEEP DISORDERED BREATHING IS ASSOCIATED WITH ENDOTHELIAL DYSFUNCTION AND ATHEROSCLEROSIS IN YOUNG ADULTS: PRELIMINARY LONGITUDINAL FINDINGS IN THE PENN STATE CHILD COHORT

Fernandez-Mendoza, J.¹ Gao, Z.¹ Brandt, K.¹ Houser, L.¹ Calhoun, S. L.¹ He, F.¹ Liao, J.¹ Vgontzas, A. N.¹ Liao, D.¹ Bixler, E. O.¹

¹Penn State College of Medicine, Hershey, PA, ²Penn State College of Medicine, Hershey, PA, ³Penn State College of Medicine, Hershey, PA.

Introduction: Sleep disordered breathing (SDB) in middle-age is an established risk factor for cardiovascular disease. However, population-based studies supporting its cardiovascular contribution at earlier stages of development are lacking, particularly with long-term follow-ups.

Methods: The Penn State Child Cohort is a population-based longitudinal sample of 700 children ($8.7\pm1.7y$), of whom 421 were followed-up 8.3 years later during adolescence ($17.0\pm2.3y$) with in-lab polysomnography (PSG). To date, 425 have been followed-up another 7.4 years later during young adulthood ($24.4\pm2.6y$) via a standardized survey and 136 of them (55.1% female, 21.3% racial/ ethnic minority) have undergone a repeat of their PSG to ascertain apnea/hypopnea index. Subjects (n=121) also underwent Doppler ultrasounds to assess flow-mediated dilation (FMD) and carotid intima-media thickness (CIMT). Linear regression models stratified by body mass index in young adulthood.

Results: SDB was cross-sectionally associated with lower FMD (β =-0.239, p=0.008) and greater CIMT (β =0.330, p<0.001) in young adulthood. Longitudinally, childhood (n=121) and ado-lescence (n=90) SDB were significantly associated with CIMT (β =0.327, p<0.001 and β =0.286, p=0.006, respectively), but not with FMD (β =-0.158, p=0.08 and β =-0.101, p=0.35, respectively). These associations, particularly longitudinal ones between childhood and adolescence SDB with CIMT in young adulthood, were stronger in overweight than normal weight subjects (e.g., β =0.310, p=0.030 and β =0.089, p=0.582, respectively).

Conclusion: SDB and obesity appear to be synergistically associated with endothelial dysfunction and atherosclerosis in young adults from the general population. These data suggest that a childhood exposure to chronic SDB is associated with long-term atherosclerosis, while endothelial dysfunction may be a short-term outcome. This ongoing 16-year longitudinal study will test whether the natural history of SDB from childhood through adolescence into young adulthood shows differential trajectories for cardiovascular morbidity.

Support: National Institutes of Health (R01HL136587, R01HL97165, R01HL63772, UL1TR000127)

0891

CARDIOVASCULAR CONTROL IS IMPAIRED IN CHILDREN WITH DOWN SYNDROME AND SLEEP DISORDERED BREATHING

Horne, R. S.¹ Sakthiakumaran, A.¹ Bassam, A.¹ Thacker, J.¹ Davey, M. J.² Nixon, G. M.²

¹Department of Paediatrics, Monash University, Melbourne, AUSTRALIA, ²Monash Children's Hospital, Melbourne, AUSTRALIA.

Introduction: Children with Down Syndrome (DS) are at increased risk for sleep disordered breathing (SDB). In typically developing (TD) children, SDB is associated with adverse cardiovascular effects including elevated heart rate and blood pressure and impaired autonomic control. The aim of this study was to compare the cardiovascular effects of SDB in children with DS to those of TD children with and without SDB.

Methods: 44 children with DS (3-18 y) were age and gender matched with 44 TD children without SDB (TD-) and with TD children with matched severity of SDB (TD+). Height, weight and blood pressure were measured and BMI, systolic and diastolic z-scores calculated. Heart rate variability (HRV) was calculated for 2 min artefact free epochs overnight. Power spectral density for the low frequency (LF), high frequency (HF), total power (TP) and the LF/HF ratio (sympathovagal balance) were calculated. Data were compared between groups with Kruskal-Wallis one-way ANOVA.

Results: Wake heart rate, systolic and diastolic z-scores were not different between groups. LF/HF was higher in the DS group compared to both TD+ (p<0.05) and TD- (p<0.01) in wake and total sleep time. During total sleep HF power was lower in DS compared to TD+ (p<0.01). In N2 TP and HF were lower and LF/HF higher in DS compared to both TD+ (p<0.01) and TD- (p<0.05). In N3 HF was lower in DS compared to TD+ (p<0.05) and LF/HF was higher compared to both TD+ and TD- (p<0.001 for both) and in REM LF/HF was higher compared to TD+ (p<0.01).

Conclusion: In children with DS and SDB, autonomic cardiovascular control is impaired compared to TD children matched for SDB severity and to non-snoring TD children. Our findings demonstrate significantly reduced parasympathetic activity (reduced HF power) and increased sympathovagal balance, that may contribute to increased risk of cardiovascular disease later in life. **Support:** This study was funded by the Jack Brockhoff Foundation.

0892

SLEEP ABNORMALITIES IN CORNELIA DE LANGE SYNDROME

*Villalobos, R.*¹ *Villalobos, A. P.*² *Villalobos Jr., R.*³ *Villarreal, I. A.*⁴ *Grimaldo, D.*⁴ *Valencia, J. A.*⁴

¹University of Texas at Rio Grande Valley, Harlingen, TX, ²Valley Baptist Medical Center, Saint Joseph Academy, TX, ³Valley Regional Medical Center, Saint Joseph Academy, TX, ⁴Valley Baptist Medical Center, Brownsville, TX.

Introduction: Cornelia de lange is an autosomal dominant disorder (when associated with NIPBL, RAD21, or SMC3 genes) with an incidence of 1:10,000 to 1:50,000 live births, patients affected are known to have a wide variety of sleep disorders, those range from insomnia and abnormal circadian cycle to sleep disordered breathing and hypoventilation. The exact etiology of increased risk of sleep-disordered breathing in patients affected is not fully understood. It is possible that some facial features in these patients expose them to a higher risk (micrognathia, high arched palate, and short neck). We wanted to analyze the sleep related problems in CDLS.

Methods: We included 3 patients with the disorder, age range from 15 months to 16 years old. All patients met criteria for CDLS diagnosis, all had intellectual disability and behavioral associated symptoms. The somnology evaluation included questionnaires of diurnal behavior and sleep focused logs. We performed nocturnal polysomnography in only 2 patients due to inability to tolerate the test in one case.

Results: Sleep clinical information was abnormal in all the cases. Overnight behavioral video evaluation was done. The behavioral abnormalities were evident in all subjects and severe in one.

Overnight polysomnography demonstrated a moderate to severe degree of OSA, delayed sleep onset suggestive of insomnia, sleepwake transition disorder with elevated WASO time, and arousal disorder with elevated spontaneous arousal index. It is of interest the finding of sleep related hypoxemia with limited evidence of obstructive component in one patient.

Conclusion: The abnormalities in sleep are frequent in CDLS, there are wide and present in the sleep architecture and the sleep ventilation, sleep apnea syndromes are frequent but are not the only major sleep-related abnormalities. When CDLS is caused by mutations in the HDAC8 or SMC1A gene, the condition has an X-linked dominant pattern inheritance. Most cases result from new mutations in the HDAC8 or SMC1A gene and occur in people with no history of the condition in their family, likely our cases are related to this mode of transmission and potential different patters of sleep disruption are dependent on different genes involved. **Support:** None

0893

MYOFUNCTIONAL THERAPY IN CHILDREN WITH MILD OBSTRUCTIVE SLEEP APNEA: A META-ANALYSIS

Bandyopadhyay, A.¹ Kaneshiro, K. N.² Camacho, M.³

¹Indiana University School of Medicine, Division of Pediatric Pulmonology, Allergy and Sleep Medicine, Indianapolis, IN, ²Ruth Lily Medical Library, Indianapolis, IN, ³Tripler Army Medical Center, Honolulu, HI.

Introduction: OSA affects 2-4% of children and untreated OSA can have adverse behavior and quality of life outcomes. 40% of children can have residual obstructive sleep apnea (OSA)despite first line treatment (adenotonsillectomy). Alternative modalities of treatment for OSA are limited. Myofunctional therapy comprises of exercises targeting upper airway muscles that can improve facial growth and have been shown to treat OSA in adults. There is paucity of data on the role of myofunctional therapy (MT) in children. The objective of this study was to systematically review

the literature for articles evaluating myofunctional therapy (MT) as treatment for OSA in children and to perform a meta-analysis on the polysomnographic and mouth breathing data.

Methods: Medline, Embase, CINAHL, Scopus, Web of Science and Cochrane were searched from inception through October 1st, 2019. The Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) statement was followed.

Results: Eight studies (91 patients) reported polysomnography and/or mouth breathing outcomes. The pre- and post-MT apnea hypopnea indices (AHI) decreased from a mean \pm standard deviation (M \pm SD) of 3.75 \pm 3.14/h to 2.08 \pm 2.48/h, mean difference (MD) -1.6 [95% confidence interval (CI) -2.42, -0.78], P =0.0001. Mean oxygen saturations improved from 96.03 \pm 1.1% to 96.67 \pm 0.95%, MD 0.42 (95% CI 0.21, 0.63), P <0.0001. Lowest oxygen saturations improved from 86.6 \pm 7.3% to 90.94 \pm 3.05%, MD 1.01 (95% CI 0.25, 1.77), P = 0.009. Mouth breathing decreased in all three studies reporting subjective outcomes.

Conclusion: Current literature demonstrates that myofunctional therapy decreases apnea-hypopnea index by approximately 45% in children with mild obstructive sleep apnea. Mean oxygen saturations, lowest oxygen saturations and mouth breathing outcomes improved in children. Myofunctional therapy could serve as an adjunct to other obstructive sleep apnea treatments. **Support:** None

0894

A TWO ITEM QUESTIONNAIRE REFLECTING PARENTAL CONCERN AS AN INDICATOR OF THE SEVERITY OF OBSTRUCTIVE SLEEP APNEA IN CHILDREN

Tauman, R. Lavi, M. Greenfeld, M. Fishman, G. Wasserzug, O. DeRowe, A.

Tel Aviv Souraski Medical Center, Tel Aviv, ISRAEL.

Introduction: The currently available questionnaires based on parental reporting of OSA symptoms include large number of items and are not sufficient to the diagnosis and assessment of OSA severity. We aimed to evaluate a simple 2-item questionnaire regarding parental concern as a predictor of the severity of OSA in children as measured by polysomnography.

Methods: Prospective analysis of parental concern regarding their children referred for PSG due to suspected OSA. Parents of all study children completed the brief Parental Concern Scale (PCS) questionnaire and the validated Pediatric Sleep Questionnaire-Sleep-Related Breathing Disorder questionnaire (PSQ-SRBD). The PCS consisted of 1 question on the need for surgery and 1 question on concerns about the child's breathing.

Results: Ninety-five children (mean age 4.2 ± 2.5 years, 52% males, mean body mass index z score 0.45 ± 1.8) were recruited. Twenty-three children (24%) had moderate-severe OSA and were referred for adenotonsillectomy. Significant correlations were found between the need for surgery score and the apnea-hypopnea index (r=0.22, P = .029), as well as the mean SpO2 levels (r=-0.24, P = .02). The likelihood for the diagnosis of moderate-severe OSA by PSG increased as parental ranking for the need for surgery increased (P = .003). The need for surgery score and the child's age were the only predictors for moderate-severe OSA (P = .01 and P = .043, respectively).

Conclusion: Querying parents on their perception of their child's need for surgery is a sensitive, practical, and easy-to-use tool that can help the clinician in prioritizing referral to PSG. **Support:**

0895

PEDIATRIC POSITIVE PRESSURE THERAPY: ENHANCING EDUCATION AND PARENT/GUARDIAN CONFIDENCE TO IMPLEMENT THERAPY

Moore, W. R. Anderson, S. E. Burr, L. E. Clark, P. R. Peine, N. A. Sydejko, D. M. Wahl, A. M. Hale, A. M. Baughn, J. M. Mayo Clinic, Rochester, MN.

Introduction: Pediatric positive airway pressure (PAP) therapy can be challenging for children and their parent/guardian (PG). These challenges are complicated by the need to facilitate behavioral competency in the management of both device equipment and therapy. Methods: As part of a larger quality management project aimed to improve PG and patient satisfaction we developed a targeted education visit provided by registered nurses prior to PAP trial initiation. Principle aims were to measure satisfaction with education and increase PG confidence in ability to manage therapy. The nurse session included: education on Obstructive Sleep Apnea (OSA) and PAP therapy, development of plan for first month of therapy, concepts of desensitization, regular practice, discussion and identification of concerns (assessment of barriers to implementation). A survey was administered pre-intervention to assess PG confidence in ability to manage therapy. Post-intervention, PG rated the interventions impact on baseline confidence, as well as an assessment of satisfaction with the education and content. Descriptive statistics were utilized to summarize the Likert survey questions.

Results: A total of 21 of 25 (84%) participants completed surveys. 100% of PG that responded was either extremely satisfied or very satisfied with the educational intervention. Prior to the intervention, 19% (N4) indicated little to no confidence, 19% (N4) indicated some confidence and 62% (N13) indicated being very confident. 62% (N13) of PG's indicated the information covered was new to them, while 24% (N5) indicated some information was new and 14% (N3) indicated none of the information was new. Regardless of baseline confidence, 100% (N21) responded that the intervention further improved confidence to a great degree.

Conclusion: The nurse visit resulted in high satisfaction with care and education and impacted PG confidence in ability to implement treatment. Future studies are needed to determine the impact on patient compliance.

Support:

0896

NUTRITIONAL STATUS IMPROVES FOLLOWING THE IMPLEMENTATION OF POSITIVE AIRWAY PRESSURE FOR THE TREATMENT OF OBSTRUCTIVE SLEEP APNEA IN YOUTH WITH DOWN SYNDROME

Arputhan, A.¹ Xanthopoulos, M. S.¹ Tapia, I. E.¹ Hernandez, P.⁴ Kelly, A.¹

¹Children's Hospital of Philadelphia, Philadelphia, PA,

²Children's Hospital of Philadelphia, Philadelphia, PA,
³Children's Hospital of Philadelphia, Philadelphia, PA, ⁴Perleman School of Medicine, University of Pennsylvania, Philadelphia, PA.

Introduction: In typically developing youth, increases in body mass index (BMI) and rates of obesity accompany treatment of obstructive sleep apnea syndrome (OSAS) with adenotonsillectomy regardless of baseline BMI and OSAS severity. Residual OSAS following adenotonsillectomy and overweight/obesity are common in Down syndrome (DS). We sought to examine the impact of positive airway pressure (PAP) on BMIZ in youth with DS and OSAS. **Methods:** Baseline, 6, and 12 month height/length and weight as well as pre-PAP PSG data were abstracted from the Children's Hospital of Philadelphia Sleep Center for patients with DS and OSAS initiated on PAP between 01/01/2014-07/11/01/2017 (N=73; Median age=6.6y IQR: 3.6-12.1; 52% White, 29% Black; 42% Male). BMIZ was calculated. Longitudinal mixed effects models adjusted for adherence from 0-6 months, baseline BMIZ, and baseline SpO₂ nadir were used to evaluate change in BMIZ at months 6 and 12 and the impact of baseline BMIZ on trajectories.

Results: OAHI (median; IQR) at initiation was 15.9 (8.1-28.9) events/hour, SpO₂ nadir was 83% (77-88), and BMIZ was 1.50 (0.94-2.34). No differences in BMIZ at 6 and 12 months compared to baseline BMIZ were found (p>0.2 for both). Baseline BMIZ was associated with BMIZ at 6- and 12 months (β -coefficient=0.99; p<0.0001); the increase in BMIZ at 12 mo (β -coefficient= 0.49, p=0.001) was offset with decreasing BMIZ (12mo*baseline BMIZ β -coefficient= -0.3; p<0.0001); such that lower BMIZ was associated with increases in BMIZ while higher BMIZ was associated with decreases in BMIZ.

Conclusion: Initiation of PAP has a beneficial impact on nutritional status in youth with DS and OSAS. In youth who are at the lower end of BMIZ, BMIZ increases to a healthier status following the initiation of PAP, and in youth who are at the higher side of BMIZ, BMIZ decreases to a healthier status. Prospective studies are needed to elaborate on these associations.

Support: None

0897

SLEEP DISORDERED BREATHING IN PEDIATRIC PATIENTS WITH TURNER SYNDROME

Yu, Y. A. Vaughn, B. V.

University of North Carolina at Chapel Hill, Chapel Hill, NC.

Introduction: Turner syndrome (TS) is a common genetic disorder that affects phenotypic females with partial or complete absence of one X chromosome. It typically presents with characteristic facial appearance, neck webbing, lymphedema, linear growth failure, and ovarian insufficiency. TS is also associated with other disorders, though sleep related disorders are not commonly reported. We present a case series of pediatric patients diagnosed with TS and assess their risk for sleep disordered breathing.

Methods: This study utilized retrospective chart review of the electronic medical record at the University of North Carolina at Chapel Hill from April 2014 to January 2019. Only pediatric patients under the age of 18 years who had previously undergone polysomnography and carrying the diagnosis of Turner syndrome were included in this study. Polysomnography results were reviewed. **Results:** Retrospective chart analysis yielded ten (10) patients who qualified for inclusion. The mean age was 8.3 years (age range 1-15 years). Nine (9) patients were found to have sleep disordered breathing ranging from upper airway resistance syndrome to moderate sleep apnea (AHI range 1.2 to 6.2). Six (6) patients were found to have elevated periodic limb movement indices (PLM index range 5.1 to 30). Parasomnias and hypoventilation were not seen.

Conclusion: Our case series illustrates that sleep disordered breathing may be more common in TS than previously realized. Eklund et al. found that females with TS had more retrognathic mandibles and maxillas, shorter mandibles, and larger cranial base angles. These findings may indicate elevated risk of sleep apnea. Further studies are needed to define the overall risk of sleep disordered breathing in TS.

Support: None.

0898

THE ACCURACY OF SPLIT-NIGHT STUDY IN ASSESSING OBSTRUCTIVE SLEEP APNEA (OSA) IN CHILDREN AND ADOLESCENTS

Bedoya, M.¹ Chini, B.¹ Armoni-Domany, K.¹ Boh, M.¹ Huang, G.² Md Hossain, M.² Simakajornboon, N.¹

¹Division of Pulmonology and Sleep Medicine, Cincinnati Children's Hospital Medical Center, Cincinnati, OH, ²Division of Biostatistics and Epidemiology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.

Introduction: Split-night polysomnography (PSG) can be used for the diagnosis of OSA, if clinically appropriate, per AASM recommendation based on adult data. However, there are limited data in pediatric population. The aim of our study is to assess the accuracy of split-night-PSG compared with a full-night-PSG in children with OSA.

Methods: A retrospective review was performed in children and adolescents who were diagnosed with OSA during a diagnostic PSG (oAHI >1). Sleep and respiratory parameters from the full-night-PSG(F) were compared with the first three hours of the same PSG(S). Subgroup analysis was performed for age and OSA severity. The results were reported as mean \pm SD. All variables were compared with paired T-test.

Results: 226 met the criteria for entry into analysis. The mean age was 7.8y \pm 5.8. For the whole cohort, there were no significant differences in the mean AHI and oAHI [7.3 \pm 11.7(F) vs6.9 \pm 13.9(S),p=0.26] between full-night and split-night studies. Subgroup analysis revealed that children aged 2-12 yo (n=120) had significant differences in the mean AHI [6.1 \pm 7.1(F)vs4.8 \pm 6.4(S) p=0.002] and the mean oAHI [4.8 \pm 5.6(F)vs3.9 \pm 5.4(S)p=0.03]. In addition, subgroup analysis of mild OSA (n=137) showed a significant difference in the mean AHI (p=0.006), but not in the mean oAHI (p=0.08). There were no significant differences in the mean AHI and oAHI in the moderate to severe OSA group. Based on the first 3 hours of PSG, 16.8%(n=38) of patients were inaccurately classified to have no OSA, while the severity was misclassified in 37.6%(n=85) of patients (underestimated in 30%(n=68) and overestimated in 7.5%(n=17)).

Conclusion: Although there were no differences in the mean AHI and oAHI, split-night-PSG misclassified diagnosis and severity in a significant proportion of our pediatric cohort. In addition, the accuracy of split-night-PSG is influenced by age and severity of OSA. Further studies are needed to identify factors that play a role in these differences.

Support: Cincinnati Children's Hospital, Children's Foundation

0899

LONG-TERM EFFECTS OF ADENOTONSILLECTOMY IN CHILDREN DIAGNOSED WITH OBSTRUCTIVE SLEEP APNEA ON RISK FACTORS FOR CARDIOVASCULAR MORBIDITY

Even Tsur, J.¹ Auhasira, R.² Shiloh, A.² Novack, V.² Goldbart, A.³ ¹Dept. of Pediatrics,Saban Pediatric Medical Center,Soroka University Medical Center, Beer Sheva, ISRAEL, ²Clinical Research Center, Soroka University Medical Center, Beer Sheva, ISRAEL, ³Dept. of Pediatrics, Saban Pediatric Medical Center, Soroka Medical Center, Beer Sheva, ISRAEL.

Introduction: Obstructive sleep apnea (OSA) is an independent risk factor for cardiovascular morbidity in adults. In children, cardiovascular morbidity associated with OSA is usually thought to

resolve after tonsillectomy and adenoidectomy (T&A). There is no information regarding the long term effects of T&A on future cardiovascular morbidity in children diagnosed with OSA. In this study, we performed data mining to assess long-term effects of adenotonsillectomy on risk factors for cardiovascular disease, in young adults.

Methods: This study retrospectively investigated the population defined by a previous study in our institution [Tarasiuk etal Pediatrics 2004] and compared a group of children diagnosed with OSA and underwent T&A(n=130) to a group of children diagnosed with OSA that did not undergo T&A(n=90) to a control group without OSA (n=505). Demographic data, vital signs, anthropometric measurements, medical diagnoses (9th revision (ICD-9) codes) and medication purchases were captured from the HMO computerized database, between the years 1998-2018. When appropriate, univariate comparisons were made using χ^2 -test or Fisher's exact test for categorical variables, and one-way ANOVA or Kruskal-Wallis tests for quantitative variables. We performed multivariate logistic regression to model the factors associated with the diagnosis of obesity. IBM SPSS software, version 25.0, was used for statistical analysis.

Results: We have found that 20 years after their OSA diagnosis, patients (25.1 years, 52.2% males, 26.2 BMI) who were diagnosed with OSA at age 5 and did not undergo T&A consumed more medications associated with cardiovascular morbidity (anti-hypertensive, statins, aspirin) than those who underwent T&A(P<0.001). Surprisingly, multivariate logistic regression revealed that only females diagnosed with OSA (with or without T&A) were diagnosed as obese in comparison to those that did not have OSA (P<0.001). **Conclusion:** Children who were diagnosed with OSA and were not operated will consume more medications (anti-hypertensive, anti-hyperlipidemia, aspirin) as young adults, a surrogate marker for early cardiovascular disease. OSA in girls seems to serve as a risk factor for obesity in their third decade of life. It is important to diagnose and treat OSA in children, and to monitor and prevent obesity, mainly in females.

Support: Israel Science Foundation (ISF) 1344/15

0900

OBSTRUCTIVE SLEEP APNEA SEVERITY, SYMPTOMS OF ATTENTION DEFICIT HYPERACTIVITY DISORDER AND OTHER COMORBID PSYCHIATRIC DISORDERS IN CHILDREN AND ADOLESCENTS: A RETROSPECTIVE DATA ANALYSIS.

Skoulos, M.¹ Sedky, K.¹ Bennett, D.³

¹Cooper University Hospital, Camden, NJ, ²Cooper University Hospital, Camden, NJ, ³Drexel University, Philadelphia, PA.

Introduction: Children and adolescents with obstructive sleep apnea (OSA) are often diagnosed with attention deficit hyperactivity disorder (ADHD). However, the connection between the severity of Apnea/Hypopnea Index (AHI) and ADHD is controversial with research evidence pointing in opposing directions.

Methods: A retrospective study was conducted in a pediatric sleep center at a university hospital setting to investigate the effect between AHI severity, ADHD and/or other comorbid psychiatric disorders. One hundred and thirty-eight participants between the age of 6 and 18 were examined in terms of AHI severity level and their correlation with scores from the Child Behavior Checklist (CBCL) using SPSS program.

Results: A negative correlation between AHI scores and Attention Problems for the entire group of participants was found.

Additionally, female adolescents had positive correlations between AHI scores and several affective disorder variables from the CBCL, while male adolescents had negative correlations between AHI levels and several CBCL scores that are typically associated with ADHD and Anxiety disorders.

Conclusion: This study suggests a relationship between OSA severity and psychiatric conditions. However, this relationship can vary depending on age, gender and AHI severity. More research is required to understand this relationship.

Support: Chervin, R.D. How many children with ADHD have sleep apnea or periodic leg movements on polysomnography? Sleep. 2005: 28(9): 1041-1042.

Sedky K, Bennett DS, Carvalho KS. Attention deficit hyperactivity disorder and sleep disordered breathing in pediatric populations: A meta-analysis. Sleep Medicine Reviews. 2014; 18: 349-356

0901

ROLE OF NASOPHARYNX RADIOGRAPHY IN DIAGNOSIS OF SLEEP DISORDERED BREATHING IN CHILDREN

Rao, H. S.

Penn State Hershey Children's Hospital, HERSHEY, PA.

Introduction: Sleep disordered breathing (SDB) in children comprises a wide spectrum of presentation ranging from primary snoring (prevalence 6-12%) to obstructive sleep apnea (OSA; prevalence 1-3%). In younger children, SDB is often secondary to adenotonsillar hypertrophy. Polysomnography is the gold standard test for evaluation of sleep disordered breathing. Yet, many providers order ancillary tests such as Radiography of Nasopharynx (NPX-ray) to assess for adenoidal hypertrophy. Previous studies evaluating efficacy of NPX-rays to diagnose airway obstruction have shown mixed results.

Methods: Retrospective chart review of 65 consecutive normally developed children who have had diagnostic polysomnography and NPX-rays over the past 3 years was performed. Data on age, sex, Obstructive Apnea Hypopnea Index (OAHI) and NPX-ray findings were collected. Our cohort mostly had the following diagnosis such as GERD, Asthma, Allergic rhinitis, ADHD and Obesity. Our cohort did not have neurological disorders or medications that could affect tone of upper airway. Standard pediatric AHI criteria was applied to categorize mild, moderate and severe OSA.

Results: Our cohort's age ranged from 3-18 years (median age 10 years); 43 were males. Of the 24 children with normal sized adenoids with no obstruction; 6 had severe OSA and 4 had moderate OSA. 3 had hypertrophied adenoids on X-ray with occlusion of nasopharynx but only had mild OSA. Of the 15 children with prominent adenoids with some narrowing but no occlusion; 9 had severe OSA. Of the 9 children that had hypertrophied adenoids with no occlusion; 3 had moderate OSA and 3 had severe OSA. Of the 12 with mild enlargement, 4 had severe OSA. None of the 19 children with severe OSA showed airway occlusion by adenoids.

Conclusion: In our study, the size of the adenoids on X-ray of the Nasopharynx did not correlate with severity of SDB in children. Airway obstruction in OSA is dynamic and can occur at multiple sites affected by anatomical factors and upper airway collapsibility. As use of Nasopharyngeal radiography involves exposure to radiation, it should be used judiciously in diagnosis of SDB in children. Polysomnography is the gold standard test for evaluation of SBD in children.

Support: None.

0902

THE USE OF AUTO-TITRATING CONTINUOUS POSITIVE AIRWAY PRESSURE (AUTO CPAP) FOR OBSTRUCTIVE SLEEP APNEA IN CHILDREN WITH NEUROLOGICAL DISORDER

Platter, L.¹ Urbano, G.² Roberto, L.³ Sudhakar, R.³ Tablizo, M.^{3,4} ¹Valley Children's Healthcare Department of Pediatrics, Madera, CA, ²Ateneo University Medical School, Manila, PHILIPPINES, ³Valley Children's Health Care Division of Pulmonary and Sleep, Madera, CA, ⁴Stanford University, Palo Alto, CA.

Introduction: Multiple studies have demonstrated the effectiveness of auto-titrating continuous positive airway pressure (auto CPAP) in the adult population, but there is limited literature on the use of auto CPAP in the pediatric population. Specifically, the use of auto CPAP in children with neurological disorder(s) has not been established. Thus, we conducted a study to review the use of auto CPAP in children ages 18 years old and younger with Obstructive Sleep Apnea Syndrome (OSAS) and associated neurological disorder to document its effectiveness, adverse events and outcomes of its use. **Methods:** A retrospective chart review was performed on patients 18 yo and younger diagnosed with OSAS and associated neurological disorder(s) who have good compliance with auto CPAP use. Good compliance was defined as >4 hours/night and >20/30 days of auto CPAP use. Compliance from the most recent 30 days was downloaded.

Results: 5 children met our criteria for inclusion, with a mean age of 11 years (6-18 years old). All had initial baseline sleep studies performed without PAP titration polysomnography. Associated neurological disorders were cerebral palsy, Arnold Chiari Malformation, seizure disorder and intellectual disability. The average length of use of auto CPAP was 4 months. Auto CPAP was used on average of 24/30 nights, with a mean of 7.35 hours/night. The mean baseline obstructive apnea-hypopnea (OAHI) index was 42 (8.2-94.4). The mean AHI on a 30 day download report showed a mean decrease in AHI to 2.9 (0.5-5.2) while on auto CPAP. Review of patient charts did not reveal any adverse outcomes associated with the use of auto CPAP in these patients.

Conclusion: This study showed that auto CPAP significantly improved the AHI in pediatric patients treated for OSA with associated neurological disorder. There were no reported adverse outcomes. Further research is needed to establish the effectiveness and safety of auto CPAP use in the pediatric population, specifically those with neurological disorder. The use of auto CPAP will help decrease the wait time for treatment in children with OSA. These patients can use auto CPAP while waiting for a titration study and for long term use.

Support: none

0903

SCREENING OF PEDIATRIC OBSTRUCTIVE SLEEP APNEA USING VIDEO MONITORING

Ikeda, K.¹ Yagi, T.² Chiba, S.²

¹Jikei University School of Medicine Daisan Hospital, Tokyo, JAPAN, ²Ota Memorial Sleep Center, Kanagawa, JAPAN.

Introduction: In Japan, the many of the patients are not able to access the specialized sleep medical facilities for overnight polysomnography(PSG) due to less availability and cost issues. Purpose of the study is to examine whether combination of video monitoring and other clinical examinations can reliably predict the severity of pediatric OSA compared with PSG.

Methods: Between April 1, 2012 and March 31, 2019, total of 175 children (3-12 years of age, boy 122, girl 53) with SDB were enrolled in this individual prospective-cohort study. In-laboratory based PSG were performed for all patients and sleep stages and respiratory events were manually scored. Video monitoring was performed during PSG. Modified video-recording test scoring system (based on Sivan et al 1996), were scored by laboratory technicians. Other clinical examinations were extracted from each PSG with ENT examinations, cephalogram, and rhinomanometry for all patient

Results: Multiple linear regression analyses was performed with a forward stepwise approach in which independent predictors that were significantly related to severity of OSA (AHI: 5/hr and 10/hr). Applying the multiple logistic regression analysis, the independent predictors for AHI 5/hr were ODI 3% >3/hr, rhinomanometry (NR>0.5 Pa/cm3/sec), enlargement of tonsils (Brodsky classification more than 2), two video monitoring items and total score, with an accuracy of predictive statistic model was 88.0% (sensitivity 78.3%, and specificity 93.0%). For the severity above AHI 10/hr, the independent predictors were Cephalogram parameter (Fx>84°), Oximetry (ODI 3% >5/hr) and BMI<15 with the video monitoring parameters of whole night inspiratory noise (loud) and chest retraction contribute to predict with the sensitivity 91.5%, the specificity 82.6% and the accuracy 88.0%.

Conclusion: Video monitor scoring parameters contributed to predict both AHI 5/hr and 10/hr with good overall sensitivity, specificity and overall accuracy compare with the combination of objective results alone. Instead of PSG, the combination of video scoring system and multiple clinical examinations could potentially provide reliable diagnostic approach for pediatric OSA with high accuracy. These results will support to establish more efficient diagnostic strategy for both patients and physicians Support: N/A

0904

RAPID EYE MOVEMENT RELATED OBSTRUCTIVE SLEEP APNEA IN THE PEDIATRIC POPULATION: CASE SERIES AND CONSIDERATIONS

Petitto, L.¹ Musso, M.²

¹Texas Children's Hospital, Houston, TX, ²Baylor College of Medicine, Houston, TX.

Introduction: Rapid Eye Movement (REM) related Obstructive Sleep Apnea (OSA) can impact sleep quality and result in negative clinical consequences. There are limited pediatric studies evaluating potential consequences, looking at optimal decision making, and discussing best treatment options. Aims: 1: Describe clinical impact and potential negative consequences; 2: Discuss treatment management of REM related OSA; 3: Evaluate clinical effectiveness of treatment.

Methods: Case series: Retrospective review of 22 pediatric patients with REM related OSA at a tertiary care center. Clinical analysis of implemented treatment modality vs. observation was reviewed. Symptomatic response to treatment modality vs. observation including alteration of quality of life was examined.

Results: REM related OSA lead to negative clinical daytime symptoms which warranted consideration and implementation of further treatment.

Conclusion: Treatment considerations for REM related OSA include tonsillectomy and adenoidectomy, positive airway pressure, medical management, and watchful waiting. Further research is necessary to increase knowledge of clinical impact of REM related OSA and treatment. Support: N/A

0905

OBSTRUCTIVE SLEEP APNEA IN PEDIATRIC PATIENTS WITH EARLY ONSET SCOLIOSIS

Panek, D.¹ MacKintosh, E.¹ DelRosso, L.¹ Ruth, C.² White, K.³ Redding, G.¹

¹Pediatric Pulmonary and Sleep Medicine, Seattle Children's Hospital, Seattle, WA, ²Diagnostic Sleep Disorders Center, Seattle Children's Hospital, Bellevue, WA, ³Orthopedic Surgery, Seattle Children's Hospital, Seattle, WA.

Introduction: Early onset scoliosis (EOS), defined as curvature of the spine >10 degrees with onset before 10 years of age, is associated with increased rates of restrictive lung disease, pain, and other factors that increase risk of poor sleep. We compared the polysomnographic findings of children with EOS to those of children without EOS. We postulated that children with EOS would have a higher rate of OSA than patients without EOS, and differences in sleep stage distribution, arousals, and limb movements. Methods: Single-center retrospective chart review performed on 58 subjects with EOS (ages 1-17yr) who underwent PSG from 2003-2019; comparison group of 58 subjects without scoliosis who underwent diagnostic PSG was chosen consecutively (ages 1-18yr). Polysomnographic parameters compared include: sleep stage distribution, arousal index (AI), obstructive/central AHI, mean and nadir oxygen saturation in REM/NREM, and periodic leg movement index. All p-values were adjusted for multiple comparisons.

Results: There was no difference in age or sex distribution between the two groups, though subjects with EOS had lower BMI than those without EOS (median 16.3 (IQR 14.7-19.3) vs. 17.5 (IQR 16.2-21.6), p=0.019). 84% of subjects with EOS had OSA, compared to 66% without EOS. Subjects with EOS and OSA had higher obstructive AHI than the OSA group without EOS, and longer duration of hypopneas. There was no significant difference in sleep stage distribution, AI, or PLMI.

Conclusion: Of pediatric patients referred for polysomnography at our institution, those with EOS had a higher rate of OSA, more severe OSA where present, and lower BMI. We advocate for routine polysomnography for children with EOS due to the high risk of OSA amongst those tested, and further study to better understand the pathophysiology of sleep disordered breathing in this population.

Support: This project is supported by the Health Resources and Services Administration (HRSA) of the US Department of Health and Human Services under grant #T72MC00007/University of Washington Pediatric Pulmonary Center/PI: Redding. The content and conclusions are those of the author and should not be construed as the official position or policy of, nor should any endorsements be inferred by HRSA, HHS or the U.S. Government.

0906

IDENTIFICATION OF PHYSICAL EXAM FINDINGS WITH HIGH PREDICTIVE VALUE FOR MODERATE TO SEVERE PEDIATRIC OBSTRUCTIVE SLEEP APNEA(OSA) IN **OVERWEIGHT/OBESE CHILDREN**

Ajisebutu, A.¹ Kak, I.² Thompson, N.³ Honomichl, R.³ Moul, D.¹ Mehra, R.¹ Shah, V.⁴

¹Sleep Disorders Center, Neurological Institute, Cleveland Clinic, Cleveland, OH, ²Department of Pediatrics, Cleveland Clinic, Cleveland, OH, ³Section of Biostatistics, Quantitative Health Sciences, Cleveland Clinic, Cleveland, OH, ⁴Sleep Disorders Center, Neurological Institute, Cleveland Clinic, Cleveland, OH.

Introduction: Obstructive sleep apnea(OSA) is highly prevalent and under-diagnosed in the overweight/obese pediatric population largely due to limitations of existing pediatric OSA screening instruments including lack of efficiency and practical implementation and lack of careful consideration of physical examination(PE) findings with high predictive value for OSA. We sought to identify PE finding(s) predictive of pediatric OSA in overweight/obese patients to inform development of an OSA screening tool.

Methods: Overweight/obese patients presenting to the Cleveland Clinic weight-management clinic between 2013-2018 with polysomnogram (PSG) data were included. The association of PE predictors: age, sex, race (white, black, other), neck (NC), waist circumference (WC), tonsil size (TS), height, systolic and diastolic blood pressure (BP) percentiles) in relation to OSA defined by apnea-hypopnea index (AHI) \geq 5,i.e. clinically significant pediatric OSA, were assessed using univariate and multivariate logistic regression models (OR,95%CI).

Results: Retrospective analysis of 180 overweight/obese patients (BMI percentile>85th for age and sex) and age 12.5 \pm 3.7 years were included. The multivariate model showed that only WC was significantly associated (1.03, 1.00 - 1.07, p=0.038) with OSA defined as AHI≥5. A statistically significant interaction of age and sex was observed such that the likelihood of OSA increased in males with older age and conversely decreased in females with older age. (1.26,1.04 -1.52, p=0.038) The reduced multivariate model, which included age, sex, WC, and age*sex interaction term, correctly discriminated AHI <5 vs. ≥ 5 66.5% of the time.

Conclusion: In this large clinic-based overweight/obese pediatric sample, males, older age and WC were significant predictors of OSA and TS was not. A significant interaction of age and sex was observed supporting increased OSA with increasing age in males. Data generated supports value of PE findings of age, sex and WC to incorporate in development of an OSA screening tool for overweight/obese children. **Support:**

0907

IMPLEMENTATION OF PATHWAY FOR SEVERE OBSTRUCTIVE SLEEP APNEA MANAGEMENT IN CHILDREN

Gehring, S.¹ *Auricchio,* L.² *Kidwell,* S.¹ *Oppy,* K.¹ *Smallwood,* S.¹ *Kalra,* M.¹

¹Division of Sleep Medicine, Dayton Children's Hospital, Dayton, OH, ²Clinical Excellence, Dayton Children's Hospital, Dayton, OH.

Introduction: Obstructive Sleep Apnea (OSA) is associated with neuro-cognitive, cardiovascular and metabolic morbidity in children. Adeno-tonsillectomy is the first line of treatment for OSA with PAP therapy and Oxygen supplementation being alternative therapeutic options in select cases. Severe Obstructive Sleep Apnea is a known risk factor for postoperative respiratory complications after adenotonsillectomy. Therefore, inpatient adenotonsillectomy with close monitoring is recommended for this group of children. Challenges to safe and timely care for this high risk group of children can be overcome with effective coordination of care between different locations and health care providers.

Methods: All children seeking treatment at Dayton Children's Division of Sleep Medicine were managed through a pathway developed by a multi-disciplinary team involving sleep medicine, oto-laryngology and clinical logistics. Severe OSA was defined as AHI \geq 15 events/hr (children <2 year old), AHI \geq 15 events/hr with three or more Oxygen desaturations <80% (children \geq 2 to <6 years old),

AHI \geq 30 events/hr with three or more Oxygen desaturations <80% (Children \geq 6 to 18 years old).

Results: A total of 78 children were diagnosed with severe OSA in 2019. All children were successfully triaged to appropriate therapeutic option (Adenonotonsillectomy, PAP, O2) within 24 hours of diagnosis. Urgent adenotonsillectomy was performed on the same day in 4 children and within 2 weeks on 12 children. There was no postoperative respiratory complication after urgent adenotonsillectomy. Thirteen children had adenotonsillectomy after 2 weeks. PAP therapy was started in 28 children (34%). Therapy was initiated on the same day in 10 children and the next day on one child. Oxygen supplementation was started in 21 children (27%).

Conclusion: A multidisciplinary collaborative approach can result in delivery of timely and safe care for severe OSA in children. **Support:** NA

0908

THE IMPACT OF ADENOTONSILLECTOMY ON OBSTRUCTIVE SLEEP APNEA IN CHILDREN WITH TRISOMY 21: A RETROSPECTIVE COHORT STUDY

Platt, J.¹ Adeleye, A.¹ Nettel-Aguirre, A.¹ Chugh, A.¹ Yunker, W.³ ¹University of Calgary, Department of Pediatrics, Calgary, AB, CANADA, ²University of Calgary, Department of Pediatrics, Calgary, AB, CANADA, ³University of Calgary, Department of Surgery, Calgary, AB, CANADA.

Introduction: Obstructive sleep apnea (OSA) has a prevalence of 1 - 5.7% in the general pediatric population. In children with Trisomy 21 (T21), OSA is estimated to be much higher, at 30-60%. The medical consequences of untreated OSA may be significant, therefore it is worthwhile to explore treatment options. Adenotonsillectomy (AT) is often the first line treatment in children, including those with T21. However, success rates of AT in patients with T21 is more variable, and postoperative complications can be higher. The aim of this study was to determine the impact of AT on the apnea hypopnea index (AHI) in patients with Trisomy 21.

Methods: A retrospective cohort study was conducted by reviewing children with T21 and the following criteria: 1) 0 to 18 years of age, 2) AT completed between January 2010 to December 2015, 3) pre- and post-operative polysomnogram (PSG). Data extraction included demographics, details of PSG both prior to and after surgery including severity of sleep apnea and oxygen levels, type of surgery, and surgical complications.

Results: Our sample consisted of 64 subjects. Mean pre-operative AHI was 32.2, while mean post-operative AHI was 8.0, for a difference of 21.7 (p = 0.0001). Mean pre-operative oxygen saturation was 92.5, while mean post-operative oxygen saturation was 93.7, for a difference of 1.2 (p = 0.01). There were 10 post-operative emergency room visits (15.3%), 2 admissions to hospital (3.1%) and 2 repeat surgeries for post-operative bleeding (3.1%).

Conclusion: Preliminary findings of this study show a statistically significant improvement in OSA severity as determined by change in AHI and mean oxygen saturations post AT in children with T21. Complication rates were low. Further data collection and analysis is underway. **Support:** None.

0909

SLEEP DURAION AND RELATED FACTORS AMONGCHINESE CHILDREN: ACROSS-SECTIONAL STUDYABSTRACT

Jun, T.

beijing children's hospital, beijing, CHINA.

Introduction: It is known short sleep duration adversely affects children's behavior and physical development. Sleep duration and sleep habit vary substantially in children with different ages, areas and races. However, our understanding on the sleep duration in Chinese children remains limited. The present study investigated the status of sleep durations in Chinese children and explored factors related to sleep loss.

Methods: A randomized, stratified, multi-stage cluster sampling method encompassed 11420 children in 25 schools from 7 districts in Beijing. Children aged 3 to 14 years were included, and their parents were invited to fill sleep habit related questionnaires about the performance of the last 3 months.

Results: The final cohort included 4736 boys and 4462 girls with a mean age of 8.8 ± 3.8 years. The mean sleep duration of children ≥ 12 years of age was 9.4h, which was significantly lower than those < 12 years old (9.7h); this was in agreement with the trend of sleep durations of weekdays. On weekends and holidays, the sleep duration was approximately 10h for most children and similar in all age groups. Gender (male), age (≥ 12 -year group), overweight, and suburban residence were significantly correlated with sleep loss in children (p< 0.001). A significantly high proportion of children with sleep loss displayed sleepiness during sitting and reading (19.1%), in the car (31.4%), in the afternoon (20.7%), and after lunch (17.5%).

Conclusion: The sleep duration reduced significantly in children ≥ 12 years of age as compared to younger children in Beijing. Factors such as gender (male), age (≥ 12 -year group), overweight, and suburban residence are related to sleep loss. Children ≥ 12 years of age older with sleep loss were likely to experience daytime sleepiness.

Support: This work was supported by Beijing Municipal Science and Technology Project grant (Z161100000116050 and Z161100003216212) and Beijing Municipal Administration of Hospitals Clinical Technology Innovation Project grant (XMLX201701).

0910

PREVALENCE AND RISK FACTORS OF OBSTRUCTIVE SLEEP APNEA SYNDROME IN 3-14 YEARSOLD CHILDREN IN BEIJING: A CROSS-SECTIONAL SURVEYABSTRACT

Wei, W.

beijing children's hospital, beijing, CHINA.

Introduction: To explore the prevalence and risk factors of obstructive sleep apnea syndrome (OSAS) in 3-14 years old children in Beijing.

Methods: A cross-sectional study of random stratified cluster sampling was conducted on 3-14 years old children and adolescents in Beijing. The preliminary screening was completed through Pediatric Sleep Questionnaire (PSQ) investigation and the diagnosis of OSAS was conducted by polysomnography.

Results: After random sampling, a total of 11 kindergartens, 7 primary schools and 8 junior high schools from 7 districts of Beijing were involved in the survey. A total of 11420 questionnaires were sent out, and 10743 (94.07%) were recovered. The final effective data were 9198 (effective response rate 85.62%). The age of the investigated children was 8.8 ± 3.8 years. PSQ positive rate was 8.86% (95CI 8.28-9.44%). With diagnostic criteria AHI≥5, the estimated prevalence of OSAS in Beijing children was 5.90% (95%CI 3.72-8.28%); with ICSD-3 diagnostic criteria (OAHI>1), the estimated prevalence was 8.08% (95% CI 5.74-10.62%). Overweight (OR=3.13), frequent allergic rhinitis (OR=6.80) and family snoring history (OR=6.14) were important risk factors for children's OSAS.

Conclusion: PSQ was used in Beijing children's OSAS epidemiological survey with good reliability and validity. The positive rate of the PSQ screening was 8.86% (95CI 8.28-9.44%); the estimated prevalence of OSAS in children aged 3-14 years in Beijing was 5.90% (95%CI 3.72-8.28%) with criteria AHI≥5 and 8.08% (95%CI 5.74-10.62%) with criteria OAHI>1, respectively. Gender, BMI, history of ENT and family history of snoring were important risk factors for children's OSAS.

Support: This work was supported by Beijing Municipal Science and Technology Project grant (Z161100000116050 and Z161100003216212) and Beijing Municipal Administration of Hospitals Clinical Technology Innovation Project grant (XMLX201701).

0911

ASSOCIATION BETWEEN CHROMOSOMAL ABNORMALITY AND SLEEP DISORDERED BREATHING CHARACTERISTICS IN CHILDREN WITH PRADER WILLI SYNDROME

Gupta, G. Veeravigrom, M. Felt, B. O'Brien, L. University of Michigan, Ann Arbor, MI.

Introduction: Prader-Willi Syndrome (PWS) is a genetic disorder caused by lack of expression of paternal genes from the chromosomal region 15 q11.2-q13. PWS is associated with excessive daytime sleepiness, sleep-disordered breathing (SDB), hypoventilation and circadian rhythm disturbance. Chromosomal deletion and uniparental disomy (UPD) are the two most common genetic etiologies of PWS. Differences in clinical features between these groups are recognized; however, limited literature exists regarding sleep characteristics.

Methods: The objective was to evaluate sleep characteristics of children with PWS between those with 15q11.2-q13 chromosomal deletion and those with other genetic etiologies. A retrospective chart review of in-laboratory polysomnograms (PSG) in children with PWS prior to growth hormone treatment was performed. The apnea hypopnea index (AHI), REM AHI, NREM AHI, Obstructive Apnea Index (OAI), Central Apnea Index (CAI), hypopnea index, arousal index, presence of hypoventilation, and positional AHI were assessed.

Results: Overall 33 PWS children were identified. Mean age was 5.9 ± 5.19 (range 1-16 years), 57% were male and 85% had SDB. Almost half (42%) had a 15q11.2-q13 chromosomal deletion. Of those without deletion, 30% had UPD. Sleep variables were similar in PWS with and without deletion: AHI (5.9 \pm 5.7 vs. 8.54 \pm 7.1; p=0.27), NREM AHI (3.3 \pm 4.6 vs. 6.2 \pm 6.3; p=0.16), CAI (1.1 \pm 1.5 vs. 1.9 \pm 3.8; p=0.43) SpO2 nadir 82.7 \pm 9.9% vs. 85.3 \pm 5.7%; p=0.36) and arousal index (10.3 \pm 7.8 vs. 15.4 \pm 7.8; p=0.09).

Conclusion: Sleep characteristics do not appear to clearly differ between genetic etiologies of PWS. Larger sample sizes are needed to support these findings.

Support:

0912

CENTRAL SLEEP APNEA PATTERNS ON PEDIATRIC POLYSOMNOGRAMS

Rao, H. S.

Penn State Hershey Children's Hospital, HERSHEY, PA.

Introduction: Central sleep apneas (CA) are frequently seen on pediatric polysomnograms (PPSG) independently or in conjunction with obstructive sleep apnea (OSA). In the pediatric population, the AASM defines CA as the absence of chest and/or

abdominal movement associated with a cessation of airflow of more than 20s or longer than 2 baseline respiratory cycles if associated with an arousal, an awakening or oxygen desaturation $\ge 3\%$. Scoring CAs on PPSG based on AASM definition can cause confusion among providers as CAs are generally associated with central nervous disorders causing reduced or absent respiratory drive.

Methods: Retrospective review of 71 consecutive diagnostic PPSGs to analyze patterns of CAs scored per AASM definition was performed. None of the children had a disorder causing reduced respiratory drive. Data on age, obstructive AHI (Apnea Hypopnea Index), CO2, Oxygen saturation, Central AHI and diagnosis were collected.

Results: 68 of 71 children had varying degree of OSA and CAs. Three main patterns of CAs were observed: occurring in NREM, following sigh breaths or arousals and CAs seen in REM sleep. CO2 and oxygen saturation were in the normal range.

Conclusion: In our study, CAs were more often seen in young children related to reduced functional residual capacity and immaturity of chest wall. CAs in REM sleep was seen more often in children with lung disorders and gastroesophageal reflux. CAs associated with arousals/awakenings were seen in conjunction with OSA or periodic limb movement disorder (PLMD). Though a finding of CAs >5/hour is considered significant, the minimum number of events required to cause a specific disorder or syndrome remains elusive and may be different in different patient populations. As such, there is no threshold of the number of central apneas associated with disease. CAs associated with disorders causing reduced or absent respiratory drive are mostly seen in NREM sleep and associated with abnormal gas exchange. The context in which the CAs are seen on PPSGs should be clearly described to avoid confusion among ordering providers. In CAs associated with arousals/ awakenings, it is important to target the cause of arousals such as OSA or PLMD.

Support: None

0913

AMBULATORY BLOOD PRESSURE MONITORING IN CHILDREN WITH OBSTRUCTIVE SLEEP APNEA SYNDROME

Geng, X.¹ Wu, Y.² Ge, W.³ Feng, G.⁴ Zheng, L.³ Xu, Z.⁵ Ni, X.³ ¹Capital Medical University, Beijing, China, Beijing, CHINA, ²Beijing Key Laboratory of Pediatric Otolaryngology, Head & Neck Surgery, Beijing Pediatric Research Institute, Beijing Children's Hospital, Capital Medical University, National Center for Children's Health, Beijing, China, BeiJing, CHINA, ³Department of Otolaryngology, Head & Neck Surgery, Beijing Children's Hospital, Capital Medical University, National Center for Children's Health, Beijing, China, BeiJing, CHINA, ⁴Research Center for Big Data and Engineering, Beijing Children's Hospital, Capital Medical University, National Center for Children's Health, Beijing, China, BeiJing, CHINA, ⁵Department of Respiratory Medicine, Beijing Children's Hospital, Capital Medical University, National Center for Children's Health, Beijing, China, BeiJing, CHINA, ⁵Department of Respiratory Medicine, Beijing Children's Hospital, Capital Medical University, National Center for Children's Health, Beijing, China, BeiJing, CHINA.

Introduction: This study was performed to investigate the differences in blood pressure among different groups of snoring children and among different sleep stages. In recent years, the incidence of OSAS in children has increased year by year. Blood pressure research of OSAS children can better understand the occurrence of OSAS related complications. Early detection and intervention of blood pressure changes in children with OSAS can effectively reduce the incidence of cardiovascular disease in adulthood and lower the disease burden.

Methods: Habitually snoring children (snoring frequency of ≥ 3 nights per week) aged 3to 11 years were recruited from Beijing Children's Hospital, Capital Medical University from 1 January 2017 to 30 June 2018. All children underwent polysomnography, and their blood pressure was monitored and calculated by the pulse transit time. The children were divided into those with primary snoring (PS), mild obstructive sleep apnea syndrome (OSAS), and moderate to severe OSAS according to their obstructive apnea-hypopnea index (OAHI).

Results: In total, 140 children were recruited. Ninety-seven had PS, 24 had mild OSAS, and 19 had moderate to severe OSAS. There were no differences in age, sex, or body mass index z-score among the groups. Statistically significant differences were found in the OAHI, oxygen desaturation index 3%, respiratory arousal index, and lowest oxygen saturation among the three groups. Children with moderate to severe OSAS had higher systolic and diastolic blood pressure than those with mild OSAS and PS (P < 0.001). In all children, systolic and diastolic blood pressure was higher in the rapid eye movement (REM) sleep stage than in the non-REM sleep stage (P < 0.05).

Conclusion: Children with moderate to severe OSAS had higher blood pressure than those with PS and mild OSAS. Blood pressure in the REM sleep stage was higher than that in other sleep stages in all groups of children.

Support: The Pediatric Medical Coordinated Development Center of Beijing Hospitals Authority (XTYB201807);Capital Health Research and Development of Special Funding (2018-1-2091);National Key Research and Development Plan (2017YFC0112502)

0914

SLEEP DISORDERED BREATHING IN PEDIATRIC PATIENTS BEFORE AND AFTER INTRATHECAL BACLOFEN THERAPY

Sobremonte-King, M. Chen, M. DelRosso, L. M. Seattle Children's Hospital, Seattle, WA.

Introduction: Refractory spasticity in children is treated with intrathecal baclofen (ITB), which may worsen both central and obstructive breathing events. Sleep disordered breathing (SDB) is seldom investigated prior and/or subsequent to placement of ITB and there are currently no standardized protocols. This study aims to compare occurrence of SDB pre/post ITB placement.

Methods: Retrospective chart review revealed 104 patients started on ITB therapy from 2009-2019 and those who had pre and/or post ITB polysomnograms (PSG) were included. Medical history and PSG parameters were extracted. Comparison of paired results will occur using the Wilcoxon Signed Rank Sum Tests once collection is complete. **Results:** Thirty-seven patients were identified having pre and/or post ITB PSGs. Results in mean \pm SD show: age was 11 ± 4 years and 65% were male. Twenty-five pre ITB PSG had an oAHI of 4 ± 5 with 22/25 (88%) having SDB. There were 15/25 (60%) with mild OSA (oAHI >1 but < 5) and 7/25 (28%) with moderate-severe OSA (oAHI > 5/hr). CAI was 1 ± 2 and oxygen saturation nadir was 88 ± 9 %. Sixteen post ITB PSG had an oAHI of 8 ± 13 with 100% having SDB. There were 11/16 (69%) with mild OSA and 5/16 (31%) with moderate-severe OSA. CAI was 3 ± 7 and oxygen saturation nadir was 84 \pm 8 %. Ten patients were initiated on non-invasive ventilation, one on supplemental oxygen and two had adenotonsillectomy.

Conclusion: Initial data shows high occurrence of SDB in patients pre and post ITB placement leading to medical or surgical intervention in 35%. Post ITB PSGs showed worsened oAHI and CAI and lower oxygen saturation nadir. Possible mechanisms include depression of central respiratory drive and decreased pulmonary reserves. This study may help stratify and address risks of ITB for those with refractory spasticity and SDB. **Support:** None

0915 SLEEP PATTERNS AND THE EFFECT OF LATE BEDTIME ON SCHOOL-AGE CHILDREN AND ADOLESCENTS: PRELIMINARY RESULTS

Matsangas, P.¹ Gratsia, S.² Cocos, A.² Vastardis, H.² Shattuck, N. L.¹

¹Human Systems Integration Program, Operations Research Department, Naval Postgraduate School, Monterey, CA, ²Orthodontics Department, School of Dentistry, National and Kapodistrian University, Athens, GREECE.

Introduction: School-age children (6-13yrs) and teenagers (14-17yrs) should receive 9-11hrs and 8-10hrs of sleep/day, respectively. Several studies have shown, however, that these age groups are chronically sleep deprived. Our study assessed the sleep patterns of a sample of children and teenagers in Athens, Greece. The study is part of a larger project investigating the association between ortho-dontic treatment and sleep disturbances.

Methods: Participants (N=27; 69% females; 21 school-age children 9-13yrs, 6 teenagers 14-17yrs) were under treatment in the Orthodontic Clinic of the National and Kapodistrian University. Sleep was assessed with actigraphy/logs for 59 ± 19 days.

Results: Participants slept on average 7.36±0.42hrs/day. Nighttime sleep was on average 7.23±0.43hrs (percentage sleep: 87.3%±3.38%). Four (14.8%) participants napped at least once/ week. Compared to the lowest sleep duration recommended for their age group, participants showed a chronic sleep deficit of 1.42±0.52hrs/day (range: 0.32-2.15hrs). The younger age group had an average sleep deficit of ~1.6hrs compared to ~0.8hrs for the teenagers (p=0.006). During the school year, daily sleep duration increased by ~0.73hrs on weekends (7.78±0.67hrs) compared to school nights (7.05±0.48hrs; p<0.001). On average, school-age participants slept from 23:13 (±31min) until 7:19 (±22min) on school nights and from 23:23 (±2:72hrs) until 8:49 (±39min) on weekends. Teenagers slept from 00:34 (±36min) until 7:40 (±14min) on school nights and from 01:34 (±41min) until 10:34 (±48min) on weekends. Conclusion: Our findings verify earlier survey results showing that restricted sleep is a problem for children and adolescents in Greece. To our surprise, both age groups go to bed quite late. The impact of late bedtime on sleep duration, however, is larger in the younger group due to their larger sleep needs. In contrast to earlier research in rural areas, napping was not common in our urban sample, probably due to extracurricular activities and studying at home. Support: N/A

0916

THE USE OF IMMERSIVE VIRTUAL REALITY AND SLOW BREATHING TO ENHANCE RELAXATION AND SLEEP IN ADOLESCENTS

Yüksel, D. Goldstone, A. Prouty, D. Forouzanfar, M. Claudatos, S. Lee, Q. Wang, R. Dulai, T. Arra, N. Volpe, L. Durley, I. Baker, F. de Zambotti, M.

SRI International, Menlo Park, CA.

Introduction: Sleep disturbances frequently emerge during adolescence amongst profound, normative, sleep maturation and biopsychosocial changes. Factors like stress, worry or rumination may make falling asleep and maintaining sleep more difficult. Here, we evaluate the efficacy of a novel intervention based on virtual reality (VR) and slow breathing to promote bedtime relaxation and facilitate sleep in high-school adolescents.

Methods: Twenty-nine 16-18 year-old adolescents with (N=9, 6 girls) and without (N=20, 11 girls) sleep difficulties underwent two counterbalanced in-lab relaxation and baseline polysomnography (PSG) nights. For the relaxation condition, immediately preceding bedtime, participants were engaged in slow diaphragmatic breathing (to promote physiological downregulation) whilst passively experiencing a relaxation immersive VR environment, designed to promote cognitive relaxation/distraction (20min). On the baseline night, participants engaged in quiet activities (e.g., reading a book) before bedtime (20min).

Results: The VR intervention resulted in a significant immediate increase in perceived relaxation and reduced worry (p<0.05). Also, heart rate dropped (~5bpm) in the pre-to-post intervention (p<0.05), while no significant change in heart rate was evident before and after the time spent in quiet activities on the baseline night. PSG-defined sleep onset latency was shorter (~6min reduction) and sleep efficiency was greater (~3% increase) on the VR relaxation night compared to the baseline night (p<0.05). In addition, baseline sleep onset latency was related to the magnitude of the baseline-to-relaxation reduction in sleep onset latency in participants (R²=0.70; p<0.01). There was no apparent difference in responses to the VR intervention between adolescents with or without insomnia.

Conclusion: Our data highlight the potential for combining cognitive relaxation/distraction strategies, using immersive VR technology and physiological downregulation, to promote bedtime relaxation and improve overall sleep quality in adolescents. Further research is needed to evaluate the feasibility and effectiveness of such interventions over time.

Support: National Heart, Lung and Blood Institute (NHLBI) R01HL139652 (to MdZ)

0917

DESIGNING A WEARABLE TECHNOLOGY-BASED SLEEP INTERVENTION TO SUPPORT SLEEP HEALTH AMONG ADOLESCENTS: USING A PARTICIPATORY DESIGN APPROACH

Beck, A. J.¹ Duffett-Leger, L.¹ Raffin Bouchal, S.¹ Ferber, R.^{1,2} Ward, T.³

¹Faculty of Nursing, University of Calgary, Calgary, AB, CANADA, ²Faculty of Kinesiology, University of Calgary, Calgary, AB, CANADA, ³University of Washington, School of Nursing, Seattle, WA.

Introduction: Sleep problems during adolescence are increasingly common and have been associated with adverse physical and psychological health outcomes. Efforts to improve insufficient sleep among adolescents have resulted in increased sleep knowledge and temporary enhancements in sleep hygiene. Good sleep hygiene is established through the development of daily routines that support healthy sleep. Wearable technology offers a potential solution whereby adolescents can acquire and manage healthy sleep habits. In this study, we are co-designing with adolescents a prototype intervention using wearable technology to promote sustained improvements in their sleep hygiene.

SLEEP, Volume 43, Abstract Supplement, 2020

Methods: Guided by participatory design approaches, the ongoing multi-phase mixed methods study is currently being conducted in a metropolitan area in western Canada. In phase 1, sleep data is being collected from a sample of 30 adolescent-parent dyads using wearable sensors (Actigraphy watches) and self-report sleep measures (questionnaires about sleep quality, hygiene, and beliefs and attitudes, as well as their general health) over a 10-day period. In phases 2 and 3, individual interviews and iterative user interface design sessions will be conducted with 25 adolescents.

Results: To date, thirteen adolescents-parent dyads (13-17 years, 9 females; 39-56 years, 11 females) have completed phase 1 of our study. Data analysis is currently being conducted to evaluate sleep onset/offset, total sleep time, wake after sleep onset, sleep efficiency, and sleep schedule differences between adolescents and their parents. Ten adolescents have completed individual interviews in phase 2 of the study. Preliminary qualitative data suggests that youth are aware of the importance of sleep to their overall health. However, they struggle with identifying credible information to act on from the various and sometimes conflicting sources (e.g. online, friends, family).

Conclusion: We anticipate that co-designing a wearable solution with adolescents will lead to a sleep intervention that is more relevant, persuasive, and useful in supporting their sleep health.

Support: This work is supported by the Sensor Technology in Monitoring Movement STiMM Program.

0918

QUEBEC ADOLESCENTS' INSOMNIA SYMPTOMS, MENTAL HEALTH & ACCESS TO SLEEP CARE

Gruber, R.¹ Somerville, G.² Finn, C.³ Boursier, J.⁴ ¹McGill Univesrity, Montreal, QC, CANADA, ²Riverside School Board, Montreal, QC, CANADA, ³L B Pearson School Board, Montreal, QC, CANADA, ⁴Heritage Regional Highschool, Saint Hubert, QC, CANADA.

Introduction: Our school boards are part of the Quebec Center Of Excellence For Mental Health, and are thus mandated to identify and then prevent/treat factors that could negatively affect the mental health of their students. As part of this mission, we conducted a study that aimed 1) to examine the prevalence of insomnia in typically developing students in Quebec and their associations with students' mental health, and; 2) to assess student access to behavioral sleep interventions in Quebec.

Methods: 145 (Age 15.53+1; 75 Girls, 70 Boys) typically developing students. *Insomnia symptoms* were measured using the Insomnia Severity Index. *Sleep* was measured by Actigraphy. The Youth Self Report was used to measure students mental health. Access to care was measured using a detailed questionnaire.

Results: The key findings were: 1) 45% of the students reported poor sleep quality, dysfunction during the day due to sleepiness, difficulty falling or staying asleep, and excessive daytime sleepiness. Total scores on self-reports sleep measures were positively significantly correlated with Actigraphy measures of sleep duration and quality and with each other; 2) Robust positive associations were found between insomnia and psychiatric symptoms after adjusting for common risk factors, including age, socioeconomic status, and gender; 3) None of the students had access to sleep care and 25% of them reported using over-the counter sleep aids with minimal success.

Conclusion: These findings are alarming because they show that: 1) insomnia symptoms are prevalent in Quebec students and are strongly associated with the symptomatology of mental distress in

students who do not meet the diagnostic criteria of a psychological disorder, and; 2) these students do not have access to insomnia care. These findings suggest that treatment of insomnia could offer an incredible opportunity to protect and improve the sleep and the mental health of these students.

Support: Canadian Institute of Health Research grant to Reut Gruber

0919

HEALTH DISPARITIES IN THE PERSISTENCE OF CHILDHOOD INSOMNIA SYMPTOMS IN THE TRANSITION TO ADOLESCENCE: THE PENN STATE CHILD COHORT

Bourchtein, E.¹ Puzino, K.¹ Calhoun, S. L.¹ Criley, C.¹ He, F.¹ Vgontzas, A. N.¹ Liao, D.¹ Bixler, E. O.¹ Fernandez-Mendoza, J.¹ ¹Penn State College of Medicine, Hershey, PA, ²Penn State College of Medicine, Hershey, PA.

Introduction: A strong body of cross-sectional evidence indicates that social determinants of health (SDH), such as race, ethnicity, socioeconomic status, and sex/gender, are linked to sleep problems, including insomnia symptoms. Few studies have examined the longitudinal association between SDH and the persistence and remission of insomnia symptoms in the transition between childhood and adolescence, a critical period for sleep health.

Methods: The Penn State Child Cohort is a random, populationbased sample of 700 children (5-12y at baseline), of whom 421 were followed up as adolescents (12-23y at follow-up). All subjects underwent polysomnography, clinical history, physical exam, and parent- and self-reported scales at baseline and follow-up. Childhood insomnia symptoms were defined as a parent- and/or self-report of difficulty falling and/or staying asleep. All subjects or their parents identified the subject's sex, race, and ethnicity, and reported on socioeconomic status (SES) of the household.

Results: Females (32.7%) and racial/ethnic minorities (25.0%) were associated with a significantly lower remission rate as compared to males (53.3%) and non-Hispanic whites (48.3%), respectively. Non-Hispanic whites of low SES were associated with a significantly lower full remission rate (26.3%) as compared to non-Hispanic whites of higher SES (42.0%), while racial/ethnic minorities were associated with the lowest full remission rates regardless of whether they were of low (9.1%) or higher (11.1%) SES.

Conclusion: Our novel data indicate that gender-, racial/ethnicand socioeconomic-related disparities in insomnia not only occur as early as childhood but are important determinants of insomnia's chronic course throughout development.

Support: National Institutes of Health (R01HL136587, R01HL97165, R01HL63772, UL1TR000127)

0920

BEHAVIORAL PROFILES ASSOCIATED WITH THE DEVELOPMENT OF INSOMNIA SYMPTOMS IN CHILDREN WITH KNOWN MENTAL HEALTH DISORDERS

Bourchtein, E.¹ Calhoun, S. L.¹ Puzino, K.¹ McQuillen, A.¹ He, F.¹ Vgontzas, A. N.¹ Liao, D.¹ Bixler, E. O.¹ Fernandez-Mendoza, J.¹ ¹Penn State College of Medicine, Hershey, PA, ²Penn State College of Medicine, Hershey, PA.

Introduction: Mental health disorders (MHD) are a known risk factor for the development of insomnia symptoms in youth. However, a number of children with MHD do not go on to develop

insomnia symptoms later on in life. Little is known about possible childhood factors that exacerbate or mitigate the risk of developing adolescent insomnia symptoms (AIS) among children with MHD. The present study examined, in an at-risk group of children with MHD, the behavioral profiles associated with the development of AIS.

Methods: The Penn State Child Cohort is a random, populationbased sample of 700 children (5-12y), of whom 421 were followed up as adolescents (12-23y). Absence of childhood insomnia symptoms was ascertained by parent-reports (n=312), while presence of AIS in this subgroup was ascertained by self-reports (n=97). Presence of MHD was ascertained based on the clinical history and physical exam at baseline (n=52). The Pediatric Behavior Scale (PBS) assessed multiple parent-reported behavioral domains.

Results: Children with MHD at baseline had greater levels of difficulty across a variety of internalizing (e.g., anxiety, depression) and externalizing (e.g., impulsivity, hyperactivity) behavioral domains than those without MHD, regardless of whether they developed AIS. However, children with MHD who went on to develop AIS had significantly greater levels of aggressive (p<0.001) and oppositional (p=0.006) behaviors relative to children with MHD who did not develop AIS. In fact, these latter children did not differ from peers without any history of MHD or AIS on levels of aggressive (p=0.820) or oppositional (p=0.436) behaviors.

Conclusion: Children with MHD who present with normative aggressive and oppositional behaviors are less likely to develop AIS. Healthcare providers should consider providing preventative sleep interventions to youth with MHD who are exhibiting comorbid externalizing behaviors.

Support: National Institutes of Health (R01HL136587, R01HL97165, R01HL63772, UL1TR000127)

0921

INSOMNIA IN ADOLESCENTS: PATIENT-CENTERED OUTCOMES AND PERSPECTIVES

Honaker, S. M.¹ Simon, S. L.² Byars, K. C.³ Graef, D. M.³ Williamson, A. A.⁴ Meltzer, L.⁵

¹Indiana University School of Medicine, Department of Pediatrics, Indianapolis, IN, ²University of Colorado Anschutz Medical Campus, Aurora, CO, ³Cincinnati Children's Hospital Medical Center, Cincinnati, OH, ⁴Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, ⁵National Jewish Health, Denver, CO.

Introduction: An estimated 25-40% of adolescents endorse symptoms of insomnia. While research has demonstrated that insomnia is associated with impaired functioning, little is known regarding which insomnia symptoms or outcomes are most burdensome for adolescents. *Patient-centered outcomes* (PCO) are clinical and research questions and outcomes that are meaningful to patients. This study is the first to evaluate PCOs in adolescents with insomnia.

Methods: We conducted an online survey of adolescents in the USA, UK, Canada, Australia, and New Zealand who were 13 - 18 years old and reported experiencing one or more insomnia symptoms. Participants were recruited using targeted advertising on Facebook. Participants reported on insomnia symptom severity, frequency, and duration, help-seeking behavior, areas of insomnia-related impairment, and research priorities.

Results: Of the N=3034 respondents, 99.3% (n=3014) met inclusion criteria. Participants were predominantly female (77.0%) and white non-Hispanic (70.8%), with a mean age of 16.1 years (SD=1.3). Most (87.5%) met DSM-V diagnostic criteria for

insomnia, yet only 29.3% reported seeking professional help. Over half (52.1%) reported a symptom duration of three or more years. Insomnia symptoms reported were sleep initiation difficulties (94.4%), sleep fragmentation (65.3%), premature awakening (54.5%), and difficulty sleeping independently (22.3%). The most burdensome areas of insomnia-related impairment were reported to be mood (72.2%), attentional focus (61.0%), pain (49.7%), worry (46.3%) and sleepiness (38.7%). Adolescents with insomnia most frequently endorsed the following research priorities: causes of insomnia (66.4%), early detection (66.1%), public education about sleep (49.1%) and non-pharmacological treatments (48.3%).

Conclusion: Adolescents with insomnia report significant insomnia-related distress and impairment, with symptoms often persisting for three or more years. However, adolescents with insomnia often do not seek professional help. Areas perceived as most problematic to adolescents living with insomnia (e.g., mood, focus, pain) should be considered as important outcomes for insomnia researchers.

Support: This publication was made possible with support from Grant Number UL1TR002529 (A Shekhar, PI) from the National Institutes of Health, National Center for Advancing Translational Sciences, Clinical and Translational Sciences Award and the Indiana University School of Medicine.

0922

NIGHTLY ASSOCIATIONS BETWEEN PRE-BEDTIME ACTIVITY, ACTIGRAPHIC LIGHT, AND SLEEP IN CHILDREN WITH ASD AND INSOMNIA

McGovney, K. D.¹ Curtis, A. F.¹ Mazurek, M.² Chan, W. S.³ Deroche, C. B.⁴ Munoz, M.⁵ Davenport, M.⁵ Takamatsu, S.⁶ Takahashi, N.⁷ Muckerman, J.⁷ McCann, D.¹ Sahota, P.⁸ Mills, B.¹ McCrae, C. S.¹

¹Department of Psychiatry, University of Missouri, Columbia, MO, ²Curry School of Education and Human Development, University of Virginia, Charlottesville, VA, ³Department of Psychology, The University of Hong Kong, Hong Kong, HONG KONG, ⁴Biostatistics and Medicine Research Design Unit, School of Medicine, University of Missouri, Columbia, MO, ⁵Department of Educational, School, and Counseling Psychology, University of Missouri, Columbia, MO, ⁶Children's Hospital Colorado, Aurora, CO, ⁷Thompson Center for Autism and Neurodevelopmental Disorders, University of Missouri, Columbia, MO, ⁸Department of Neurology, University of Missouri, Columbia, MO.

Introduction: Approximately two thirds of children with Autism Spectrum Disorder (ASD) suffer from chronic insomnia. Current behavioral interventions for insomnia in children with ASD use sleep hygiene guidelines to educate parents and their children regarding sleep promoting habits. However, the relationship between pre-bedtime physical activity/light and sleep is understudied in ASD. The current study examined daily associations between pre-bedtime actigraphically assessed activity/light levels and objective/ subjective sleep outcomes in children with ASD and insomnia.

Methods: Thirty children (Mage=8.5 yrs, SD=1.78 yrs) with comorbid ASD and insomnia completed 14 days of actigraphy measuring ambient white light intensity and activity levels every 30 seconds. Validated sleep scoring algorithms (in Actiware V. 6.0.9) estimated objective sleep onset latency (SOL), total sleep time (TST), wake time after sleep onset (WASO), and average activity/ light levels 30, 60, and 120 mins prior to bedtime. Additionally,

average activity/light levels 120-240, and 240-360 mins prior to bedtime were computed. Children also completed 14 daily sleep diaries (with parental assistance) measuring subjective reports of the same sleep parameters. Associations between daily estimations of pre-bedtime activity levels, light, and nighttime objective and subjective sleep were examined through multilevel modelling. Bonferroni corrections were performed to account for multiple comparisons.

Results: After Bonferroni corrections (p<.025 significance level), greater activity within 30 minutes (B=0.0465, p=.0093) and 60 minutes (B=0.0681, p=.0005) of bedtime were associated with longer subjective SOL. Pre-bedtime light exposure was not a significant predictor of sleep outcomes.

Conclusion: Results suggest that in general, variations in daily prebedtime activity, but not light, are associated with worse nightly subjective SOL in children with ASD and insomnia. Findings support that sleep hygiene recommendations in children with ASD include avoidance of higher levels of pre-sleep physical activity. Prospective studies examining temporal causal relationships between pre-bedtime activity and sleep in ASD are warranted.

Support: Research was supported by a University of Missouri Research Board award (McCrae, PI; Mazurek, Co-PI). Data collected as part of clinical trial NCT02755051 Targeting Sleep in Kids with Autism Spectrum Disorder at the University of Missouri (PI: McCrae).

0923

DOXEPIN IN CHILDREN AND ADOLESCENTS WITH SYMPTOMS OF INSOMNIA: A SINGLE CENTER EXPERIENCE

Shah, Y.¹ Kothare, S.²

¹alexandra and steven cohen children's medical center, Northwell Health, New Hyde Park, NY, ²steven and alexandra cohen children's medical center, New Hyde Park, NY.

Introduction: Pediatric insomnia is a widespread problem and especially difficult to manage in children with neurodevelopmental disorders. There are currently no FDA- approved medications for pediatric patients to use once first line therapy fails. Doxepin is FDA-approved at low doses for use in transient or chronic sleep maintenance insomnia in adults. The objective of this study is to determine the tolerability and efficacy of doxepin in the pediatric population.

Methods: This is a retrospective single center chart review of children and adolescents (2-17 years of age) whose sleep failed to improve with behavioral intervention and melatonin who were then trialed on doxepin. Treatment was initiated at a median starting dose of 2mg and slowly escalated to a median maintenance dose of 10mg. Improvement in sleep was recorded using a 4-point Likert scale reported by parents on follow up visits.

Results: Total of 29 patients were included in analysis. Mean follow-up duration was 6.5 months (\pm 3.5). Out of 29 patients, 4 (13.8%) patients discontinued doxepin due to lack of efficacy or side effects. 8 (27.6%) patients showed significant improvement of their insomnia, 8 (27.6%) showed moderate, 10 (34.5%) showed mild and 3 (10.3%) showed minimal to no improvement on treatment with doxepin (P<0.05) Only two patients (6.8%) experienced adverse effects in the form of behavioral side effects (aggression) and enuresis.

Conclusion: Our data suggests that doxepin is effective, safe and well-tolerated in the treatment of sleep initiation and maintenance insomnia as well as psychophysiological insomnia in child and adolescents with Ausitsm specutrm disorder, other neuro-developmental disorders and attention deficit hyperactivity disorder. It is also an effective, safe, and well-tolerated alternative in children suffering from chronic persistent insomnia. The results of this study suggests a promising emerging therapy for the treatment of insomnia in the pediatric population. **Support:** None

Support: NO

0924

ADOLESCENT PERCEPTIONS OF INSOMNIA TREATMENT

Simon, S. L.¹ Meltzer, L. J.² Williamson, A. A.³ Graef, D. M.⁴ Byars, K. C.⁴ Honaker, S. M.⁵

¹University of Colorado Anschutz Medical Campus, Aurora, CO, ²National Jewish Health, Denver, CO, ³Children's Hospital of Philadelphia, Philadelphia, PA, ⁴Cincinnati Children's Hospital Medical Center, Cincinnati, OH, ⁵Indiana University School of Medicine, Indianapolis, IN.

Introduction: Approximately 10% of adolescents meet diagnostic criteria for insomnia, which is associated with increased health problems, academic difficulties, and psychological morbidity. Empirical evidence supports cognitive-behavioral treatments for insomnia, yet research suggests adolescent insomnia is undertreated. Thus, the goal of this study was to evaluate adolescent perceptions about insomnia treatment seeking and strategies.

Methods: Adolescents ages 13-18 years with self-reported insomnia symptoms completed an online survey assessing treatmentseeking behaviors and management strategies. English-speaking participants were recruited using targeted Facebook advertising. Descriptive statistics were used to summarize results.

Results: Of the 3,014 survey respondents, participants were predominantly female (77%) and white non-Hispanic (71%), with a mean age of 16±1.3 years. Most (87%) met DSM-V diagnostic criteria for insomnia, but only 29% reported seeking professional help for insomnia. Of these, participants reported waiting an average of 1-2 years after symptom onset to seek treatment. Participants most often sought help from a mental health professional (18%) or primary care provider (13%), while <2% saw a sleep specialist. Across adolescents, the most commonly endorsed strategies to manage insomnia symptoms were daytime caffeine consumption (48%), staving out of bed except when trying to sleep (38%), and daily exercise (28%). Nearly half of participants endorsed currently using medications to help with sleep, most commonly melatonin (18%) and antihistaminebased sleep aids (10%). Participants reported greatest preference to take medication (64%), meet individually with a sleep specialist (48%), or use a phone or tablet application (40%) to help with sleep. While 30% of participants felt that sleep researchers should prioritize increasing access to insomnia treatment, 20% encouraged developing new modes for treatment delivery (e.g., online).

Conclusion: Adolescents with insomnia reported using behavioral and pharmacological strategies to help with sleep, with very few receiving treatment from a sleep specialist. Further research is needed to increase accessibility and acceptability of interventions for adolescent insomnia.

Support: N/A

0925

ASSOCIATIONS BETWEEN CO-SLEEPING AND SLEEP QUALITY OF URBAN PRESCHOOL CHILDREN

Stein, M.¹ DiSanti, N.¹ Weaver-Rogers, S.¹ Garcia, W.¹ Bonilla-Santiago, G.¹ Daniel, L. C.¹

¹Rutgers University Camden, Camden, NJ, ²Rutgers University Camden, Camden, NJ.

Introduction: Perspectives on co-sleeping vary by family cultural background and socio-demographic characteristics; furthermore, families may choose to co-sleep based on family values or out of necessity. To better understand the role of co-sleeping in young children from ethnic minority backgrounds, the current study sought to test the relationship between co-sleeping and sleep outcomes in an urban early childhood setting.

Methods: 141 Parent-child dyads with children aged 1-5 years old (N=141,50.4% female, M=4.01 [SD=1.05]; 41% African American; 41% Latinx; median income \$20-30,000) were recruited through an urban preschool. Parents completed demographic information and the Brief Child Sleep Questionnaire, which yielded insomnia and sleep hygiene indices. Step-wise regressions were used to examine the relationship between sleeping location and sleep outcomes (insomnia, sleep health, and child sleep quality), controlling for child age.

Results: Approximately half of the sample (n=71) reported that their child sleeps in a space shared by caregivers or siblings. Co-sleeping did not differ by race/ethnicity [$\chi^2(3)=1.45$, p=.694], child age [F(1, 140)=2.15, p=.145], or income [$\chi^2(5)=7.05$, p=.217]. Controlling for age, insomnia was higher in co-sleeping children [F(2,140)=4.10, p=.019], although sleep location was not a significant independent predictor. Sleep hygiene [F(2,140)=2.39, p=.095] and sleep quality [F(2,139)=0.94, p=.394] did not differ by sleeping location, when controlling for age.

Conclusion: Co-sleeping was common but was not related to sociodemographic factors as described in prior research. Controlling for age, co-sleeping predicted higher insomnia scores suggesting that co-sleeping may be related to symptoms of behavioral insomnia. Sleep hygiene practices and sleep quality did not differ by sleeping location, suggesting that in children without behavioral insomnia symptoms, co-sleeping may not affect sleep. Future studies that seek to better understand caregiver preference and intentions regarding co-sleeping may be important to intervention development seeking to improve behavioral insomnia in ethnic/minority samples. **Support:**

0926

EXAMINING SLEEP IN PARENTS AND CHILDREN WITH BEHAVIORAL OR CLINICAL SLEEP DISTURBANCES

Varma, P.¹ Jackson, M. L.² Junge, M.³ Conduit, R.¹

¹RMIT University, Melbourne, AUSTRALIA, ²Turner Institute for Brain and Mental Health, School of Psychological Sciences, Monash University, Clayton, AUSTRALIA, ³The Sleep Health Foundation, Blacktown, AUSTRALIA.

Introduction: Sleep problems, such as insomnia are frequently reported in children. These sleep disturbances have either a behavioral (e.g. difficulties initiating or maintaining sleep) or clinical etiology (e.g. autism, asthma and T1 diabetes). Unlike clinical populations, outcomes in parents of children with behavioral sleep problems are underexamined. This study aimed to examine sleep in parents and children with behavioral or clinical sleep disturbances. **Methods:** 290 parents (parents $M_{age} = 35.9\pm5.2$ y, children $M_{age} = 4.1\pm2.3$ y) were recruited. Child's sleep was reported using Child's sleep habits questionnaire (CSHQ). Parent sleep was assessed using Pittsburgh sleep quality index (PSQI), Pre-sleep arousal scale (PSAS), and Glasgow sleep effort scale (GSES). A demographic question on the nature of child's sleep disturbance was used to categorize parents into a) behavioral (n=206) or b) clinical group (n=84).

Results: Overall, 71% of parents and 67% of children had clinically significant sleep disturbance (PSQI \geq 5 and CSHQ \geq 41 respectively). Significant associations were observed between CSHQ and a) PSQI (r=0.47, p<.001), b) GSES (r=0.21, p<.001), and c) PSAS (r=0.46, p<.001). Step-wise regression reported that CSHQ was the strongest predictor of PSQI, accounting for 22% variance in scores (p<.001), followed by PSAS (7%). Independent groups t-tests found no differences in parents' sleep quality (p=.06) and pre-sleep arousal (p=.38) between clinical and behavioral groups. However, 47% of parents in the clinical group took longer than 30 minutes to fall asleep, as opposed to 30% of parents in behavioral group (t(289)=-2.39, p=.01).

Conclusion: Parents report having poor sleep irrespective of the nature of child's sleep difficulties. It is possible that parents in the clinical group may underreport their sleep problems due to increased attention towards child's symptoms and diagnosis. Nevertheless, if any sleep related issues occur in children, the impact on parental sleep should be considered during assessment. **Support:** N/A

0927

VARIATIONS IN SLEEP AND GLUCOSE IN ADOLESCENTS WITH TYPE 1 DIABETES

Griggs, S. Redeker, N. S. Jeon, S. Grey, M. Yale School of Nursing, West Haven, CT.

Introduction: The association between short sleep duration and poorer glycemic control in adolescents ages 10-16 with type 1 diabetes (T1D) is well established. Researchers have used cross-sectional, between-subjects' methods, with limited focus on the potential intraindividual variation among these variables. The purpose of this analysis was to examine the within person associations between glucose variability indices (J index, low/high blood glucose index, time in range) and sleep characteristics (bedtime, waketime, total sleep time, sleep efficiency, wake after sleep onset [WASO], awakenings, and sleep fragmentation index) in adolescents with T1D.

Methods: Adolescents monitored their sleep and glucose patterns concurrently for 3-7 days with a wrist actigraph on their non-dominant wrist and either their own continuous glucose monitor (CGM) or a provided blinded CGM. General linear mixed models (GLMM) were used to determine within-person and day level associations.

Results: The sample included 38 adolescents (M age 13.4±1.8; 37.8% male; M A1C 8.2±1.2%). Average glucose levels were controlled in all GLMMs. Adolescents had earlier waketimes on days when more time was spent in hypoglycemia <70mg/dL (β =-0.15, p<0.001). At the person level, adolescents had greater WASO with more % time spent in severe hypoglycemia <54mg/dL with more severe low blood glucose indices (β =0.35, p<0.01 and β =0.34, p<0.01 respectively). At the daily level, adolescents had greater WASO (β =0.20, p=0.01) and more awakenings (β =0.16, p=0.04) on the days they had more overall glucose variability (J index) and more severe high blood glucose indices (β =0.17, p=0.04), but were less likely to have more % time in hypoglycemia (β =-0.15, p=0.02). Conclusion: Glucose variability was positively associated with poor sleep (e.g., WASO and awakenings) in adolescents with T1D both at the daily and intraindividual level. Monitoring over a longer period of time in subsequent studies would allow researchers to determine the within person associations between habitual short sleep duration and glucose variability.

Support: NINR T32NR0008346 & P20NR014126, Medtronic MiniMed provided CGMs at a discounted rate for the study.

0928

HEART RATE VARIABILITY DURING SLEEP IN CHILDREN AND ADOLESCENTS WITH RESTLESS SLEEP DISORDER OR RESTLESS LEGS SYNDROME AND NORMAL CONTROLS

Ferri, R.¹ Bruni, O.² DelRosso, L. M.³

¹Oasi Research Institute - IRCCS, Troina, ITALY, ²Department of Social and Developmental Psychology, Sapienza University, Rome, ITALY, ³Seattle Children's Hospital, and University of Washington, Seattle, WA.

Introduction: Restless sleep disorder (RSD) has been recently characterized clinically and polysomnographically in children and differentiated from restless legs syndrome (RLS). Heart rate variability (HRV) is a reliable method to quantify autonomic changes during sleep. The aim of this study was to characterize HRV in children with RSD, RLS and normal controls, with the hypothesis that children with RSD have a shift toward sympathetic predominance during sleep.

Methods: Polysomnographic recordings from thirty-two children who fulfilled RSD diagnostic criteria (19 boys and 13 girls), 32 children with RLS (20 boys and 12 girls) and 33 controls (17 boys and 16 girls) were included. Four ECG epochs were chosen, one for each stage, and were analyzed for automatic detection of R waves. Time domain and frequency domain HRV parameters were obtained and analyzed.

Results: Age and gender were not statistically different between groups. In terms of time domain only the standard deviation of the average RR interval during stage N3 was slightly but significantly higher in RSD than in RLS patients. In terms of frequency domains, the LF band and the LF/HF ratio were increased in RSD and the HF percentage was lower in RSD during sleep stages N3 and R. The LF band and the LF/HF ratio increased in RLS and the HF percentage was lower in RLS during stage W.

Conclusion: Children with RSD have increased sympathetic activation during sleep, particularly N3 and REM, compared to controls but, as expected, not during wakefulness. Differently, children with RLS have sympathetic activation during relaxed wakefulness preceding sleep and during sleep.

Support: Partial support by a grant of the Italian Ministry of Health RC n. 2751598 (R.F.)

0929

CAREGIVER-REPORTED VERSUS CLINICIAN-DOCUMENTED CHILD SLEEP PROBLEMS AND SLEEP-RELATED HEALTH BEHAVIORS IN PRIMARY CARE

Williamson, A. A.^{1,2,3} *Bhandari, E.*⁴ *Cicalese, O.*^{1,2} *Heaps, E.*⁵ *Ostan, A.*⁵ *Collins, M.*⁵ *Lupini, F.*⁵ *Mindell, J. A.*^{5,1,3}

¹Sleep Center, Children's Hospital of Philadelphia, Philadelphia, PA, ²Department of Child and Adolescent Psychiatry and Behavioral Sciences, Children's Hospital of Philadelphia, Philadelphia, PA, ³Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, ⁴Wesleyan University, Middletown, CT, ⁵Department of Psychology, Saint Joseph's University, Philadelphia, PA.

Introduction: Sleep problems are highly prevalent in early childhood but often under-identified in pediatric primary care. This study identified the prevalence of caregiver-reported versus primary care provider (PCP)-documented sleep problems and sleep-related health behaviors in young children presenting to well child visits (WCVs).

Methods: Caregivers (85.4% mothers) of 198 young children (2-5 years, M = 3.3, 53.7% female, 62.9% Black) presenting to urban (74.1%) and suburban (25.9%) primary care sites for well child visits (WCVs) completed research surveys on child behavioral sleep problems, snoring, and sleep-related health behaviors (e.g., caffeine consumption) on the day of their child's WCV. Electronic medical record review was used to identify the rate of PCP-documented sleep problems and related recommendations in the WCV progress note.

Results: Fifteen percent of caregivers reported a child sleep problem according to questionnaire data, which did not significantly differ from the 12.0% of children with a PCP-documented sleep problem in the WCV progress note (p = .31). However, significantly more caregivers (28.3%) reported bedtime difficulties (resistance; tantrums) on questionnaires (p < .001), which were not captured in the 12.0% of WCVs noting sleep problems. A total of 8% of WCVs included child sleep recommendations. Child snoring was reported by 17.0% of caregivers, but was less frequently documented in WCVs (4.5%, p <.001). Although many caregivers reported poor child sleep-related health behaviors, including daily child caffeine consumption (21.1%) and bedroom electronics (62.9%), significantly fewer PCPs documented these issues (caffeine: 2.0%; electronics: 6.6%) or related recommendations (decrease caffeine: 1.0%; eliminate electronics: 3.5%) in the progress note (all p-values <.001).

Conclusion: Although caregiver-endorsed child sleep problems on surveys did not differ from PCP-documented concerns, there are gaps in documenting other problematic sleep-related health behaviors, such as caffeine consumption and electronics use. More resources to address sleep-related health behaviors, as well as sleep problems, in pediatric primary care are needed.

Support: Sleep Research Society Foundation and K23HD094905 (AAW)

0930

PRENATAL CANNABIS USE AND SLEEP OUTCOMES IN CHILDREN 9-10 YEARS OF AGE IN THE ADOLESCENCE BRAIN COGNITIVE DEVELOPMENT STUDY

Winiger, E. Hewitt, J.

Institute for Behavioral Genetics, Boulder, CO.

Introduction: The fetal brain is densely populated with CB1 receptors that increase in number throughout gestation and might be involved in sleep processes since they are found in many brain areas related to the regulation of the sleep-wake cycle. THC binds to CB1 receptors, possibly altering neurodevelopment and fetal cortical circuitry in the womb. Studies have found prenatal cannabis use is associated with early sleep factors from as early as few days after birth to 3 years of age, yet no studies have examined associations in later childhood.

Methods: We used data from the Adolescent Brain Cognitive Development (ABCD) study to determine whether maternal reports of cannabis use while pregnant were associated with child sleep outcomes (The Sleep Disturbance Scale for Children) among 11,875 children ages 9-10. Regression analyses accounted for the nested nature of families (twin and non-twin sibling) and were estimated controlling for potential covariates including daily tobacco and weekly alcohol use during pregnancy, mother's education, combined household income, parental marital status, child sex, and child age.

Results: Amongst mothers in our sample, 6% endorsed using cannabis while pregnant. Prenatal cannabis use was associated with lower sleep duration, disorders of initiating and maintaining sleep, disorders of arousal, sleep wake disorders, disorders of excessive somnolence, and a summed sleep score (all b >0.09 and p < 0.04) but not with sleep latency, sleep breathing disorders, and sleep hyperhidrosis (all b <0.08 and p >0.09).

Conclusion: Prenatal cannabis use was associated with increased childhood sleep deficits including shorter sleep duration and higher endorsements of sleep disorder symptoms. This is the first report of prenatal cannabis use being associated with sleep in childhood as late as 9-10 years of age. Although causality is not established, the results suggest potential long-term effects of prenatal cannabis use on sleep and the need for abstinence from cannabis use while pregnant.

Support: T32 DA017637. The ABCD Study is supported by the National Institutes of Health and additional federal partners under award numbers U01DA041022, U01DA041028, U01DA041048, U01DA041089, U01DA041106, U01DA041017, U01DA041120, U01DA041134, U01DA041148, U01DA041156, U01DA041174, U24DA041123, U24DA041147, U01DA041093, and U01DA041025 (https:// abcdstudy.org/federal-partners.html).

0931

PARENTING STRESS CAN BE PREDICTED BY PARENT AND CHILD SLEEP, AND CAN BE REDUCED BY AN INTERVENTION TARGETING CHILD SLEEP IN VULNERABLE FAMILIES

Potvin, J. Mercier, K. Bérubé, A. Raymond, N. Forest, G. Université du Québec en Outaouais, Gatineau, QC, CANADA.

Introduction: Studies suggest that family intervention programs should consider different sources of parenting stress (PS). Knowing that child sleep has an impact on PS, this study aimed to examine if a child sleep intervention can affect PS in vulnerable families.

Methods: In a first study, parent and child sleep habits along with PS were assessed using Parenting Stress Index-Short Form, Pittsburgh Sleep Quality Index and homemade questions assessing child sleep. Measures were administered to 138 community mothers (children 3-79 months). Multiple linear regression analyses examined mothers and child sleep associations to PS, controlled by family income. Following results of this first study, 11 vulnerable mothers (children 39-68 months) participated in a child sleep intervention specifically developed for vulnerable families. Measures administered were the same as for study 1, but in this study, the Child Sleep Habits Questionnaire was used to assess the child sleep. Wilcoxon's t-test compared pre and post-intervention measures.

Results: In the first study, sleep onset latency (SOL; $\beta = -.25$; p = .003), waketime ($\beta = -.28$; p = .003) and sleep aids ($\beta = -.19$; p = .048) explained 23.6% of PS variance. When child sleep variables were added to the model, only bedtime resistance was significant ($\beta = -.23$; p = .023) and increased the explained variance to 30.6%. Following the child sleep intervention, no significant changes in parent sleep were observed. However, results show improvement of child bedtime resistance ($M_{pre}=10.73$; $M_{post}=8.36$; p = .027) and PS ($M_{pre}=3.50$; $M_{post}=3.82$; p = .05).

Conclusion: Our results suggest that higher PS is explained by greater struggles in parents sleep (higher SOL, later waketime and higher use of sleep aids) as well as bedtime resistance behaviors in children. Furthermore, PS can be reduced by an intervention

improving child bedtime resistance, even if parent sleep is not improved. **Support:**

_ _

0932 MEDIA USE AND SHORT SLEEP DURATION AMONG CHILDREN AGED 3-17 YEARS—NATIONAL SURVEY OF CHILDREN'S HEALTH, 2016-2017

Wheaton, A. G. Greenlund, K. J. CDC, Atlanta, GA.

Introduction: Media use, such as television viewing and computer use, has been associated with insufficient sleep among children.

Methods: Analyses used data collected from parents of children aged 3-17 years via the National Survey of Children's Health in 2016 and 2017 (N=60,547). The prevalence of age-specific short sleep duration (<10 hours for ages 3-5 years, <9 hours for ages 6-12 years, and <8 hours for ages 13-17 years) was calculated by time spent (none, <1 hour, 1 hour, 2 hours, 3 hours, ≥4 hours) in front of a television (television time) and with computers or other electronic devices not for homework (computer time) on an average weekday. The prevalence of short sleep duration was also calculated adjusting for child sex and race/ethnicity, household income, parental education, special health care needs, general health, and bedtime regularity.

Results: The prevalence of short sleep duration was 35% for ages 3-5 years, 37% for ages 6-12 years, and 32% for ages 13-17 years. There was a significant linear relationship between both television and computer time and short sleep duration prevalence for all age groups, with the exception of television time for 13-17 year olds. For ages 3-5 years, prevalence of short sleep duration ranged from 18% (none) to 48% (≥4 hours) for television time (linear trend p<0.0001) and 23% (none) to 49% (≥4 hours) for computer time (linear trend p<0.0001). For ages 6-12 years, prevalence ranged from 23% (none) to 57% (≥4 hours) for television time (linear trend p<0.0001) and 26% (none) to 58% (≥4 hours) for computer time (linear trend p<0.0001). For ages 13-17 years, prevalence ranged from 15% (none) to 41% (≥4 hours) for computer time (linear trend p<0.0001). The associations remained significant after adjustment for covariates.

Conclusion: Reducing media use may increase sleep duration for children. The association between media use and sleep duration is similar for television and computer time for younger children; however, focusing on recreational computer use may be beneficial among teens. **Support:**

0933

PRESCHOOLERS SLEEP EXPLAINS PARENT COGNITIONS AND PARENTING BEHAVIORS

Raymond, N. S. Bérubé, A. Mercier, K. Forest, G. Université du Québec en Outaouais, Gatineau, QC, CANADA.

Introduction: Children sleep is influenced by biological and socioenvironmental factors. The contribution of various bedtime practices on child sleep is now well established. Emerging literature now seeks to understand the influence of more general parenting practices on child sleep. Thus, the current study examined how perceived children needs and parenting behaviors are associated with children sleep.

Methods: In a first study, 88 mothers (children 2-71 months) recruited during community activities completed the Child's Sleep

Habit Questionnaire, as well as a questionnaire regarding child needs (CN) and parents response to those needs (RN). Multiple linear regression analyses examined child sleep associations first to CN and then to RN, controlled by the child developmental score. Following this first study, 12 vulnerable mothers (children 39-68 months) participated in a child sleep intervention specifically developed for vulnerable families. The same measures were administered and Wilcoxon t-tests were calculated to compared pre and post intervention scores.

Results: Daytime sleepiness (β =.26, p=.008), sleep anxiety (β =.29, p=.007) and children cognitive development (β =-.32, p=.008) explained 31,6% of CN variance. Daytime sleepiness (β =.26, p=.03), and bedtime routine (β =-.68, p=.00), explained 20,8% of RN variance. Following the child sleep intervention, no change in CN were obtained, but a significant improvement in RN was found (M_{pre} =5.28 ± 1.60, M_{post} =4.33 ± .65, p=.045).

Conclusion: Results suggest that parents perceived more needs in their child when they present higher daytime sleepiness, higher sleep anxiety and cognitive developmental difficulties. On the other hand, parents have more difficulty responding to their child needs when they see sleepiness in their child or struggle with bedtime routine. A child sleep intervention does not seem to change the perception of child needs but have a positive impact on the parent response to those needs.

Support:

0934

AMBULATORY BLOOD PRESSURE MONITORING DURING OVERNIGHT POLYSOMNOGRAPHY IN CHILDREN AND ADOLESCENTS

DelRosso, L. M.¹ Chan, J.¹ Ruth, C.¹ Arp, M.¹ Ferri, R.² ¹Seattle Children's Hospital, Seattle, WA, ²Sleep Research Centre, Oasi Research Institute - IRCCS, Troina, ITALY.

Introduction: During NREM sleep, sympathetic nervous system input decreases and systolic blood pressure drops. Sleep disorders can alter this mechanism. Ambulatory blood pressure monitors (ABPM) are increasingly used in children and are superior to clinic blood pressure in predicting cardiovascular morbidity and mortality. We aim to assess the effect of ABPM use during a sleep study in children and adolescents.

Methods: Subjects were children ages 7-18 from Seattle Children's Hospital who underwent a sleep study for evaluation of suspected obstructive sleep apnea. Excluded children with known hypertension or taking a medication that altered blood pressure. An ABPM was placed on the right arm during the overnight sleep study and programmed to record BP every hour. Studies from children without OSA were analyzed for all sleep parameters. Data was compared with a control of children who underwent PSG and did not wear the ABPM. BP measurement was recorded and the PSG was reviewed for sleep stage, arousal, or awakening.

Results: To date, we have 15 children with ABPM age mean 11.6, SD 2.9 and 35 controls age mean 10.97 and SD 3.1, age, sex and BMI did not vary between groups. None of the PSG parameters was statistically different between groups including total sleep time, sleep efficiency, arousal index or sleep stage distribution. An average of 10 BP readings was obtained per patient. 53% of BP readings in N1 resulted in awakenings, compared to 39% in N2, 5% in N3 and 14.8% in REM. Systolic, diastolic, heart rate and mean arterial pressure showed appropriate dipping during sleep with appropriate circadian increase at 4 AM. Pulse pressure remained the same.

Conclusion: We have demonstrated the feasibility of using ABPM during PSG in children. Although sleep macroarchitecture did not show any significant difference in sleep efficiency or arousal index, further evaluation of each ABPM insufflation during the sleep study showed a sleep stage specific, sleep disruption with awakenings more likely to occur during N1 or N2 and less likely to occur during N3 or REM sleep. There was normal dipping and as expected circadian variability in the values. **Support:**

0935

ASSOCIATION BETWEEN CHRONOTYPE, SLEEP DURATION, WEEKEND CATCH-UP SLEEP, AND DEPRESSION AMONG KOREAN HIGH SCHOOL STUDENTS

Yang, K.¹ Jee Hyun, K.² Hwangbo, Y.³ Koo, D.⁴ Kim, D.⁵ Sunwoo, J.⁶ Hong, S.⁷

¹Sleep Disorders Center, Department of Neurology, Soonchunhyang University College of Medicine, Cheonan Hospital, Cheonan, KOREA, REPUBLIC OF, ²Dankook University College of Medicine, Dankook University Hospital, Cheonan, KOREA, REPUBLIC OF, ³Department of Preventive Medicine, Soonchunhyang University College of Medicine, Cheonan, KOREA, REPUBLIC OF, ⁴Department of Neurology, Seoul National University Boramae, Seoul, KOREA, REPUBLIC OF, ⁵Department of Neurology, Chungnam National University College of Medicine, Daejeon, KOREA, REPUBLIC OF, ⁶Department of Neurosurgery, Seoul National University Hospital, Seoul, KOREA, REPUBLIC OF, ⁷Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, KOREA, REPUBLIC OF.

Introduction: The present study aimed to examine the association between chronotype, sleep duration, weekend catch-up sleep (CUS) duration, and depression among Korean high school students. Methods: A total of 8,565 high school students who were analyzed from 15 nationwide districts in South Korea completed an online self-report questionnaire. Depressive mood was assessed using the Korean version of the Beck Depression Inventory. The following sleep characteristics were assessed: weekday and weekend sleep durations, weekend CUS duration, chronotype, perceived sufficiency of sleep, self-reported snoring and sleep apnea, daytime sleepiness, and sleep environment. Age, sex, body mass index, number of private classes, and proneness to internet addiction were also measured. Logistic regression analysis was conducted to compute odds ratios for the association between depression and sleep characteristics, after controlling for relevant covariates.

Results: The prevalence of depression (BDI \ge 16) was 1,794 (20.9%). In the analyses of multivariate logistic regression, the late chronotype (odds ratio [OR], 1.71; 95% CI, 1.47-1.99), female (OR, 2.24; 95% CI, 1.99-2.53), underweight (OR, 1.27; 95% CI, 1.02-1.57) and obesity (OR, 1.41; 95% CI, 1.13-1.75), weekday sleep duration (OR, 0.86; 95% CI, 0.81-0.91), weekend CUS duration \ge 2 hours (OR, 0.68; 95% CI, 0.55-0.85), ESS (OR, 1.08; 95% CI, 1.07-1.10), much (OR, 2.15; 95% CI, 1.63-2.84) and insufficient (OR, 1.71; 95% CI, 1.46-2.01) perceived sleep, snoring (OR, 1.27; 95% CI, 1.11-1.46) and witnessed apnea (OR, 2.10; 95% CI, 1.05-1.06), high number of private education (OR, 0.76; 95% CI, 0.60-0.95), and poor sleep environment (OR, 1.86; 95% CI, 1.56-2.21) were associated with depression.

Conclusion: Eveningness preference, insufficient weekday sleep duration, short weekend CUS duration, and self-reported snoring and sleep apnea were associated with an increased risk for depression. **Support:**

. . . .

0936 CARDIOMETABOLIC DISORDERS ARE INDEPENDENTLY ASSOCIATED WITH EXCESSIVE DAYTIME SLEEPINESS IN YOUNG ADULTS

*Fernandez-Mendoza, J.*¹ *Puzino, K.*¹ *Calhoun, S. L.*¹ *Qureshi, M.*¹ *He, F.*¹ *Liao, J.*¹ *Vgontzas, A. N.*¹ *Liao, D.*¹ *Bixler, E. O.*¹ ¹Penn State College of Medicine, Hershey, PA, ²Penn State College of Medicine, Hershey, PA.

Introduction: Cardiometabolic risk factors (CMR), including obesity, hypertension, diabetes and hypercholesterolemia, have been associated with sleep apnea and insufficient sleep, both of which can lead to excessive daytime sleepiness (EDS). We hypothesized that CMR are associated with EDS in young adults independent of sleep apnea, sleep duration and mental health disorders (MHD).

Methods: The Penn State Child Cohort is a population-based longitudinal sample of 700 children $(8.7\pm1.7y)$, of whom 421 were followed-up 8.3 years later during adolescence $(17.0\pm2.3y)$ and 425 another 7.0 years later during young adulthood $(24.4\pm2.6y)$. Subjects underwent a 9-h in-lab polysomnography in childhood and adolescence and parent- or self-reported standardized surveys at all time points. Self-reports in young adulthood and in-lab measurements in childhood were used to ascertain CMR and sleep apnea. Parent-reports in childhood and self-reports in young adulthood were used to ascertain the presence of MHD and EDS. Logistic regression models adjusted for age, race, sex, snoring/observed apneas, insomnia symptoms, and sleep duration in young adulthood as well as mean arterial blood pressure, body mass index percentile and apnea/hypopnea index in childhood.

Results: CMR (OR=2.71, 95%CI=1.69-4.36) and MHD (OR=4.61, 95%CI=2.79-7.62) were associated with EDS in univariate models. After adjusting for covariates in childhood and young adulthood, CMR and MHD remained independently associated with EDS (OR=2.32, 95%CI=1.29-4.16 and OR=2.78, 95%CI=1.59-4.87, respectively).

Conclusion: EDS in young adults with CMR or MHD does not solely arise from sleep apnea, insufficient sleep or other sleep disturbances. EDS may be the result of central pathophysiologic mechanisms or the functional impairment associated with cardiovascular, metabolic and mental health disorders. These data further support that youth with these disorders should be screened for EDS and appropriately managed.

Support: National Institutes of Health (R01HL136587, R01HL97165, R01HL63772, UL1TR000127)

0937

THE EVALUATION OF BRAIN MATURATION BY REM SLEEP ANALYSES DURING PUBERTY USING FAST FOURIER TRANSFORM

Lopes, M.¹ Roizenblatt, S.²

¹University of Sao Paulo, Sao Paulo, BRAZIL, ²Federal University of Sao Paulo, Sao Paulo, BRAZIL.

Introduction: Brain maturation has been associated with electroencephalogram (EEG) changes during rapid eye movement (REM) sleep. There is a higher delta power during sleep in the first decade of the human EEG and this fact might be related to puberty period. Most studies assessed EEG during wakefulness and NREM sleep. The aim of this study was to evaluate changes in the REM sleep EEG spectral analysis across puberty.

Methods: Twenty healthy children were studied. They were divided into two groups: early puberty (n=10, ageranging from 6 to 12) and late puberty (n=10, age= ranging from 13 to 18). Polysomnography was performed in 2 nights, one for adaptation purpose. The Tanner scales were obtained and exclusion criteria were the presence of sleep and daytime complaints at least 14 days before recruitment. Fast Fourier Transform (FFT) was performed in C3-A2 derivation throughout all night. The FFT was calculated in 4s windows and the mean of delta (0.5-2.0 Hz), delta 2 (2.0-4.0 Hz), theta (4.0-8.0 Hz), alpha (8.0 - 12.0 Hz), sigma (12.0-16.0 Hz) and beta (16.0 -20.0 Hz) were obtained.

Results: We found differences during NREM and REM sleep between two groups (U-test, p<0.05). In REM sleep, the delta 2 (U-test, p=0.02)and theta power were higher in early puberty group (U-test p=0.04). The delta power correlated negatively with the duration in minutes of stage 1 (r_s =-0.46 p<0.05), and the wake time after sleep onset (r_s =-0.48, p<0.05) and correlated positively with sleep efficiency (r_s =0.45, p<0.05). Theta power during REM sleep also correlated positively with N3 sleep stage (r_s =0.45, p<0.05).

Conclusion: The REM sleep can be an extremely useful biomarker of brain function for future therapeutic protocols. The present results suggest that there are changes in REM sleep EEG throughout puberty, and that they may be related to puberty brain maturation. The hormone therapy may have an action in the REM behavioral Sleep Disorder. Future studies are need to evaluate this hypothesis. **Support:** N/A

0938

SLEEP, PERFECTIONISM AND COGNITIVE ANXIETY IN SPORTS: AN EXPLORATORY STUDY

Ramos Socarras, L. Bourgon, V. Mercier, K. Forest, G. Université du Québec en Outaouais, Gatineau, QC, CANADA.

Introduction: Perfectionism has been related to cognitive anxiety in sports. The bidirectional association between sleep and anxiety has also been well studied. However, the relationship between sleep habits, anxiety and perfectionism in physically active young individuals hasn't been documented yet. This was the objective of the present study.

Methods: 150 young, physically active participants (10 to 18 years old; 47% boys, 51% girls) completed an online survey comprised of questions extracted from the Sport Multidimensional Perfectionism Scale-2, the Competitive State Anxiety Inventory-2R and the Adolescent Sleep Habits Survey. First, independent t-tests were conducted to compare sleep habits, personal standards (PS), perceived parental pressure (PPP) and perceived coach pressure (PCP) of more anxious (ANX) to less anxious (NOANX) participants. Then, stepwise multiple linear regression analyses were computed to examine the significant sleep habits and perfectionism variables associations with competitive cognitive anxiety.

Results: Results show that ANX participants have higher PS (t(148)=3.19, p=0.002), less total sleep time on weekend (TST; t(148)=-2.94, p=0.004), longer sleep onset latency (SOL) on weeknights (t(128.09)=2.28, p=0.03) and report more daytime sleepiness (t(148)=3.06, p=0.003) compared to NOANX participants. The significant sleep variables and PS collectively explained 17.7% of competitive cognitive anxiety variance (p=0.00). PS was

the largest predictor (β =0.27, p=0.00), followed by daytime sleepiness (β =0.21, p=0.007), weekend TST (β =-0.17, p=0.023) and weeknight SOL (β =0.16, p=.038).

Conclusion: These results suggest that, even though PS is associated with cognitive anxiety, sleep seem to be an important factor to consider. Specifically, increased daytime sleepiness accompanied by difficulties falling asleep during school nights and no recovery sleep during weekends significantly contribute to competitive cognitive anxiety. These results could have important implications when addressing competitive anxiety issues in young athletes. **Support:** N/A

0939

ANXIETY SYMPTOMS MODERATE THE EFFECTS OF SLEEP LOSS ON CHILDREN'S EMOTIONS

Alfano, C. A.¹ Bower, J.² Harvey, A.³ Beidel, D.⁴ Sharp, C.¹ Palmer, C. A.⁵

¹University of Houston, Houston, TX, ²DeMontfort University, Leicester, UNITED KINGDOM, ³University of California, Berkeley, Berkeley, CA, ⁴University of Central Florida, Orlando, FL, ⁵Montana State University, Bozeman, MT.

Introduction: An abundance of cross-sectional research links inadequate sleep with poor emotional health, but experimental studies in children are rare. Further, the impact of sleep loss is not uniform across individuals, and pre-existing anxiety might potentiate the effects of poor sleep on children's emotional functioning.

Methods: N=53 children (mean age 9.0 years; 56% female) completed multi-modal, emotional assessments in the lab when rested and after two nights of sleep restriction (7h and 6h in bed, respectively). Sleep was monitored with polysomnography and actigraphy. Subjective reports of affect and arousal, psychophysiological reactivity, and objective emotional expression were examined during two emotional processing tasks, including one where children were asked to suppress their emotional responses.

Results: After sleep restriction, deleterious alterations were observed in children's affect and their emotional reactivity, expression, and regulation. These effects were primarily limited to positive emotional stimuli. The presence of anxiety symptoms moderated most of the alterations in emotional processing observed after sleep restriction.

Conclusion: Results suggest inadequate sleep preferentially impacts positive compared to negative emotion in pre-pubertal children and that pre-existing anxiety symptoms amplify these effects. Implications for children's everyday socio-emotional lives and long-term affective risk are highlighted.

Support: NIMH grant #R21MH099351

0940

ADOLESCENT SLEEP QUANTITY, PARENTING PERMISSIVENESS, AND SUGARY BEVERAGE CONSUMPTION

Rubens, S. L. Thai, C. L. Santa Clara University, Santa Clara, CA.

Introduction: Research suggests that poor sleep is associated with sugary beverage consumption (SBC), however, little is known about contextual factors that may play a role in this association. This study examined the association between sleep quantity and SBC while accounting for parenting permissiveness among a national sample of adolescents.

Methods: Participants included 1542 adolescents (Mean age = 14.47, SD=1.60) participating in the National Cancer Institute's Family Life, Activity, Sun, Health, and Eating survey, a web-based survey administered in 2014. Multivariable regression models were used to examine associations between sleep quantity (<8 hrs, 8-10hrs, >10hrs) and frequency of sugary beverage consumption in the past week (soda, energy drinks, sports drinks; not at all to 3+ times/day) while controlling for parenting permissiveness and sociodemographic factors (age, sex, parent education level, race).

Results: Approximately 58% (n = 1075) of participants reported receiving the recommended 8-10 hours of sleep, while 35% (n = 646) reported receiving less than this recommended amount and 7% (n = 138) reported sleeping more than the recommended amount of time. Findings from multiple regression models indicated that, when accounting for parenting permissiveness and sociodemographic factors, adolescents who slept the recommended number of hours (8-10) reported less frequent consumption of soda ($\beta = -.18$, p = .02) and energy drinks ($\beta = -.12$, p = .003) compared to those who slept less than 8 hours. Sleep quantity was not associated with frequency of sports drink consumption in unadjusted and adjusted models (p > .05).

Conclusion: While effect sizes are small, findings suggest that sleep quantity is associated with caffeinated sugary beverage consumption above and beyond parental permissiveness. Given the public health concerns of SBC, examining the influence of developmentally appropriate sleep interventions on SBCs in adolescents is essential.

Support: n/a

0941

DEFINING DISRUPTED NIGHTTIME SLEEP IN PEDIATRIC NARCOLEPSY

Maski*, K. P.¹ Pizza*, F.² Colclasure, A.³ Steinhart, E.¹ Little, E.¹ Diniz Behn, C.³ Vandi, S.² Antelmi, E.² Plazzi**, G.² Scammell**, T.⁴

¹Boston Children's Hospital, Boston, MA, ²Department of Biomedical Science and Neuromotor Sciences, University of Bologna, Bologna, ITALY, ³Department of Applied Math and Statistics, Colorado School of Mines, Golden, CO, ⁴Beth Israel Deaconess Medical Center, Boston, MA.

Introduction: Disrupted nighttime sleep (DNS) is a core narcolepsy symptom subjectively described as spontaneous awakenings during the night, but researchers use varied polysomnogram (PSG) definitions based on sleep state transitions, NREM 1% and poor sleep efficiency. These sleep measures have yet to be validated to determine the best objective measure of DNS. Furthermore, it unknown to what extent DNS occurs in pediatric narcolepsy as children have greater sleep drive than adults. Here, we assess the construct validity of various DNS objective measures and evaluate its diagnostic utility for pediatric Narcolepsy Type 1 (NT1) when combined with a nocturnal Sleep Onset REM period (nSOREMP) in a large cohort of pediatric patients with CNS hypersomnias.

Methods: Retrospective, cross-sectional study of consecutive PSGs and multiple sleep latency tests (MSLTs) obtained at Boston Children's Hospital and University of Bologna. Participants were drug-free or drug naïve, ages 6-18 years and slept at least 6 hours during the PSG. We analyzed associations between objective DNS measures and outcomes of self-reported sleep disturbance, Epworth Sleepiness Score, mean sleep latency, NT1 diagnosis, and CSF hypocretin values when available. We then combined the best performing DNS measure with the presence of a nSOREMP to determine the diagnostic value for NT1 using bootstrap analysis. We included n=151 NT1, n=21 narcolepsy type 2 (NT2), n=27 idiopathic hypersomnia (IH) and n= 117 subjectively sleepy controls in this analysis.

Results: Across groups, the Wake and NREM 1 bouts index had the most robust associations with objective sleepiness, subjective sleep disturbance and CSF hypocretin levels (p's <0.0001). From 1000 bootstrap samples, the Wake/N1 index and presence of a nSOREMP have greater diagnostic accuracy for NT1 than the nSOREMP alone (p<0.0001).

Conclusion: Among a variety of sleep quality measures, we find that a Wake and NREM 1 bout index is the best objective measure of DNS. In combination with a nSOREMP, this DNS measure can aid in pediatric NT1 diagnosis using PSG alone and potentially reduce diagnostic delays.

Support: This study was supported by K23 National Institutes of Health (NINDS, K23 NS104267-01A1) grant and Investigator Initiated Research grant from Jazz Pharmaceuticals, Inc. to Dr. Maski

0942

ALTERNATING LEG MUSCLE ACTIVATION AND HYPNAGOGIC FOOT TREMOR IN CHILDREN: COMPARATIVE STUDY OF POLYSOMNOGRAPHIC AND CLINICO-DEMOGRAPHIC VARIABLES

de Menezes, G. M. Almeida, L. A. Sander, H. H. Fernandes, R. M. Éckeli, Á. L.

Ribeirao Preto Medical School - University of Sao Paulo, Ribeirão Preto - SP, BRAZIL.

Introduction: The clinical and polysomnographic meaning of the Alternating Leg Muscle Activation (ALMA) and Hypnagogic Foot Tremor (HFT) patterns in children is not known.

Methods: A descriptive study was carried out to identify the prevalence and polysomnographic characteristics of ALMA and HFT sequences in a sample of 122 children sequentially admitted in the sleep laboratory, with the analysis of clinical and demographic characteristics of the ALMA/HFT group in relation to a comparison group without this condition, paired by age and gender.

Results: Sample prevalence was 14.8% for any HFT/ALMA event, 13.1% for ALMA and 10.7% for HFT. In the HFT/ALMA group, the mean age was 8 years old (2-12 years old), 66.7% of males. Obstructive Sleep Apnea was observed in 75% of children, but HFT / ALMA sequences only occasionally occurred in association with respiratory events. The use of medications with monoaminergic activity was associated with the occurrence of HFT/ALMA group, p=0,019. There was higher N1 sleep content in the HFT / ALMA group, p=0,0301. There was no significant difference between both groups regarding the other clinical-demographic or polysomnographic parameters analyzed. Autonomic activation represented by heart rate fluctuations often occurred in association with the HFT / ALMA sequences, irrespective of the occurrence of arousals, awakenings, other motor or respiratory events.

Conclusion: HFT / ALMA is a frequent condition in children that are referred to the sleep lab.The stereotypy of the HFT / ALMA series suggests that their origin might be motor central pattern generators, which are potentially influenced by substances with monoaminergic effect. The finding of higher superficial sleep content in children with HFT / ALMA may indicate greater susceptibility to alteration of pediatric sleep architecture by such subtle motor events. The possibility of clinical consequences and cardiovascular diseases should be considered in relation to the association of HFT / ALMA with observed autonomic activation. **Support:** None.

0943

INCREASED NON-REM SLEEP INSTABILITY IN CHILDREN WITH RESTLESS SLEEP DISORDER

DelRosso, L. M.¹ Hartmann, S.² Baumert, M.² Bruni, O.³ Ferri, R.⁴ ¹Seattle Children's Hospital, Seattle, WA, ²The University of Adelaide, School of Electrical and Electronic Engineering, Adelaide, AUSTRALIA, ³Department of Social and Developmental Psychology, Sapienza University, Rome, ITALY, ⁴Oasi Research Institute, Troina, ITALY.

Introduction: Restless sleep disorder (RSD) is a newly recognized condition characterized by motor movements involving large muscle groups with frequent repositioning or bed sheets disruption. We analyzed cyclic alternating pattern (CAP) in these children, a marker of sleep instability that might be associated with the motor episodes of RSD and may play a role in their daytime symptoms.

Methods: Polysomnographic recordings from thirty-eight children who fulfilled RSD diagnostic criteria (23 boys and 15 girls), 23 children with restless legs syndrome (RLS, 18 boys and 5 girls) and 19 controls (10 boys and 9 girls) were included. For CAP analysis, a previously developed, highly precise automated system, based on a deep learning recurrent neural network, was used.

Results: Age and gender were not statistically different between groups. RSD patients showed a lower percentage of A3 CAP subtypes than controls (median 9.8 vs. 18.2, p=0.0089), accompanied by shorter duration of the B phase of the CAP cycle (median 28.2 vs. 29.8 in controls, 30.2 in RLS, p=0.005) and shorter CAP cycle duration than both controls and RLS subjects (median 33.8 vs. 35.0 in controls, 35.8 in RLS, p=0.002). Finally, RSD children also showed a longer duration of CAP cycle sequences, when compared to controls (median 172.7 vs. 141.9, p=0.0063).

Conclusion: In conclusion, our study indicates that NREM sleep EEG shows an increased instability in RSD; these findings add to the current knowledge on the mechanisms of this newly recognized sleep disorder and suggest that sleep instability might be a favoring mechanism for the emergence of the motor episodes characterizing RSD.

Support: Partial support by a grant of the Italian Ministry of Health RC n. 2751598 (R.F.)

0944

SHHH! INITIATIVE: SLEEP HEALTH PRACTICES IN PEDIATRIC HOSPITALS

Benoit, K. M.¹ Harkins, E.² Olsen, S.¹ Sterni, L.² Wolfson, A. R.¹ ¹Loyola University Maryland, Baltimore, MD, ²Johns Hopkins University, Baltimore, MD.

Introduction: Hospitalizations often result in significant sleep disruption, despite the importance of sleep in healing (Cmiel, et al., 2004). Research-to-date has focused primarily on adult intensive care (ICU) with minimal focus on pediatric patients. The aim of this study was to assess pediatric inpatient healthcare providers' understanding of and attitudes towards sleep in the hospital environment with the goal of developing a sleep health educational intervention as well as modifications to standards of care that unnecessarily interrupt sleep of pediatric inpatients.

Methods: An online survey was administered to pediatric inpatient staff (nurses, physicians, residents) at a Mid-Atlantic children's hospital focused on assessing their understanding of sleep in the context of inpatient care (N = 316). Respondents were 30-50 years old (54%), primarily identified as female (88%), and most (60%) reported being in a nursing position.

Results: Quantitative findings (N = 316) revealed that 65% reported patients were sometimes, rarely, or never allowed to sleep without being awakened from administration of non-critical medications. A majority (63.8%) reported that sometimes, rarely, or never do they consider interruption of sleep in decisions on when to give medications, while 54.9% reported the quantity and quality of sleep is rarely/never considered in a patient's treatment. Qualitative responses (N = 248) confirmed these findings with 34.3% reporting that they considered re-scheduling medications to minimize sleep interruptions. Despite this finding, only 15.7% reported they would assess or give attention to sleep in the context of patient recovery and treatment.

Conclusion: Pediatric healthcare providers are aware of the importance of sleep for their patients; however, they are not prioritizing sleep as a part of treatment in their behaviors and decisions. Next steps include developing and implementing an intervention for pediatric healthcare providers to follow through on limiting sleep interruptions as well as focusing on sleep in the treatment process.

Support: N/A

0945

ADOLESCENT SLEEP MEDIATES MATERNAL DEPRESSION AND HARSH PARENTING

Stearns, M.¹ Wilkerson, A.² Speed, K. J.³

¹Mississippi State University, Starkville, MS, ²Medical University of South Carolina, Charleston, SC, ³VISN ² Center of Excellence for Suicide Prevention, Canandaigua, NY.

Introduction: Mothers dealing with depressive problems often report using more harsh parenting practices. This occurs, in part, due to a scarcity of effective coping mechanisms and increased irritability. In addition, depressed mothers are less likely to set consistent rules and expectations within the home, which may result in children who stay up late. Children who get inadequate levels of sleep also are more likely to have behavior problems, irritability, and defiance toward their parents, particularly in adolescence. However, no studies have examined the potential of adolescent sleep as a contributor to the association between maternal depression and the use of harsh parenting. The current study examined whether mothers' perceptions of inadequate adolescent sleep duration mediated the relationship between maternal depression and harsh parenting, with child gender as a moderator.

Methods: The sample (N=318) consisted of mothers reporting on adolescents aged 16-18 (M=16.89, SD = .429; 53.4% female) from the 10th wave of the Schools and Families Educating Children Study (SAFE). The SAFE study was a randomized control trial conducted from 1997-2008 designed to investigate children and families living in inner-city Chicago, II. Measures included the Child Behavior Checklist (CBCL), Center for Epidemiologic Studies Depression Scale (CESD), and the Parenting Practices Questionnaire (PPQ).

Results: Too little adolescent sleep mediated ($\beta = .15$) the relation between maternal depression and her reported use of harsh parenting. Mediation was further moderated by child gender, such that the mediation occurred for sons ($\beta = .12$) but not daughters.

Conclusion: These results suggest that too little adolescent sleep is the process through which mothers experiencing depressive problems engage in more harsh parenting. In addition, important child gender differences were apparent, such that sons' lack of sleep may be more related to maternal depression and the use of harsh discipline.

Support: United States Department of Health and Human Services. National Institutes of Health. National Institute on Drug Abuse (5 R01 DA020829)

0946

PARENT-PERCEIVED SLEEP PROBLEMS ASSOCIATED WITH COMMON MEDICAL ISSUES DURING INFANCY

Mindell, J. A.^{1,2} Leichman, E. S.² Williamson, A. A.^{1,3} Gould, R. A.⁴ Hiscock, H.^{5,6,7} Ouach, J.^{8,9}

¹Children's Hospital of Philadelphia, Philadelphia, PA, ²Saint Joseph's University, Philadelphia, PA, ³Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, ⁴Janssen R&D, World Without Disease Accelerator, Johnson & Johnson, Skillman, NJ, ⁵Health Services Research Unit, The Royal Children's Hospital, Melbourne, AUSTRALIA, ⁶Centre for Community Child Health, Murdoch Children's Research Institute, Melbourne, AUSTRALIA, ⁷Department of Pediatrics, University of Melbourne, Melbourne, AUSTRALIA, ⁸Melbourne Graduate School of Education, The University of Melbourne, Murdoch Children's Research Institute, Melbourne, AUSTRALIA, ⁹Policy, Equity and Translation, Murdoch Children's Research Institute, Melbourne, AUSTRALIA, ⁹Nelbourne, AUSTRALIA, ⁹Nelbourne, Murdoch Children's Research Institute, Melbourne, AUSTRALIA, ⁹Nelbourne, AUSTRALIA, ⁹Nelbourne, Murdoch Children's Research Institute, Melbourne, AUSTRALIA, ⁹Nelbourne, AUSTRALIA, ⁹Nelbourne, Murdoch Children's Research Institute, Melbourne, AUSTRALIA, ⁹Nelbourne, AUSTRALIA, ⁹Nelbourne, Murdoch Children's Research Institute, Melbourne, AUSTRALIA, ⁹Nelbourne, ⁹Nelbourne, ⁹Nelbourne, ⁹Nelbourne, ⁹Nelbourne, ⁹Nelbourne, ⁹Nelbourne,

Introduction: Sleep problems are highly prevalent during infancy. However, little research has been conducted on associations between these sleep issues and common medical concerns in early development. Thus, the purpose of this study was to assess the prevalence of parent-perceived sleep problems in infants with common medical problems.

Methods: Participants were 5,097 children from the Longitudinal Study of Australian Children—Birth Cohort. Caregiver-reported child sleep problems and medical concerns were assessed at ages 0-1 year. Chi-square analyses were used to examine associations between the presence of a parent-perceived sleep problem and medical concerns.

Results: Wheezing (29.6%), eczema (14.9%), and food/digestive allergies (5.0%) were the most commonly identified medical concerns. In addition, 17.1% of caregivers reported a moderate/severe child sleep problem. Infants who had a moderate to severe parent-identified sleep problem experienced higher rates of overall medical care/needs, wheezing, eczema, food/digestive allergies (p<.001), ear infections (p<.05), and other illnesses (p<.01) than those infants without a sleep problem. No differences were observed with regard to hearing problems, vision problems, developmental delay, diarrhea/colitis, anemia, or other (non-ear) infections. Furthermore, parents reported higher rates of sleep problems for infants with medical problems (20.0-37.5%) than for infants without medical problems = 27%), food/digestive allergies (27%), eczema (23%), and wheezing (20%), p=.001.

Conclusion: Overall, common medical issues during infancy, including food/digestive allergies, eczema, and wheezing, are associated with greater parent-endorsed child sleep problems. Primary care providers should assess for and address sleep problems when treating common medical concerns during infancy.

Support: This project was partially supported by Johnson and Johnson Consumer Health (JAM, ESL, and RAG) and NIH K23HD094905 (AAW).

0947

PARENT-PERCEIVED SLEEP PROBLEMS AND SLEEP GOALS IN INFANTS AND TODDLERS: A GLOBAL SAMPLE

Mindell, J. A.^{1,2} *Goh, D.*³ *Collins, M.*¹ *Bartle, A.*⁴ *Kohyama, J.*⁵ *Sekartini, R.*⁶ *Veeravigrom, M.*⁷ *Leichman, E. S.*¹

¹Saint Joseph's University, Philadelphia, PA, ²Children's Hospital of Philadelphia, Philadelphia, PA, ³National University Hospital, Singapore, SINGAPORE, ⁴Sleep Well Clinics, Auckland, NEW ZEALAND, ⁵Tokyo Bay Urayasu/Ichikawa Medical Center, Urayasu, JAPAN, ⁶Medical School University of Indonesia, Jakarta, INDONESIA, ⁷Chulalongkorn University and King Chulalongkorn Memorial Hospital/ The Thai Red Cross Society, Bangkok, THAILAND.

Introduction: The aim of this study was to assess parent perceptions of sleep problems in young children and parent-identified areas of change in a global sample.

Methods: Caregivers (95.6% mothers) of 1555 infants/toddlers (birth-37 mos; M=12.2 mos; 49.5% male) completed an online survey, representing Indonesia (n=187), Japan (n=718), New Zealand (n=231), Singapore (n=199), and Thailand (n=221). The survey included an abbreviated version of the Brief Infant Sleep Questionnaire, and a list of potential sleep-related areas of change. **Results:** 36.9% reported a perceived sleep-problem, whereas 92.9% indicated an area of desired change related to their child's sleep. In terms of areas of change, 82.5% endorsed bedtime/how child falls asleep, 70.0% nighttime sleep, and 57.8% related to the morning. As expected, 99.7% of parents who endorsed a problem indicated a desired change compared to 88.9% who did not perceive a problem, p < .001. Those who noted a problem were more likely to endorse a change at bedtime (92.5%) and during the night (90.1%), compared to the morning (68.8%). There were country-based differences, with caregivers in New Zealand (47.0%) and Singapore (44.2%) more likely to report a child sleep problem compared to Thailand (35.3%), Japan (34.1%) and Indonesia (29.4%), p <.001. No differences were noted in parent-report of desired change across Japan, New Zealand, Singapore, and Thailand (94-96%) but were significantly higher than Indonesia (83.4%).

Conclusion: Although one-third of parents of young children in a global sample indicate a perceived sleep problem, almost all parents wish to change something about their child's sleep, primarily relate to bedtime and during the night. Sleep education and assessment delivered by health care providers should focus not only on what families consider to be "problematic," but also what families would like to modify, or improve, about their child's sleep within a developmentally appropriate framework.

Support: Johnson & Johnson Consumer Inc., Skillman, NJ, USA.

0948

SLEEP QUALITY, DEPRESSIVE SYMPTOMS, AND STRESS IN MATERNAL CAREGIVERS OF YOUNG CHILDREN WITH BRONCHOPULMONARY DYSPLASIA

Feeley, C. Chasens, E.

University of Pittsburgh, Pittsburgh, PA.

Introduction: Maternal caregivers of young children with a chronic illness, like bronchopulmonary dysplasia (BPD), often report

increased levels of stress and depressive symptoms, as well as poor quality of sleep. Caregiving duties are time-intensive and extend into the nighttime hours. The purpose of this study is to examine sleep, depressive symptoms, and stress in maternal caregivers of young children with BPD, as well as determine if depressive symptoms is a mediating variable between sleep quality and stress.

Methods: 61 maternal caregivers (mean age 29 yrs) of young children with BPD (mean age 14 mos) were recruited. Mothers had no reported history of a sleep disorder, and had a child diagnosed with BPD. The child had to have been home from the hospital for at least two months, and not require a ventilator or tracheotomy. Maternal caregivers were asked to complete a demographic questionnaire, as well as the Pittsburgh Sleep Quality Index (PSQI), the Perceived Stress Scale (PSS), and the Center for Epidemiological Studies-Depression Scale (CES-D). Upon completion, 56.9% of the sample was single, 67.2% were African American, and child had been home from hospital a mean of 8 mos.

Results: Over two thirds (67.2%) of the sample reported sleeping 6 or fewer hours a night, with a mean PSQI score of 7.6. Significant correlations were found between PSQI and CESD (; r=.546; p=.000), as well as PSQI and stress (r=.284; p=.031). Depressive symptoms were not found to mediate the relationship between sleep quality and stress, however, sleep quality was a significant predictor of stress (t=2.171; p=.034) and depressive symptoms (t=4.876; p=.000).

Conclusion: The majority of maternal caregivers of children with BPD reported insufficient and poor quality of sleep, which may affect their stress and depressive symptoms. Healthcare providers need to discuss the importance of sleep with caregivers, and ensure the child's care schedule allows for sleep during the nighttime hours.

Support:

0949

SHOULD PARENTS AND CHILDREN SHARE THE SAME BED? A SYSTEMATIC REVIEW OF THE CONSEQUENCES OF BEDSHARING ON SLEEP AND CHILD DEVELOPMENT

Morneau, A. Vallieres, A.

Ecole de psychologie, Universite Laval, Quebec, QC, CANADA.

Introduction: Bedsharing is a common practice worldwide except in Western societies where it is more controversial. The advantages and disadvantages of bedsharing on child health, safety, and development have been widely debated. This study examines the potential consequences of bedsharing on sleep and child development.

Methods: The systematic review was conducted in accordance with PRISMA guidelines and the University librarian advisor. Height electronic data base were searched with no limit on date. To be selected, studies must include only regular practice of bedsharing, have been conducted with children sharing the bed with their parents before the age of three, use an observational design, and present results on sleep quality or duration. The effect of bedsharing was assessed with six outcome measures: sleep quality, sleep duration, attachment quality, autonomy and independence, cognitive skills, as well as emotional and behavioural problems. Due to high heterogeneity in the measures used between studies, a narrative synthesis was done. Studies' quality was assessed using the *Scottish Intercollegiate Guidelines Network* framework.

Results: A total of 28 publications were included, 17 of which were cross-sectional and 11 prospective studies. The studies were

carried out between 1989 and 2017 in 25 countries. Sample sizes range from 41 to 55,831 participants aged from 6-week to 18-year old. Samples presented an equivalent amount of boys and girls. 16 studies reported data only on sleep, 7 only on child development, and 5 on both. The quality of the studies was considered low and moderate. For sleep, studies have shown a significant increase in the number of nocturnal awakenings among children who share the parental bed. Evidence of an association between bedsharing and other sleep parameters (difficulty falling asleep, resistance to bedtime, and sleep duration) is inconclusive. Regarding child development, only three studies found a positive impact on autonomy, cognitive skills, and anxiety. The absence of significant results in the remaining studies suggests that bedsharing is not a determining factor in child development.

Conclusion: The heterogeneity between studies and the quality of available data prevents definitive conclusions about the effects of regular bedsharing on sleep and child development.

Support: No financial support

0950

EFFECTS OF SODIUM OXYBATE (SXB) ON BODY MASS INDEX (BMI) IN PEDIATRIC PATIENTS WITH NARCOLEPSY

Dauvilliers, Y.¹ Lammers, G. J.² Lecendreux, M.³ Plazzi, G.⁴ Maski, K.⁵ Kansagra, S.⁶ Mignot, E.⁷ Menno, D.⁸ Wang, Y.⁹ Rosen, C. L.¹⁰

¹Sleep and Wake Disorders Centre, Department of Neurology, Gui de Chauliac Hospital, Montpellier, FRANCE, ²Sleep-Wake Center, Stichting Epilepsie Instellingen Nederland (SEIN), Heemstede, NETHERLANDS, ³Centre Pédiatrique des Pathologies du Sommeil, Hôpital Robert Debré, Paris, FRANCE, ⁴Department of Biomedical and Neuromotor Sciences, University of Bologna, Bologna, ITALY, ⁵Department of Neurology, Boston Children's Hospital, Boston, MA, ⁶Department of Pediatrics, Duke University Medical Center, Durham, NC, ⁷Stanford Center for Sleep Sciences and Medicine, Palo Alto, CA, ⁸Jazz Pharmaceuticals, Inc., Philadelphia, PA, ⁹Jazz Pharmaceuticals, Inc., Palo Alto, CA, ¹⁰Division of Pediatric Pulmonology and Sleep Medicine, University Hospitals Cleveland Medical Center, Rainbow Babies & Children's Hospital, Cleveland, OH.

Introduction: Obesity is a common comorbidity of pediatric narcolepsy. SXB is a standard of care for cataplexy and excessive daytime sleepiness in narcolepsy. BMI decreases have been observed with SXB treatment. We examined BMI changes by BMI percentile category at study entry in pediatric participants.

Methods: Participants were aged 7-17 years with narcolepsy with cataplexy. SXB-naive participants were titrated to an optimal SXB dose, then entered a 2-week stable-dose period; participants taking SXB at study entry entered a 3-week stable-dose period. After a 2-week, placebo-controlled, double-blind, randomized-withdrawal period, all participants entered an open-label safety period (total study duration: <1 year). Weight categories were defined using BMI percentiles at study entry based on growth charts from the Centers for Disease Control. BMI percentile was categorized as: underweight (<5%ile), normal (\geq 5%ile to <85%ile), overweight (\geq 85%ile) to <95%ile).

Results: Among SXB-naive participants, median (Q1, Q3) BMI percentile decreased with SXB treatment in participants who were categorized as normal-weight and overweight/obese at baseline (normal-weight, n=16: 76.5 [57.8, 82.4] at baseline, 35.0 [20.5, 62.6]

at week 52; overweight/obese, n=35: 97.6 [93.6, 99.1] at baseline, 86.7 [72.5, 97.9] at week 52). Of participants who were normal-weight at baseline, 15/16 remained normal-weight at week 52. Of participants who were overweight at baseline, 9/10 were normal-weight at week 52. Of participants who were obese at baseline, 7/25 were normal-weight, 3/25 were overweight, and 15/25 remained obese at week 52. Among participants taking SXB at study entry, BMI percentile decreased, but to a lesser degree. Decreased weight or weight loss was reported as an adverse event in 13 participants (11 SXB-naive; 1 participant withdrew). Four participants became underweight during the study but returned to normal-weight by study end.

Conclusion: Decreases in BMI percentile and category were observed with SXB treatment in pediatric participants with narcolepsy.

Support: Jazz Pharmaceuticals

0951

SLEEP STABILIZATION PATTERNS DEFINE PEDIATRIC CNS HYPERSOMNIA CONDITIONS

Maski, K. P.¹ Colclasure, A.² Little, E.¹ Steinhart, E.¹ Scammell, T.³ Navidi, W.² Diniz Behn, C.²

¹Boston Children's Hospital, Boston, MA, ²Department of Applied Math & Statistics, Colorado School of Mines, Golden, CO, ³Beth Israel Deaconess Medical Center, Boston, MA.

Introduction: Narcolepsy type 1 (NT1) is caused by loss of hypocretins, neuropeptides that promote consolidated nocturnal sleep and sustain daytime wakefulness. In mouse models of NT1, sleep in the light period is characterized by more brief wake bouts, fewer long wake bouts, and longer REM sleep bouts. It is unknown if this sleep pattern is present in NT1 patients and whether it can distinguish NT1 sleep from other CNS hypersomnia conditions [narcolepsy type 2 (NT2) and idiopathic hypersomnia (IH)].

Methods: Participants (6-18 years of age, drug -naïve or drug free) had diagnostic PSG/MSLT testing at Boston Children's Hospital between 2009-2018. PSG records were rescored blinded to diagnosis. We calculated Kaplan Meier survival curves for nocturnal wake and sleep stages extracted from the nocturnal PSGs. To adjust for differences in survival related to age, sex, and race, we used Cox proportional hazards models. In total, we performed survival analysis and compared wake/sleep stages for 4 groups: NT1 (n=46), NT2 (n=12), IH (n=18) and subjective sleepy controls (n=48).

Results: NT1 patients had worse survival of wake bouts compared to controls (p<0.001). In addition, NT1 patients had decreased survival of both NREM 2 and REM sleep bouts compared to all groups (all p<0.001), and, the survival of REM sleep bouts decreased with age (p=0.006). Compared to controls, NREM 2 bouts survived longer in IH patients and whereas NREM 1 bouts survived longer in the NT2 group (p's<0.006). There were no group effects for NREM 3, but survival of NREM 3 was less in the older IH patients compared to older controls (p<0.02).

Conclusion: Pediatric NT1 patients have unique sleep fragmentation characterized by unstable wake, NREM 2 and REM bouts. Though less severe as NT1, NT2 patients sustain lighter sleep. In contrast, IH patients show overly stable NREM stage 2 sleep and plausibly this contributes to their characteristic sleep inertia. Further research is needed to determine if sleep stability patterns can be used to diagnose CNS hypersomnia conditions and differentiate treatment responsiveness. Support: K23 National Institutes of Health (NINDS, K23 NS104267-01A1) and Investigator Initiated Research grants from Jazz Pharmaceuticals, Inc. (Dr. Maski)

0952

PARENT-CHILD PERCEPTIONS ABOUT HEALTHY SLEEP **PROMOTION IN A MOBILE HEALTH SLEEP EXTENSION INTERVENTION**

Mitchell, J. A.^{1,2} Eck, C.¹ Hickey, J.¹ Huffnagle, N.¹ Fiks, A. G.^{1,2} Zemel, B. S.^{1,2} Dinges, D. F.² Williamson, A. A.^{1,2}

¹Children's Hospital of Philadelphia, Philadelphia, PA, ²Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA. ³Children's Hospital of Philadelphia, Philadelphia, PA.

Introduction: Over half of school-aged children sleep insufficiently and sleep promoting interventions are urgently needed. To effectively promote pediatric sleep health, it is critical to obtain feedback from youth and their families about acceptable intervention strategies. This qualitative study examined perceptions about healthy sleep promotion in parent-child dyads participating in a mobile health sleep extension intervention.

Methods: A total of 26 parent-child dyads (child mean age 11 years, SD = 0.67; 46% non-Latinx White; 19% Black) participated in a mobile intervention to extend child sleep duration over 11 weeks (2-week baseline; 7-week intervention; 2-week follow-up). Participants wore a FitBit during the study, were provided with a sleep duration goal, and received general sleep health-promoting electronic messages using the University of Pennsylvania's Way to Health platform. Following the intervention, parents and their children separately completed a semistructured telephone interview to capture perceptions of the intervention strategies. Three coders developed a codebook using an inductive approach to identify emergent themes and conducted coding in NVivo.

Results: Emergent themes fell into domains of intervention acceptability/feasibility and barriers. Mobile messaging about the child's sleep duration goal was well-received, although child participants in particular desired more personalized messaging, with sleep promotion targeted to their specific sleep habits. Parents and children both discussed ancillary benefits to intervention participation, including an enhanced focus on sleep. Barriers to sleep extension during the intervention and maintenance of any gains post-intervention were related to: competing child academic, social, and extracurricular demands; family factors (work schedules; family rules and norms); and the challenges of limiting ubiquitous electronic devices.

Conclusion: Despite high parent-child acceptability of a mobile child sleep extension intervention, individual and contextual barriers may limit long-term adherence. Tailoring healthy sleep messages to target these factors could improve sustained benefits to child sleep.

Support: Sleep Research Society Foundation and K23HD094905 (AAW); NIH/NCATS UL1TR001878 (JAM and DFD) and K01HL123612 (JAM).

0953

THE MAINTENANCE OF WAKEFULNESS TEST (MWT) IN PEDIATRIC HYPERSOMNIA

Jaroenying, R.¹ Hantragool, S.² Xu, Y.³ Hossain, M. M.³ Simakajornboon, N.4

¹Department of pediatrics, Phramongkutklao Hospital, Bangkok, THAILAND, ²Department of pediatrics, Chulalongkorn Hospital, Bangkok, THAILAND, ³Division of Biostatistics and Epidemiology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH, ⁴Division of Pulmonary and Sleep Medicine, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.

Introduction: The Maintenance of Wakefulness Test (MWT) is a recommended procedure to evaluate the efficacy of treatment for hypersomnia. Limited data have been published on the use of the MWT in children and adolescents. This study aims to describe the clinical characteristics, MWT findings and their implication in the management of children with hypersomnia.

Methods: This study reviewed the charts of children with hypersomnia who had MWT performed at Cincinnati Children's Hospital Medical Center (CCHMC) between January 1, 2007 and January 31, 2018. Demographics, clinical characteristics, MWT findings, diagnoses and managements of children with hypersomnia were obtained.

Results: Fifty-three patients with hypersomnia who had MWT were included (mean age 17.29 years, range 12.5-22.75 years), 32 (60%) were male, and 40 (75%) were Caucasian. The diagnosis included narcolepsy (41, 77%), idiopathic hypersomnia (7, 13.2%), narcolepsy with OSA (11, 20%) and OSA (4, 7.5%). A mean sleep latency for all studies was 23.24 minutes (range 1.25-40 minutes). Twenty seven (50.9%) patients had mean sleep latency >20 minutes (passed MWT) which indicate adequate control of treatment, while 26 (49.1%) had mean sleep latency <20 minutes (failed MWT) including 5 (9.4%) with mean sleep latency <8 minutes. There was no difference between patients who had passed MWT and failed MWT in the mean of Epworth sleepiness scale (12 vs 11), age (16.8 vs 17.6 years), or BMI (29.2 vs 26.3). Higher percentage of narcolepsy with cataplexy was found in patients who failed MWT (46.2% vs 22.2%, P 0.06). Findings from the MWT caused the changes of management in 25/26 (96.1%) who failed MWT, and 8/27 (29.6%) who passed MWT (P<0.001).

Conclusion: Our result suggests that the MWT has clinical usefulness in evaluating responses to treatment for conditions associated with hypersomnia in children. Changes in management occurred in almost all patient who failed MWT. Interesting, there was no difference in subjective sleepiness between adolescents who passed and failed MWT, indicating the need to obtain objective data in this population. Future study is required to explore normative MWT data in pediatric population and to compare MWT with other tools such as driving simulation test. Support: None

0954

SLEEP OF GIFTED CHILDREN USING ACTIGRAPHY

Bastien, L.^{1,2} Théoret, R.^{1,2} Godbout, R.^{1,2}

¹University of Montreal, Montreal, QC, CANADA, ²Sleep Laboratory & Clinic, Hôpital en santé mentale Rivière-des-Prairies, CIUSSS du Nord-de-l'Île-de-Montréal, Montreal, QC, CANADA.

Introduction: Intellectual giftedness is characterized by an intellectual development superior to peers (QI > 120) while emotional and relational development corresponds to the age norms. Anecdotal reports from parents suggest that they sleep poorly compared to typically developing (TD) peers. We measured sleep of gifted children using actigraphy.

Methods: Thirteen gifted children (10 boys, mean age = 10.58, SD = 2.11) were studied. Giftedness was identified using Renzulli's three-factor definition of giftedness conceptualise in terms of above-average ability and high levels of task commitment (refined or focused form of motivation), and creativity. Sleep was measured with actigraphy for two weeks and compared to normative data from TD children using T-tests.

Results: Compared to normative data from TD children, gifted children had a significantly shorter sleep latency (p < 0.001), longer sleep periods (p = 0.001), shorter total sleep time and more wake time after sleep onset (p = 0.03). These differences were present both on week nights and weekend nights except that total sleep time was shorter in gifted children only during weekends (p < 0.001).

Conclusion: These data suggest that gifted children sleep poorly, and more so upon weekends. Whether this correlates with daytime functioning remains to be determined.

Support: N/A

0955

DOES PRIMARY CAREGIVER INFLUENCE SLEEP IN PRESCHOOLERS? EVIDENCE FROM A LARGE-SCALE LONGITUDINAL STUDY IN CHINA

Li, W.^{1,2} Wang, G.^{1,2} Zhang, Y.² Zhao, J.^{1,2} Jiang, F.^{1,2} ¹Department of Developmental and Behavioral Pediatrics, Shanghai Children's Medical Center, Shanghai Jiao Tong University School of Medicine, Shanghai, CHINA, ²Ministry of Education-Shanghai Key Laboratory of Children's Environmental Health, Shanghai, CHINA.

Introduction: The aim of the study is to characterize sleep disturbances and sleep patterns in preschoolers, and to examine the potential role of primary caregivers and particularly its transition on children's sleep in a large-scale cohort study in China.

Methods: A representative sample of 20324 newly enrolled preschoolers(age 3-4 years) were recruited in Shanghai, China at 2016.11. Sleep disturbances were examined at 3 timepoints from age 3-4 years to 5-6 years by the Children's Sleep Habits Questionnaire(CSHQ). Data on bedtime, onset time and offset time were included, along with primary caregivers derived from each follow-up. Prevalence and trend estimates of sleep disturbances and sleep patterns were presented. Multivariable logistic regression and propensity score-adjusted models for sleep disturbances at wave3 on different caregiving models were analysed adjusting for demographics, socioeconomic status, family characteristics, maternal mental health and CSHQ total score at baseline.

Results: Prevalence of global sleep disturbances (CSHQ total score>41) steadily fell from 84.2% to 73.8%. Specifically, behavioral sleep problem domains of "Bedtime besistance"and "Sleep anxiety" decreased from 66.4% to 47.3% and 53.4% to 37.8%, respectively. Meanwhile, Bedtime gradually delayed from 21:04(SD=0:37) to 21:20(SD=0:33) while offset time changed slightly from 7:13(SD=0:29) to 7:17(SD=0:27). Compared with counterparts in western countries, children slept for a shorter nocturnal sleep duration (NSD) with 9.51(SD=0.57) hours at wave1, 9.42(SD=0.57) hours at wave2 and 9.42(SD=0.52) hours at wave3. Different caregiving models were independently associated with sleep disturbances at wave3. Taking "Predominant parental care" as reference, "Predominant grandparental care" were associated with higher

odds of sleep disturbances (OR=1.32, 95% CI [1.17, 1.48], p<0.001 and OR=1.25, 95% CI [1.03, 1.52], p=0.020, respectively). While "Transition from grandparental to parental care" has no significant effect(OR=1.04, 95% CI [0.94, 1.15], p=0.436).

Conclusion: Grandparental care is associated with increased risk of sleep disturbances among preschoolers in China. Parenting and Grandparenting factors should be considered when assessing and treating sleep disturbances and optimizeing sleep health in preschoolers.

Support: Supported by the Chinese National Natural Science Foundation of China (81773443, 81728017, 81728017); Ministry of Science and Technology (2016YFC1305203); Shanghai Science and Technology Commission (17XD1402800, 18695840200, 2018SHZDZX05, 18JC1420305);

0956

VITAMIN D AND SLEEP IN CHILDREN

Al-Shawwa, B.¹ Ehsan, Z.² Ingram, D. G.² ¹Children's Mercy Hospital, kansas City, KS, ²Children's Mercy Hospital, Kansas City, MO.

Introduction: The impact of vitamin D on human health including sleep has been well described in adults. Its deficiency has been associated with multiple sleep disorders such as decrease in sleep duration, worsening of sleep quality and even obstructive sleep apnea. Such correlation is less evident in pediatric population. In the current study, we examined the relationship between sleep architecture and vitamin D status in children referred to a sleep clinic.

Methods: Retrospective-cohort study in a tertiary care children's hospital over a one-year period. Children who underwent an in-laboratory-overnight-polysomnogram and had a 25-hydroxy vitamin D level (25-OH-vitD) obtained within 120 days of the sleep study were included. Patients with obstructive or central sleep apnea were excluded. Data from polysomnograms (PSG) and Pediatric Sleep Questionnaires (PSQ) were collected and analyzed. Results: A total of 39 patients were included in the study with mean age of 6.6 years and 46% females. Twenty (51%) patients had vitamin D deficiency (25-OH-vitD less than 30 ng/ml). Children with vitamin D deficiency had less total sleep time (470.3 minutes +/-35.6 vs 420.3 minutes +/-61.7, p=0.004) and poorer sleep efficiency (91.9 % +/-5.6 vs 84.5 % +/-9.5, p=0.015) compared to vitamin D sufficient children. In addition, vitamin D deficient children had later weekday bedtimes (21:02 +/- 1:01 vs 20:19 +/- 0:55, p=0.037) and later weekend bedtimes (21:42 +/- 0:59 vs 20:47 +/-1:08, p=0.016) with tendency for later wake up time that did not reach statistical significance. The remainder of polysomnographic findings and PSO data were not different between the two groups. Conclusion: Vitamin D deficiency in children is associated with objectively measured decreased sleep duration and poorer sleep efficiency. Furthermore, vitamin D deficiency was associated with delayed bedtimes, suggesting that vitamin D may influence circadian rhythm. Future prospective studies in children would be helpful in validating the effect of vitamin D on sleep. Support: None

0957

THE ASSOCIATION BETWEEN SLEEP AND SUSTAINED ATTENTION DIFFERS IN CHILDREN VS. ADOLESCENTS WITH ADHD

Gagnon, K.^{1,2} Theoret, R.^{1,3} Rudd, E.^{1,3} Lepage, C.^{1,3} Chirica, A.^{1,3} Godbout, R.^{1,2}

¹Sleep Laboratory & Clinic, Hôpital Rivière-des-Prairies, Montréal, QC, CANADA, ²Department of Psychiatry, Université de Montréal, Montreal, QC, CANADA, ³Departement of Psychology, Université de Montréal, Montreal, QC, CANADA.

Introduction: Sleep disturbance in children with attention-deficit/ hyperactivity disorder (ADHD) is frequent, and lead to shorter sleep duration which has been associated with lower performance on sustained attention tasks. However, no study has investigated this association in adolescents with ADHD. We sought to explore whether the association between sleep and sustained attention performance of children with ADHD is similar in adolescents with ADHD given that sleep patterns are different.

Methods: Parents of 32 children (mean age = 8.0; SD = 1.3) and 10 adolescents (mean = 15.2; SD = 1.3) with ADHD completed a developmental questionnaire including sleep questions. Children and adolescents were medication free and underwent a comprehensive neuropsychological evaluation. Three sleep variables were extracted from the questionnaire, namely the duration of the sleep period during week nights and weekends as well as the difference between the two ("weekend shift"). The Continuous Performance Test was used to measure sustained attention (omission, commission, hit reaction time). Pearson correlations between sleep variables and sustained attention measures were calculated.

Results: Children showed a positive correlation between hit reaction time and the duration of the sleep period during week nights (r = 0.37; p = 0.04), weekends (r = 0.51; p = 0.004) and the weekend shift (r = 0.37; p = 0.04). No significant correlations were found in the adolescent group.

Conclusion: The fact that no significant associations were found in the adolescent group suggest an improvement of the arousal system through brain development in ADHD, or that other mechanisms could be involved in the etiology of ADHD in adolescents. **Support:** Centre d'apprentissage aux 1001 astuces; Fonds de recherche du Québec - Santé

0958

THE PREVALENCE OF DEPRESSION IN CHILDREN WITH CHRONIC SLEEP AND RESPIRATORY DISORDERS

Thakkar, N.¹ Trivedi, R.² Greenberg, P.² RAMAGOPAL, M.¹ ¹RUTGERS-ROBERT WOOD JOHNSON MEDICAL SCHOOL, NEW BRUNSWICK, NJ, ²Biostatistics and Epidemiology Services Center, Rutgers School of Public Health, Piscataway, NJ, ³RUTGERS-ROBERT WOOD JOHNSON MEDICAL SCHOOL, NEW BRUNSWICK, NJ.

Introduction: In 2017, 13.3% of US adolescents aged 12 to 17 had at least one major depressive episode (NIMH, 2019). The risk of depression is higher in children with sleep apnea, and children with depression or anxiety have a 1.2-1.8 times higher rate of asthma related ED visits. Therefore, the purpose of this study was to determine the prevalence of depression symptoms in children with chronic disorders. We hypothesized that subjects with sleep disorders would have a higher prevalence of positive depression scores than subjects with respiratory disease, and that the prevalence would be highest in subjects with both conditions.

Methods: The Patient Health Questionnaire-9 (PHQ-9) was administered to children between the ages of 12 and 18 to screen for depression as part of a pulmonary or sleep clinic visit. Each patient's PHQ-9 results were scored as normal, mild, moderate, or severe for levels of depression severity. Additionally, a chart review was conducted to gather their demographic and clinical data.

Results: Of a total of 87 subjects,71 (81.6%) had a respiratory disorder and 40 (46.0%) subjects had a sleep disorder. Due to the amount of overlap of respiratory and sleep disorders amongst the subjects, depression severity rather than chronic disease was chosen as the primary outcome. Using multiple linear regression, when all other factors were held constant, the average depression score increased by 5.0 when patients had a combination of asthma and obstructive sleep apnea (p-value = 0.02) and also increased by 2.4 for subjects who were female (p-value = 0.01).

Conclusion: It is important to identify depression in children with chronic illness, as it can lead to higher healthcare utilization. Additionally, as mental health status may significantly impact health outcomes for patients with chronic disease, it would be beneficial to evaluate mental health in all pediatric patients with chronic disease. However, further research is needed to investigate these associations.

Support: None

0959

ASSOCIATIONS BETWEEN SLEEP AND ACADEMIC PERFORMANCE IN TYPICALLY DEVELOPING ADOLESCENT GIRLS

Gruber, R.¹ Lin, L.² Santisteban, J.² Boursier, J.³ Somerville, G.⁴ ¹McGill Univesrity, Montreal, QC, CANADA, ²Attention Behavior and Sleep Lab, Douglas Mental Health University Institute, Montreal, QC, CANADA, ³Heritage Regional High School, Saint Hubert, QC, CANADA, ⁴Riverside School Board, Montreal, QC, CANADA.

Introduction: Healthy sleep promotes cognitive functioning and is associated with better academic performance, whereas insufficient, poor, and inconsistent sleep schedules are associated with poor school performance. Several studies have identified gender differences in the timing, duration, and quality of sleep in adolescence, with adolescent girls having poorer sleep compared to adolescent boys, yet research shows that girls outperform boys academically. This could be because sleep might not affect all subjects similarly and previous studies regarding sleep and academic performance combined both genders. The goal of the present study was to determine which aspects of academic performance are specifically associated with short or poor sleep in typically developing adolescents girls.

Methods: 80 adolescent girls aged 12-17 years participated in the study. Sleep was assessed in the home environment for five consecutive weeknights using actigraphy. Academic performance was assessed using report card grades.

Results: Hierarchical regression analyses adjusted for age, pubertal status, and socioeconomic status revealed that longer average sleep time was significantly associated with higher grades in mathematics. No significant associations were found between sleep variables and grades in other subjects.

Conclusion: Longer average weekday sleep duration is associated with better mathematics grades in typically developing adolescent girls.

Support: NSERC grant to Reut Gruber

0960

PERCEPTION OF SLEEP IMPORTANCE IN CHILDREN WITH CYSTIC FIBROSIS

Reiling, K. Patel, A. Baylor College of Medicine, Houston, TX.

Introduction: Children with cystic fibrosis (CF) are known to have poor sleep efficiency and increased nighttime awakenings secondary to factors such as nocturnal cough and time spent on therapies for airway clearance. Studies have shown that children with poor lung function have a higher Pediatric Daytime Sleepiness Score (PDSS). An increase in sleep disturbance has been associated with poorer perceived health in children with CF. There have been limited studies to date that report the perception of sleep importance in CF patients. We aim to characterize the perception of sleep importance in children with CF as measured through a self-reported questionnaire and identify barriers to sleep.

Methods: After IRB approval, subjects with CF aged 3-17 years were prospectively recruited from routine pulmonology clinic visits (n=28, 17 male). A questionnaire was provided consisting of 35 questions regarding sleep practices, perception of sleep importance, and PDSS. Recent pulmonary function tests (PFTs) were also collected.

Results: The mean PDSS was 11.3, with a range of 4 to 24. The questionnaire responses were as follows: 82% of participants reported sleep as "very important" overall, 92% reported sleep being "very important" for health, and 75% reported sleep being "very important" for lung function. In addition, 39% reported airway clearance as part of their nighttime routine and 89% reported utilizing electronic screens 2 hours prior to bed. The most frequent barriers to sleep identified were technology and bedtime resistance (14% each), and homework, excitability, and vest/airway treatments (11% each). 86% of participants had at least one symptom of disordered sleep.

Conclusion: Screening for sleep problems in the CF population may be beneficial and may contribute to improved quality of life. Further recruitment is ongoing. **Support:**

0961

COMPARISON OF CLINICAL CHARACTERISTICS OF CHILDREN WITH NARCOLEPSY WITH AND WITHOUT SLEEP RELATED MOVEMENT DISORDERS

McIntyre, E. A. Oles, S. K. Bandyopadhyay, A. Daftary, A. Indiana University School of Medicine, Division of Pediatric Pulmonology, Allergy and Sleep Medicine, Indianapolis, IN.

Introduction: Narcolepsy is known to be associated with sleep related movement disorders (SRMD) including periodic limb movement and restless leg syndrome. However, there is paucity of data comparing the clinical characteristics of children with narcolepsy with and without SRMD.

Methods: Retrospective chart review of all children presenting to the sleep clinic for sleep problems between March 2016 to June 2017 was performed. Demographics, sleep intake patient questionnaires and ICD-10 codes for comorbidities and sleep diagnoses were collected. Children with diagnosis of narcolepsy (ICD-10 G47.4) were included in this study. Cohort was divided into 2 groups- with and without co-existing diagnoses of sleep related movement disorders (ICD-10 G47.6). Demographics, presenting symptoms, Epworth sleepiness scores and prevalence of sleep comorbidities were compared using T-test (continuous) and Chi square (categorical). Unadjusted odds ratio was calculated for demographics and presence of SRMD. P value of <0.05 was considered significant. **Results:** 28 (F=14,50%) children with narcolepsy were included. 25% children were diagnosed with SRMD. Mean (SD) age of children with SRMD presenting to the sleep clinic was 11.14 (5.08) years while mean age (SD) of children without SRMD was 9.52 (3.87) years. Age and race of children with and without SRMD were not statistically different. There was an increased prevalence of females in the group with narcolepsy and SRMD compared to the group without SRMD (86%vs38%, p=0.029). Epworth sleepiness score was not statistically different between the 2 groups. Female gender increased the odds of SRMD in children with narcolepsy (OR:9.75, 0.98-96.56).

Conclusion: Children with narcolepsy can present with comorbid sleep related movement disorder. Females were more likely to present with associated SRMD compared to males. **Support:** None

Support. Non

0962

SLEEP AND DAYTIME FUNCTIONING IN GIFTED AND TWICE EXCEPTIONAL CHILDREN

Théoret, R.^{1,2} Bastien, L.^{1,2} Godbout, R.^{1,3}

¹Sleep Laboratory & Clinic, Hôpital en santé mentale Rivière-des-Prairies, Montréal, QC, CANADA, ²Department of Psychology, Université de Montréal, Montréal, QC, CANADA, ³Department of Psychiatry, Université de Montréal, Montréal, QC, CANADA.

Introduction: Gifted (G) children display an asynchrony between intellectual development and social and emotional development. Twice exceptional (2e) children are G children with a neuropsychological disability. We compared the sleep and daytime behavior of G, 2e and typically developing (TD) children and we sought for group-specific relationships between sleep and daytime behavior.

Methods: 23 children were recruited: seven G (8.7 years old, SD = 1.7), six 2e (9.8 years old, SD = 1.8) and 10 TD children (10.0 years old, SD = 2.2). Giftedness was diagnosed with neuropsychological tools. The Children's Sleep Habits Questionnaire (CSHQ) assessed sleep quality, the Child Behavior Checklist (CBCL) assessed daytime functioning. Sleep quality and its impact on daytime functioning was measured with a MANCOVA, with the CBCL's three main factors as dependent variables (internalizing problems, IP; externalizing problems, EP and total problems, TP), children group as the independent variable and the CSHQ total score as the covariate.

Results: G, 2e and TD groups scored 39.86, 39.17 and 39.70 on the CSHQ, respectively (n.s.). The three groups were not different on the CBCL, with respective mean T scores of 57.86, 50.33 and 48.60 for IP, 56.43, 55.67 and 47.80 for EP and 55.29, 53.83 and 46.40 for TP. Pillai's trace statistics disclosed a significant relationship between CSHQ and CBCL scores regardless of groups (p = 0.04) but the influence of sleep quality did not differ among the groups for any of the three factors. The CSHQ total score was positively and significantly related to IP (p = 0.03, r = 0.47); relationships were not significant for EP (p = 0.96, r = -0.01) and TP (p = 0.17, r = 0.31).

Conclusion: Sleep quality influences internalizing problems in children, without group-specific relationships, but this association does not seem to differ between gifted, twice exceptional and TD children.

Support: N/A

SLEEP, Volume 43, Abstract Supplement, 2020

0963

PERCEIVED SLEEP NEED IN ADOLESCENTS AND ACCEPTABILITY OF A MORNING BRIGHT LIGHT INTERVENTION

Culnan, E.¹ Eastman, C. I.² Crowley, S. J.²

¹Rush University Medical Center, Department of Psychiatry and Behavioral Sciences, Sleep Disorders Service and Research Center, Chicago, IL, ²Rush University Medical Center, Department of Psychiatry and Behavioral Sciences, Biological Rhythms Research Laboratory, Chicago, IL.

Introduction: We are testing strategies to make fall asleep times earlier to extend sleep duration of sleep-deprived, delayed adolescents. We explored whether perceived sleep need was associated with the acceptability of an intervention which included earlier bedtimes on school nights and early morning bright light (BL) on one weekend.

Methods: Twenty-three participants (16.0±0.7 years; N=11 female) from the intervention group of a study of adolescents reporting ≤7 h sleep on school nights and late bedtimes (school-night≥23:00; non-school night≥midnight) were included. Participants estimated nightly sleep need during baseline. The intervention included shifting school-night bedtimes 1h earlier than each participant's usual school night bedtime during the first week and 2h earlier than baseline during the second week. Evening time management goals were used to help participants get to bed earlier. Wake times remained stable because of early school start times. During the weekend in between these 2 weeks, participants lived in the laboratory and received very early morning BL on Saturday and Sunday from two light boxes (~6000 lux; three 50-minute exposures with 10-minute breaks). At the end of the study, an acceptability questionnaire asked about hypothetically engaging in different elements of the intervention (earlier bedtimes, morning BL) at home. Items were summed to create a composite acceptability score.

Results: Participants reported needing 7.8 h (SD=1.2; range=5-9.5h) of sleep. A linear regression demonstrated that needing more sleep was associated with less acceptability when controlling for age and sex, b=1.37, SE_b=.60, p=.03.

Conclusion: Adolescents who felt they needed less sleep were more accepting of an intervention that required earlier bedtimes on school nights and waking up very early to receive BL on the weekend. Interventions that produce less weekend sleep deprivation, like including naps, might be more acceptable to all. **Support:** R01HL105395 (S.J.C.)

0964

OUT LIKE A LIGHT: PRELIMINARY RESULTS OF PARENT-CHILD DYAD USE OF AN AUDIO-BASED MOBILE APPLICATION AIDING BEDTIME ROUTINE AND SLEEP HEALTH

*Chung, A.*¹ *Chanko, N.*¹ *Blanc, J.*¹ *Donley, T.*¹ *Robbins, R.*² *Brotman, L.*¹ *Jean-Louis, G.*¹

¹NYU Grossman School of Medicine, New York, NY, ²Harvard University, Cambridge, MA.

Introduction: Adequate sleep is essential for a child's growth and development. However, a growing number of children are experiencing trouble falling asleep. Smartphone audio-based mobile

applications with soothing melodies and calming nighttime stories may improve sleep onset. Our study examined the efficacy of Moshi Twilight, an app designed to improve sleep onset, among children ages 3-8 years old using a parent-child dyadic approach. **Methods:** Our within-subjects pre-post study design focused on healthy children studied over 10 days, spanning 3 weeknights and 2 weekend nights. During the baseline (Days 1-5) and exposure (Days 6-10) conditions the Child Sleep Health Questionnaire was used to measure children's sleep behavior. The PROMIS and Pittsburgh Sleep Quality Index were used to assess parents' sleep quality. Parents exposed their child to 1 story per night (15-20 minutes) during the exposured condition. Statistical analysis was based on paired t-tests, independent t-tests, and correlations.

Results: On average, participating parents were 37 (SD +9.6) year-old mothers. The sample was: 60% Black; 20% White, 20% other race/ ethnicity. On average, children were 4 (SD + 0.78) years old and 50% male. Paired t-tests showed significant differences in children's sleep onset within 20 minutes (t=2.582, 95% CI 0.116, 2.634, p= 0.036). Significant correlations were noted for children's bedtime consistency (r = -0.755, p = 0.030), falling asleep in own bed (r = 0.735, p=0.015) and sleep duration (r = -0.715, p=0.046,) Significant correlations and paired t-test in parents' sleep onset were also found (r = 0.744, p = 0.014); (mean= -1.2, t= -3.674, 95% CI -1.939, -0.461, p=0.005) **Conclusion:** Our results showed that the audio-based sleep app, Moshi Twilight, might be useful in improving sleep health among both children and parents. This could be included in enhance bed-time routine among preschool-aged children.

Support: Bezos Grant and Community Service Plan grant.

0965

THE ASSOCIATION BETWEEN LIQUID CONSUMPTION AND SLEEP PATTERNS IN SCHOOL CHILDREN

Raine, P. Wang, J. Pitt, S. University of Pennsylvania, Philadelphia, PA.

Introduction: Liquid consumption is essential for daily function and may also play a role in sleep regulation. The aim of this study was to assess 1) the association between the frequency of liquid consumption and sleep patterns; and 2) the different types of liquids on the association between liquid consumption and sleep.

Methods: Participants included 597 children ages 9-13 years old from the China Jintan Child Cohort Study. To assess child liquid intake, children self-reported the types of liquids consumed and the frequency these liquids were consumed. To assess sleep patterns, both parents and children reported sleep patterns using the parentreported Child Sleep Habit Questionnaire (CSHQ) and a child selfreported questionnaire. Descriptive statistics and independent sample t-tests analyses were performed to examine the differential effects of liquid consumption frequency and sleeping habits.

Results: Overall, a slight dose-dependent relationship between liquid consumption and sleep quality was observed. Less sleep problems and improved sleep quality were observed for water (bedtime resistance, sleep anxiety, night awakenings, parasomnias, sleep-disordered breathing, daytime sleepiness; p<0.05) and milk (parasomnias, sleep-disordered breathing, p<0.05) consumption. Caffeinated soda tended to increase sleep problems (sleep-disordered breathing, p<0.05). Sleep onset delay had a different pattern from that of other subscales, in which water increased sleep problems and caffeinated soda **Conclusion:** Children who consumed more liquid, especially water and milk, were more likely to experience less sleep problems. However, caffeinated soda consumption may be linked to increased sleep problems. Findings suggest that school children may need consume more healthy liquids for better sleep patterns. Future randomized-controlled trial studies are needed to verify these findings.

Support: This study was funded by the National Institutes of Environmental Health Sciences and the National Institutes of Health (R01-ES-018858, K02-ES-019878, and K01-ES015877).

0966

SLEEP-WAKE PATTERNS IN MOTHERS AND CHILDREN IN A RURAL COMMUNITY WITH LIMITED ACCESS TO ELECTRICITY: RESULTS FROM THE GHANA RANDOMIZED AIR POLLUTION AND HEALTH STUDY

*Kundel, V.*¹ *Darko, P.*² *Taweesedt, P.*³ *Parekh, A.*¹ *Ayappa, I.*¹ *Lee, A.*¹ *Darby, J.*⁴ *Kaali, S.*² *Asante, K.*²

¹Icahn School of Medicine at Mount Sinai, Division of Pulmonary, Critical Care and Sleep Medicine, New York, NY, ²Kintampo Health Research Centre, Ghana Health Service, Brong Ahafo Region, Kintampo, GHANA, ³Icahn School of Medicine at Mount Sinai, James J. Peters VA Medical Center, Department of Medicine, Bronx, NY, ⁴Mailman School of Public Health, Columbia University, New York, NY.

Introduction: Studies measuring objective sleep duration in rural/ indigenous populations are limited, showing sleep duration similar to that of industrialized countries. Little is known about sleep duration in women of reproductive age, and children within these populations. Our study is the first to objectively characterize sleep in mothers and children in an agrarian community with limited access to electricity, utilizing data from the Ghana Randomized Air Pollution and Heath Study (GRAPHS).

Methods: The GRAPHS cohort, a cluster-randomized trial, evaluated the efficacy of clean fuels on birthweight and infant pneumonia incidence in central Ghana. The study initially recruited pregnant women and newborns in 2013. This study is now utilizing wrist-actigraphy to analyze sleep-wake patterns among mothers and children of GRAPHS. We have thus far analyzed actigraphy in 39 mothers and 49 children (including 25 mother-child pairs), using the Motionlogger-MicroWatch with the Cole-Kripke algorithm to assess total sleep time (TST). We report baseline characteristics and sleep-wake patterns of our sample.

Results: Mean age of mothers was 33.5 years, (range 22-48), and mean age of children was 3.9 years (3-4). Average nights recorded were 4 (standard deviation [SD] 2.1). For mothers, average median time-in-bed was 7.9 (SD 1.2) hours, TST was 6.4 (SD .9) hours, and sleep efficiency was 82% (SD 7.9). Median bedtime was 9:33pm (SD 1.5 hours), and median wake-time was 5:56am (SD 1.4 hours). For children, average median time-in-bed was 9.9 (SD 1.0) hours, TST was 8.2 (SD 0.9) hours, and sleep efficiency was 83% (SD 6). Median bedtime was 8:03pm (SD 0.8 hours), and median wake-time was 6:06am (SD 0.6 hours). There was no correlation between sleep measures in mother-child pairs.

Conclusion: In an agrarian Ghanaian community with limited access to electricity, objective sleep measures in women were similar to prior studies conducted in indigenous/rural populations of developing African countries (Ndiaye et.al.2007,

Samson et.al.2016), though data in children is lacking for comparison. When compared with post-industrialized countries, objective sleep measures for this age group of non-gravid women are sparse. In toddlers however, TST was lower in our cohort when compared with objective sleep amongst toddlers in industrialized nations.

Support: 2K24HL109156-06A1

0967

CLINICAL CHARACTERISTICS OF CHILDREN WITH SLEEP PROBLEMS AND COMORBID PSYCHIATRIC DISORDERS

McIntyre, E. Oles, S. K. Walsh, K. Bandyopadhyay, A. Indiana University School of Medicine, Division of Pediatric Pulmonology, Allergy and Sleep Medicine, Indianapolis, IN.

Introduction: Anxiety and Attention Deficit Hyperactive Disorder (ADHD) are common psychiatric comorbidities in children with sleep disorders. It is known that comorbid psychiatric disorders increase the risk of sleep problems. However, no study has compared the clinical characteristics of children presenting with sleep problems and various common psychiatric disorders.

Methods: Retrospective chart review of all children presenting to the sleep clinic for sleep problems between March 2016 to June 2017 was performed. Demographics, sleep intake patient questionnaires, polysomnograms and ICD-9/10 codes for comorbidities and sleep diagnoses were collected. In children with diagnoses of anxiety (ICD-9 300/ICD-10 F41) and ADHD (ICD-9 314/ICD-10 F90), demographics, presenting symptoms, Epworth sleepiness scores and prevalence of sleep comorbidities were compared. T-test (continuous) and Chi Square (categorical) were used. Unadjusted odds ratio was calculated for presenting symptoms and sleep comorbidities. P value of <0.05 was considered significant.

Results: 250 (F=145, 58%) children were evaluated. 71.2% children were diagnosed with anxiety and 28.8% diagnosed with ADHD. Mean age at presentation was 8.53 ± 4.2 years. Age, gender and race of children presenting with sleep problems and comorbid anxiety/ADHD were statistically similar. Children with anxiety spent less time in stage N3 sleep ($25.2\% \pm 9.1$ versus $28.6\% \pm 9.2$) and had lower arousal indices (7.19 ± 3.8 versus 8.86 ± 5.5) compared to children with ADHD. Children with anxiety were more likely to present with chief complaint of "feeling tired or sleepy during the day" (OR:2.38, 1.32-4.37) and were more likely to have a diagnosis of hypersomnia (OR: 11.67, 3.19-42.75) versus children with ADHD.

Conclusion: Children with psychiatric comorbidities have distinct polysomnographic characteristics. Children with anxiety are more likely to present with daytime sleepiness and have a significantly higher prevalence of hypersomnia compared to children with ADHD.

Support: None

0968

SLEEP-DISORDERED BREATHING IN SCHAAF-YANG SYNDROME

Powell, W.¹ Rech, M.² Schaaf, C.³ Wrede, J.¹

¹Seattle Children's Hospital/University of Washington, Seattle, WA, ²The Menninger Clinic, Houston, TX, ³Institute of Human Genetics, Heidelberg University, Heidelberg, GERMANY, ⁴Seattle Children's Hospital/University of Washington, Seattle, WA. **Introduction:** Schaaf-Yang Syndrome (SYS) is a genetic disorder caused by truncating variants in the MAGEL2 gene located in the maternally imprinted, paternally expressed Prader-Willi syndrome (PWS) region at 15q11-13. The SYS phenotype shares features with PWS, a disorder with known high incidence of central and obstructive sleep apnea (OSA). However the spectrum of sleep-disordered breathing in SYS has not been described.

Methods: We performed a retrospective analysis of polysomnograms from 22 of the known 115 patients with molecular diagnosis of SYS. Sleep characteristics including total sleep time, latency, efficiency, % sleep stages, apnea-hypopnea index (AHI), obstructive index, central index, and oxygenation were analyzed for the whole group and by truncation location (c.1996dupC variants [n=11] or other locations [n=11]). Only the initial diagnostic study or initial diagnostic portion of a split-night study was used in analysis (analytic n=21).

Results: We collected 33 sleep study reports from 22 patients, ages 2 months - 18.5 years. Mean analyzed sleep time was 357 minutes (129-589 min) with mean sleep efficiency of 71.45% (45-94%) and sleep latency of 24.8 minutes (0-146 min). The mean apnea-hypopnea index (AHI) was 19.1/hr (0.9 -49/hr) with mean obstructive AHI of 16.3 (0.6-49/hr). Mean central index was 2.8/hr (0-14/hr). 18/21 (86%) were diagnosed with OSA, and 13/21 (62%) with moderate or severe OSA (oAHI >5/hr). Central sleep apnea was diagnosed in 2/21 (9.5%). 15 studies reported periodic limb movement index (PLMI) with mean of 7.8 (0-67/hr) and 4/15 (26%) with PLMI >5. Comparison of genotype groups did not reveal any difference in presence of OSA or severity of OSA.

Conclusion: OSA is frequently identified on polysomnography in patients with SYS. Central sleep apnea is less common, which is in contrast to PWS. The majority of patients with OSA had moderate or severe OSA, and 47% had severe OSA. **Support:** N/A

0969

EARLY LIFE SLEEP DISTURBANCE AMONG CHILDREN WITH AUTISM SPECTRUM DISORDERS: A QUESTIONNAIRE-BASED RETROSPECTIVE STUDY.

*Gupta, A.*¹ *Shukla, G.*¹ *Poornima, S.*¹ *Mohd, A.*¹ *Katoch, J.*¹ *Taneja, D.*² *Singhal, N.*²

¹Department of Neurology, All India Institute of Medical Sciences, New Delhi, New Delhi, INDIA, ²Action for Autism, New Delhi, INDIA.

Introduction: Autism spectrum disorders(ASD) and sleep has hand to hand relationship and few recently published studies have shown that, disturbed infant sleep is associated with Attention deficit hyperactive disorder and Autism. The aim of the study is to evaluate the sleep disturbance in first year of life among the children with ASD.

Methods: In this case control study, pre-diagnosed ASD children between 3-12 year of age (group 1) and controls; sibling of autistic children and community dwelling children(group 2), were enrolled in between June 2014 to November 2017. After giving consent every child underwent, Detailed Clinical [Childhood Autism rating scale-2(CARS-2)] and Sleep evaluation [Brief Infant sleep Questionnaire (BISQ) - Retrospectively, Child sleep habits questionnaire (CSHQ)]. **Results:** Sixty children in group1 and 60 in group 2 were enrolled and both groups were age [Median 7(2.5-12) vs. 8.5 (2.5-12), p = 0.14] and sex [53(88.33%) vs. 45(75.00 %), p=0.15] matched. On CHSQ poor sleepers are more common among the group 1[40(66.66%) vs. 20(33.33%), p=.003, respectively]. On BISQ children had severe sleep problems during infancy were significantly more among the group 1 [46(76.66%) vs. 6(10%), p=<.001, respectively]. Duration of nocturnal awakening was significantly correlated (r=0.87, p=.001) with the T score of the CARS-2.

Conclusion: Sleep is significantly disturbed since infancy among children with autism spectrum disorder and duration of nocturnal awakenings during infancy is strongly correlated with severity of autism.

Support: NA

0970

LONGITUDINAL STUDY OF INFANT SLEEP PROBLEMS AND INFLUENCING FACTORS

Zhou, Z.¹ Wang, N.²

¹Chongqing health center for women and children, Chongqing, CHINA, ²chongqing health center for women and children, Chongqing, CHINA.

Introduction: To investigate the sleep problems and influence factors in infants at the age of 1 month and 6 months through longitudinal questionnaire survey.

Methods: 600 healthy infants aged 1 month were selected from May to August 2014 from Chongqing health center for women and children. A self-made sleep questionnaire was used to investigate the infants's sleep at the age of 1 month and 6 months respectively. Logistic regression model was used to analyze the correlation between sleep problems and factors.

Results: The incidence of nigt-waking, difficulty in falling asleep and night crying at 1 month of age were 30%, 17% and 14.1%, respectively, and at 6 months were 7.3%, 9.3% and 13.1%, respectively. Results of the Logistic regression analysis model of sleep problems of infants at 1 month old were: 1. body length grade, baby touch, sleep alone, putting the infants back into bed after being picked up and quiet, night feeding were the protective factors of night-waking, while diaper changing is the risk factor; 2.body length grade, listening to music, orderly bedtime routine, comforting without holding and putting the infants back into bed after being picked up and quiet were protective factors for difficulty in falling asleep, while diaper changing is the risk factor; 3. listening to music, orderly bedtime routine, watching and waiting, holding until sleep were all protective factors of night-crying. Logistic regression model analysis results of sleep problems of infants aged 6 months were: 1. The risk factors of night-waking include putting the infants back into bed after being picked up and quiet, holding the infants until falling asleep, diapers changing, and parent-children interaction before bedtime. 2. Listening to music and sleeping alone in a bed were protective factors for difficulty in falling asleep. 3. Touching, orderly bedtime routine and comforting without holding are protective factors of night-crying.

Conclusion: Sleep problems in infants are common and decrease with age. The factors of sleep problems at the age of 1 and 6 months are different, Targeted prevention are beneficial to the prevention of infant sleep problems.

Support: Chinese center for disease control and prevention grant

0971

MOTHER'S PARENTING STYLE AS A MODERATOR IN THE RELATIONSHIP BETWEEN TEMPERAMENT AND SLEEP PROBLEMS IN CHILDREN WITH OBESITY

NAM, *H*.¹ *Kim*, *J*.¹ *Woo*, *S*.² *Park*, *S*.³ *Lee*, *H*.³ *Jang*, *H*.³ *Park*†, *K*.⁴ *Suh*†, *S*.¹

¹SUNGSHIN WOMEN'S UNIVERSITY, Seoul, KOREA, REPUBLIC OF, ²Department of Medical Science, Hallym University School of Medicine, Chuncheon, KOREA, REPUBLIC OF, ³Center for Biomedical Sciences, Korea National Institute of Health, Cheongju, Chungbuk, KOREA, REPUBLIC OF, ⁴Department of Family Medicine, Hallym University Sacred Heart Hospital, Hallym University School of Medicine, Anayng, KOREA, REPUBLIC OF.

Introduction: Based on past studies, a child's temperament and parenting style greatly affect a child's sleep. However, there are limited studies that have investigated how parenting styles and a child's temperament interact and affect the child's sleep in obese children. Thus, this study investigated parenting styles as a moderator in the relationship between temperament and sleep in obese children.

Methods: Seventy-seven obese children (male=66.2%, average BMI = 27.35 ± 2.78) participated in the study. The mean age of the participants was $10.82(\pm 1.00)$ years. The primary caregiver (mother) of the participants completed the Junior Temperament and Character Inventory (J-TCI), and Parents as Social Context Questionnaire (K-PSCQ), and Children's Sleep Habits Questionnaire (CSHQ). Among the different subscales, only Novelty Seeking (NS) and Reward Dependence (RD) were used for analysis among Junior Temperament and Character Inventory (J-TCI) components.

Results: In this sample, 66 children (85.7%) reported significant levels of sleep problems based on the CSHQ. Novelty seeking (NS) and reward dependence (RD) significantly predicted sleep problems (B=-.771, p<.05, B=-.683, p<.01). Additionally, mother's negative parenting style moderated the relationship between NS and the child's sleep problem [B=.03, 95% CI=.007, .049] and the relationship between RD and the child's sleep problem [B=.031, 95% CI=.013, .049]. The more negative mother's parenting style, the higher the child's NS or RD scores had a negative effect on sleep.

Conclusion: The results of this study show that obese children experience high levels of sleep disturbance. Additionally, the mother's negative parenting style moderated the relationship between temperament and sleep problems in obese children. The results suggest that sleep interventions for obese children should include the mother, especially in children with high novelty seeking and reward dependence.

Support: This work was supported by Korea Centers for Disease Control and Prevention & Korea National Research Institute of Health (2019020660E-00)

0972

PERSISTENCE, REMISSION, ACQUISITION OF SLEEP DISTURBANCES CONTRIBUTES TO THE TRANSITION OF EMOTIONAL/BEHAVIORAL PROBLEMS IN PRESCHOOL CHILDREN

Deng, Y.¹ Wang, G.¹ Li, W.¹ Zhang, Y.² Zhao, J.² Zhang, Z.² Jiang, F.¹

¹Department of Developmental and Behavioral Pediatrics, Shanghai Children's Medical Center, Shanghai Jiao Tong University School of Medicine, Shanghai, CHINA, ²Child Health Advocacy Institute, Shanghai Children's Medical Center, Shanghai Jiao Tong University School of Medicine, Shanghai, CHINA.

Introduction: Cross-sectional and longitudinal studies have consistently reported associations between sleep disturbances and emotional/behavioral problems in children. However, few studies have examined whether the remission, acquisition of sleep disturbances contribute to the transition of emotional and behavioral problems during preschool years.

Methods: This study used data from the Shanghai Children's Health, Education and Lifestyle Evaluation-Preschool (SCHEDULEA-P), a prospective, population-based cohort study of newly enrolled preschoolers in Shanghai kindergartens in Nov. 2016. In total, 17182 children with complete data on parentreported Strength and Difficulties Questionnaire (SDQ) both at school enrollment(wave 1) and the third year(wave 2) were included in the study. Children's sleep disturbances were measured using Children Sleep Habit Questionnaire (CSHQ) at both waves.

Results: The 17182 participants included 8935(52.0%) males, with a mean (SD) age of 3.73(0.29) years at wave 1. 66.9%, 7.2%, 17.4% and 8.5% of these children were divided into persistent sleep disturbance, acquired, remitted group and persistent normal sleep group, respectively. The proportion of persistent, acquired, remitted emotional/behavioral problems and normal group was 13.7%, 9.4%, 21.1%, 55.8%. SDQ scores of acquired sleep disturbances group stayed high at wave 3, while SDQ scores of remitted sleep disturbances group decreased sharply during the preschool years. After adjusted for confounding factors, the odds of remission from emotional/behavioral problems among children who experienced remission of sleep disturbances, who had persistent normal sleep were both much higher compared to those who had persistent sleep disturbances (OR=2.53(2.12-3.01), p<0.001; OR=2.74(2.01-3.75), p<0.001). Meanwhile, the odds of acquisition of emotional/behavioral problems at wave 2 among subjects who newly acquired sleep disturbances at wave 2 and who had persistent sleep disturbances was similarly higher than those who never have sleep disturbances(OR= 2.75, P<0.001 VS OR=2.77, P<0.001). Besides, those who experience remission of sleep disturbances still have 1.48 times the odds of acquisition of emotional / behavioral problems(P=0.006).

Conclusion: The remission of sleep disturbances contributed to the remission of emotional/behavioral problems, while the emergence of sleep disturbances throughout preschool years increased the risk of the acquisition of emotional/behavioral problems.

Support: Supported by the Chinese National Natural Science Foundation of China (81773443, 81728017, 81602870, 81601162, 81602868)

0973

MATERNAL DEPRESSION AND INFANT SLEEP DURATION TRAJECTORY IN THE FIRST 3 YEARS: A PROSPECTIVE COHORT STUDY.

Gui, Y. Wang, G. Deng, Y. Li, W. Jiang, F. Shanghai Children's Medical Center Affiliated to Shanghai Jiao Tong University School of Medicine, Shanghai, CHINA. **Introduction:** The study was to investigate trajectories of infant sleep duration and associations with trajectories of maternal depression status during 3 years post-partum.

Methods: Data were from the Child Health Promotion Project in Shanghai (CHPPS). Mothers were recruited at the third trimester of pregnancy and followed up together with the infants until 36 months postpartum. Between 2012 and 2013, 262 women (M_{age}=29.5, SD_{age}=3.2, range: 22-39 years old) were recruited and were followed from June 2012 to August 2015. Sleep duration of the children was assessed using Brief Infant Sleep Questionnaire (BISQ) at 42 days, 3 months, 6 months, 9 months, 12 months, 18 months, 24 months, and 36 months postpartum. Center for Epidemiological Survey-Depression Scale (CESD), Edinburgh Postnatal Depression Scale (EPDS), and the Profile of Mood States (POMS) were used to measure the mother's depression status at late pregnancy, 42 days postpartum, and 12-36 months postpartum, respectively. The group-based trajectory models (GBTM) were used to estimate patterns of infant sleep duration development and maternal depression status.

Results: Two trajectories of infant day sleep duration were identified, defined as "initial short sleepers" (54.8%) and "initial long sleepers" (45.2%). Three trajectories of infant night sleep duration were identified, labeled as "increasing" (8.5%), "stable" (61.7%), and "mild declining" (29.8%). Two trajectories infant total sleep duration were identified, defined as "initial short sleepers" (51.5%) and "initial long sleepers" (48.5%). Two trajectories of maternal depression status were identified, labeled as "low" (74.2%) and "high" (25.8%). After controlling for covariates, women who have higher depression status had infants of shorter day sleep duration. There was no significant association with infant night sleep duration.

Conclusion: Our study suggests that maternal postpartum depression is associated with short infant day sleep duration, but not with infant night sleep duration.

Support: Supported by the Chinese National Natural Science Foundation of China (81773443, 81728017, 81602870, 81601162, 81602868)

0974

FAMILY-BASED STUDY OF SLEEP IN AUTISM SPECTRUM DISORDER WITHOUT INTELLECTUAL DISABILITY

Elkhatib Smidt, S. D.^{1,2} Ghorai, A.² Gehringer, B.² Dow, H. C.² Smernoff, Z.² Taylor, S. C.² Zhang, J.² Rader, D. J.² Almasy, L.^{1,2} Brodkin, E. S.² Bucan, M.²

¹Children's Hospital of Philadelphia, Philadelphia, PA,

²Perelman School of Medicine at the University of Pennsylvania, Philadelphia, PA.

Introduction: Sleep problems are a common concern in children with autism spectrum disorder (ASD) that can persist into adulthood. This study aims to further explore sleep in ASD without intellectual disability (ASD w/o ID).

Methods: We recruited individuals with ASD w/o ID (probands) and relatives as part of the Autism Spectrum Program of Excellence (ASPE) at the University of Pennsylvania. Actimetry data were collected via a wrist-worn tri-axial accelerometer for 21 days. Data from 212 participants were considered. We analyzed sleep data using the algorithms GGIR, ChronoSapiens, and PennZzz. The sleep traits of proband and sibling pairs were compared using paired t-test or Wilcoxon signed-rank test. We used the Social Responsiveness Scale, Second Edition (SRS-2) to assess social impairment and restricted/repetitive traits. We compared SRS-2 scores to sleep traits using partial Spearman or Pearson correlations adjusting for age (171 participants).

Results: Probands demonstrated later sleep onset (p = 0.03), decreased M10 average (10-hour period of highest activity/ day; p = 0.006), decreased relative amplitude (measure of rest-activity rhythm; p<0.001), and decreased total daytime activity (p = 0.005) compared to siblings. Regarding social function and restricted/repetitive traits, adult males showed an inverse correlation between SRS-2 total score and sleep efficiency (r = -0.2, p = 0.04) and a positive correlation between SRS-2 total score and sleep efficiency (r = -0.2, p = 0.04) and a positive correlation between SRS-2 total score and intradaily variability (r = 0.3, p = 0.02). Adult females showed an inverse (r = -0.3, p = 0.02) and between SRS-2 total score and relative amplitude (self-report r = -0.4, p = 0.001; informant r = -0.3, p = 0.005).

Conclusion: This study focuses on the analysis of sleep traits in ASD including the relationship between social function and sleep. Thus far, the most robust findings are decreased daytime activity and relative amplitude in individuals with ASD w/o ID compared to siblings. We have also shown that ASD social impairment may be related to sleep dysfunction.

Support: NIH T32HL07713, anonymous donor, and the Institute for Translational Medicine and Therapeutics of the Perelman School of Medicine at the University of Pennsylvania.

0975

IRON INFUSION PROTOCOL FOR RESTLESS LEG SYNDROME IN PEDIATRIC PATIENTS

Taravath, S. Peedin, M.

Nunnelee Pediatric Speciality, Wilmington, NC.

Introduction: Iron dextran protocols have been studied for adults with restless leg syndrome; this study aims to determine the benefit of iron infusions (iron dextran or sucrose) in the improvement or resolution of restless sleep in pediatric patients.

Methods: IRB approval was granted by New Hanover Regional Medical Center. A retrospective chart review was performed using the hospital's electronic medical record (EMR). A data report was requested for all pediatric patients who received the iron dextran protocol. Patients who received at least one dose of iron dextran or iron sucrose per the protocol were included. Manual data collection included patient demographics, CBCs, iron panels, the dose of iron they received, polysomnogram results, symptom resolution (as described by the physician), and adverse events.

Results: Between January 1, 2017 and January 3, 2018, 55 patients received iron dextran per the weight-based protocol. Patients' ages ranged from 23 months to 17 years and 5 months old. Of the 55 patients, 27 patients (49%) had documented restless leg syndrome and/or symptoms of leg pain or movement during sleep. All patients experienced restlessness or difficulty sleeping prior to the infusion. Of the 27 patients who experienced leg pain, 10 patients (37%) experienced an improvement in their leg pain or restlessness. Fifty-five percent (55%) of patients in this group reported an improvement in sleep symptoms.

Conclusion: Iron dextran protocols have been studied for adults with restless leg syndrome, but this is the first published data for a weight-based protocol in pediatric patients. While only 27 patients included in this study had a diagnosis of restless leg syndrome or leg pain symptoms, all patients experienced restlessness or other negative sleep symptoms. Overall, most patients with restless leg syndrome experienced an improvement in their leg and sleep symptoms. No patients reported worsening of symptoms. Intravenous iron dextran infusion may greatly improve the quality of life for

SLEEP, Volume 43, Abstract Supplement, 2020

children with restless leg syndrome or poor sleep quality. With the overall safety of the iron infusions, this protocol's benefits outweigh their risks and should be considered in this patient population. **Support:**

0976

TEMPORAL ASSOCIATIONS BETWEEN SLEEP AND SUICIDALITY IN ULTRA-HIGH RISK ADOLESCENTS AND COLLEGE STUDENTS DURING AN INTENSIVE LONGITUDINAL STUDY

*Franzen, P. L.*¹ *Merranko, J.*¹ *Zelazny, J. H.*² *Hamilton, J. L.*¹ *Sewell, C.*³ *Goldstein, T. R.*¹

¹University of Pittsburgh School of Medicine, Pittsburgh, PA, ²University of Pittsburgh School of Nursing, Pittsburgh, PA, ³University of Pittsburgh School of Social Work, Pittsburgh, PA.

Introduction: Studies consistently demonstrate a link between subjective sleep disturbances and the continuum of suicidality, although this evidence primarily comes from retrospective, cross-sectional studies using limited items to assess sleep. Longitudinal assessment of well-defined and measured sleep/wake behaviors with high-risk individuals are needed to enhance the specificity of nearterm suicide risk detection and render concrete targets for suicide prevention.

Methods: Participants (N=46) included ultra-high-risk adolescents (N=29 ages 12-18) and college students (N=17 ages 18-24). For up to 12 weeks, participants wore an actigraph to yield objective data on sleep/wake, and concurrently completed daily cellphone-based ratings of subjective sleep and suicidality. Generalized estimating equations were used to examine the association between sleep parameters (subjective and objective) and the odds of next-day suicidal outcomes (i.e., passive death wish [PDW], suicidal ideation, suicidal intent) controlling for age, gender, and depression severity. Results: Significant quadratic relationships were observed between actigraphy-derived total sleep time (TST) and probability of nextday PDW (Z=3.7, p=0.0002), suicidal ideation (Z=2.1, p=0.04), and suicidal intent (Z=2.78, p=0.006), with increasing suicidality at low and high values of TST. Low sleep efficiency (<75%) was associated with increased odds of next-day PDW (OR=1.24, Z=2.07, p=0.038). Subjectively (sleep diary measures), low sleep quality (<50 on 100-point scale) was associated with increased odds of next-day suicidal ideation (OR=1.57, Z=3.42, p<0.001), and longer sleep onset latency (>20 minutes) with next-day suicidal intent (OR=3.00, Z=2.37, p=0.018).

Conclusion: Poor sleep health may signal increasing suicide risk, and are modifiable risk factors. We document a significant temporal association whereby objectively-derived short and long TST and low sleep efficiency, as well as subjective sleep quality and sleep onset latency, predicts next day's suicidality. Further understanding of the temporal association between sleep and suicidality may hold promise to inform real-time monitoring and preventive strategies. Interventions targeting these factors may therefore help reduce suicidality in high-risk youth.

Support: American Foundation for Suicide Prevention; University of Pittsburgh Clinical and Translational Science Institute

0977

RISK FACTORS FOR DEVELOPING SLEEP DISORDERS IN CHILDREN

Johnson, A. K.^{1,2} Santos, A. A.² Araujo, L. G.² Gonsalves, V. S.² Walker, B. L.² Santos, A. B.² Ajayi, A. O.¹ ¹Children's Lung, Asthma and Sleep Specialists, Winter Park, FL, ²AdventHealth University, Orlando, FL.

Introduction: Unidentified sleep disorders can affect emotional, cognitive and social development in children. Screening for sleep disorders within the pediatric population is not common practice during medical visits. The objective of this study is to identify specific questions related to behavioral and physiological factors having potential to screen and detect those at risk for sleep disorders in a general pediatric clinic.

Methods: A retrospective archive from electronic medical records was analyzed from 1,361 children patients, 0-18 years old, that visited a pediatric clinic from March-November of 2019. Children or their parents reported on the presence of eight objective behavioral and physiological factors on the Kids Sleep Screener Questionnaire (KSSQ), which were used as potential risk factors for sleep disorders. Propensity of daytime sleepiness was measured through the Epworth Sleepiness Scale for Children and Adolescents (ESS-CHAD). Scores higher than 11 were considered a positive indicator of potential sleep disorders because of excessive daytime sleepiness. Positive scores from the ESS-CHAD were used for comparison with the KSSQ factors using chi-square test of SAS software.

Results: Among the eight factors, snoring was the strongest risk factor and increased sleep duration was the weakest risk factor associated with a positive ESS-CHAD. Relationships among risk factors and the increased likelihood for developing sleep disorders were statistically significant (p<0.05-p<0.0001) and identified as following: snoring by 2.46 times, restless sleeper by 2.03 times, behavioral or learning difficulties by 1.43 times, nocturnal awakenings by 1.16 times, excessive sleepiness during the day by 1.10 times. Sleep onset latency and increased sleep duration were weak indicators due to a likeliness of less than one time (p<0.05) to be associated with a positive ESS-CHAD. Abnormal sleep behavior was not a statistically significant risk factor ($p\ge0.05$) for potential sleep disorders in children.

Conclusion: There were associations between seven behavioral and physiological risk factors with overall sleep propensity in children. These results exhibit that the KSSQ is an important tool to identify potential sleep disorders in children and the need for follow up with a sleep specialist. The KSSQ is under validation for becoming a standard sleep screener in pediatrics.

Support: N/A

0978

SNOO: A WELLNESS DEVICE TO IMPROVE INFANT SLEEP

Okun, M.¹ Karp, H.² Balasubramanian, S.²

¹University of Colorado Colorado Springs, Colorado Springs, CO, ²Happiest Baby, Los Angeles, CA.

Introduction: One of the primary contributors to new parent sleep deprivation is infant sleep disturbances. Evidence shows that increasing sleep in infants has a positive effect on parental sleep. SNOO, a wellness device, was developed to provide sleeping babies 3 of the 5 S's (safe swaddling, sound and swinging) in a response fashion (increasing levels of motion and white noise if babies cry) to soothe crying and improve infant sleep.

Methods: The current study compares infant sleep derived from SNOO to a reference group of a compilation of 13 peer reviewed studies of normative sleep data on sleep in traditional cribs/bassinets. Participants were 7157 babies using SNOO beginning within

one week of birth and for at least 6 hours per night for 6 months. Sleep metrics calculated on a day to day basis include *Longest Sleep Period*: Maximum uninterrupted sleep at night (7 PM - 7 AM); *Total Sleep Duration*: Total time spent sleeping at night (7 PM - 7 AM); and *Night Awakenings*: Number of times parents attended to the baby (10 PM - 6 AM).

Results: Improvement in the longest sleep duration varied with age. Across the 6 month collection period, longest sleep period increased between 42 minutes - 2 hours 0 minutes and total sleep duration increased between 33 minutes - 1 hour 24 minutes. Babies in SNOO averaged one less waking per night compared to the reference population $(1.09\pm.89 \text{ vs} 1.89\pm1.1)$. The improvements in all three sleep metrics were statistically significant with p's < 0.0005 across all ages from birth to 6 months.

Conclusion: This large-scale study suggests that infant sleep can be significantly improved by using SNOO compared to babies who slept in normal cribs or bassinets. We believe there are myriad areas of public health that may be positively impacted as a result of this significant level of improvement of infant sleep.

Support: Happiest Baby, Inc.

0979

OBSERVATIONAL ANALYSIS OF JUVENILE JUSTICE SLEEP-WAKE ENVIRONMENT

Adornetti, J. P.¹ Carlucci, M.¹ Crowley, S. J.² Fleshman, C. M.³ Jobe, S. L.⁴ Wolfson, A. R.¹

¹Loyola University Maryland, Baltimore, MD, ²Rush Medical University Medical Center, Chicago, IL, ³Warren Alpert Medical School of Brown, Providence, RI, ⁴University of Maryland School of Medicine, Baltimore, MD.

Introduction: Adolescence is associated with sleep regulatory changes that prompt sleep and circadian timing to shift later (delay). Poor quality, insufficient sleep, and misaligned sleep-wake schedules increase adolescents' risk for physical and mental health consequences. Little data exists on potential sleep health risks and sleep-wake environments of juvenile justice facilities. This descriptive study examined the sleep-wake environment and daily schedules at juvenile detention and treatment centers in a Mid-Atlantic state.

Methods: Using our Sleep Justice Observational Checklist, researchers recorded number of windows in sleep and non-sleep areas, and number of beds in sleeping quarters. Illuminance was measured with a light meter during the daytime (standing, sitting, etc.) and averaged. Facility-level 24-hour schedules were obtained to determine youth's daily routines during the observation period. **Results:** In comparison to treatment centers, detention centers have earlier lights-on ($M_{Det} = 6:07 \text{ am}$, $SD_{Det} =:40 \text{ vs}$. $M_{Treat} = 6:54 \text{ am}$, $SD_{Treat} =:07$, p = .04) and lights-off ($M_{Det} = 8:42 \text{ pm}$, $SD_{Det} =:36 \text{ vs}$. $M_{Treat} = 9:06 \text{ pm}$, $SD_{Treat} =:19$, N.S.) times. Treatment center illuminance levels (M = 296.60 lux, SD = 150.30) were greater (brighter) compared to detention centers (M = 124.00 lux, SD = 60.40, p = .01). Per sleep area, treatment centers had more windows ($M_{Treat} = 7.84$, $SD_{Treat} = 6.70 \text{ vs}$. $M_{Det} = 1.73$, $SD_{Det} = .77$, p = .02) and more beds ($M_{Treat} = 13.30$, $SD_{Treat} = 14.00 \text{ vs}$. $M_{Det} = 1.46$, $SD_{Det} = .96$, p = .03) than detention centers.

Conclusion: Preliminary results indicate a variation in the sleepwake environments and daily schedules in this sample of juvenile justice centers. Early lights-on and lights-off times can impose a higher risk for circadian misalignment in adolescents, though schedule consistency may reduce this risk. Ongoing data collection will help to further understand the sleep environment of adolescents in the juvenile justice system.

Support: Kolvenbach Research Grant, Loyola University Maryland

0980

SLEEP AND ACTIVITY RHYTHMS IN CHILDREN WITH DUCHENNE MUSCULAR DYSTROPHY

Hartman, A. G.¹ Smagula, S. F.¹ Bendixen, R.¹ ¹University of Pittsburgh, Pittsburgh, PA, ²University of Pittsburgh, Pittsburgh, PA.

Introduction: Duchenne muscular dystrophy (DMD) is a rare, progressive, neurological disorder with high rates of depression, anxiety, and attention issues. Rest-activity rhythm (RAR) disturbances are also associated with these co-morbidities in other populations. However, RAR patterns and timing have not been explored in DMD and could provide important information regarding mechanisms of sleep in which to intervene. To address this knowledge gap, we used wrist actigraphy to explore the timing of rest-activity rhythms in boys with and without DMD.

Methods: Boys aged 4-15 with DMD (n=17) and age matched controls (n=17) wore an ActiGraph GT9X for ten consecutive days. We averaged activity counts each hour and computed Cohen's *d* effect sizes and t-tests with Benjamini-Hochberg corrections. Extended cosine models were fit to derive RAR measures using custom R code.

Results: An overall delay in RAR timing was found in the DMD group due to three specific, large effect-size differences in RAR patterns. Boys with DMD were: (1) less active at 8AM and 9AM (e.g., at 9AM: d=-0.82, p=0.02, FDR=0.14); (2) more active from 10PM-midnight (e.g., at 11PM: d=1.1, p=0.003, FDR=0.07) and (3) more active at 4AM (d=0.74, p=0.04, FDR=0.14). Extended-cosine modeling showed that, while amplitude of activity was similar across groups (d=-0.29, p= 0.41), boys with DMD had later morning activity onset (d= 0.70, p= 0.05), later peak activity timing (d= 0.86, p= 0.02), and later evening activity offset (d= 0.96, p= 0.01).

Conclusion: A significant delay in activity was evident in the DMD group compared with controls. Although the mechanism causing this shift is currently unknown, delayed chronotypes have been linked to poor health outcomes like depression and anxiety in other populations. Further research should explore the causes and consequences of the RAR timing delays we observed and inform future care.

Support: Foundation to Eradicate Duchenne, PI: Bendixen, 1/1/2017-12/31/2019.

0981

SLEEP PRACTICES IN PEDIATRIC CANCER—DOES SLEEP HYGIENE MATTER FOR REDUCING CANCER SYMPTOM BURDEN?

Daniel, L. C.¹ Gross, Y.² Meltzer, L.³ Forrest, C.⁴ Barakat, L.⁴ ¹Rutgers University Camden, Camden, NJ, ²Lehigh University, Bethlehem, PA, ³National Jewish Health, Denver, CO, ⁴The Children's Hospital of Philadelphia, Philadelphia, PA.

Introduction: Sleep disturbances are common during pediatric cancer treatment and recent evidence suggests a correlation between sleep and symptom burden. Improving nighttime sleep may impact patients' ability to cope with symptoms. The current study tests the interaction between sleep hygiene and sleep disturbances

in predicting cancer-related symptoms to determine if the relationship between sleep and symptoms is different for patients with better sleep hygiene.

Methods: 102 caregivers of children with cancer (ages 5-17, M=10.12, SD=4.02; 58% female) completed parent-proxy Pediatric Sleep Practices Questionnaire (yielding routine consistency and sleep opportunity scores) and measures of cancer-related symptoms (PROMIS Sleep Disturbance, Fatigue, and Pain Interference; PedsQL—Cancer Module-Nausea subscale). The interaction between sleep disturbances and sleep hygiene (consistency, opportunity) on each symptom (pain, nausea, fatigue) were tested using PROCESS moderation.

Results: 81% of caregivers report that their child receives sufficient sleep but only 12% reported regular consistent sleep patterns/routines. Sleep opportunity was not related to sleep disturbances or cancer-related symptoms, but more routine consistency was related to fewer sleep disturbances (r=.30, p=.003). The interaction between sleep disturbances and routine consistency significantly predicted pain interference [$R^2=.16$, F(3, 98)=6.37, p<.001; B_{int}=-0.17, p=.028] and nausea [$R^2=.16$, F(3, 98)=6.47, p<.001; B_{int}=0.46, p=.004]. The interaction between sleep disturbances and sleep opportunity significantly predicted nausea [$R^2=.15$, F(3, 98)=5.76, p=.001; B_{int}=0.68, p=.016] but not pain interference. Both interaction models predicting fatigue were not significant.

Conclusion: The sleep/pain and sleep/nausea relationships are stronger in patients with more consistent sleep routines and the sleep/nausea relationship is also stronger in patients with sufficient/ well-timed sleep opportunities. Sleep and fatigue were moderately related across all levels of both sleep hygiene components. Clinical interventions that target sleep hygiene together with sleep disturbances such as nighttime awakenings and poor sleep quality may be more effective in addressing cancer-related symptoms such as pain and nausea.

Support: This work was supported in part by funding from the Patient-Centered Outcomes Research Institute (PCORI-D-17-00187; PI Christopher Forrest).

0982

CONTENT ANALYSIS OF THE FEATURES AND CLAIMS OF SMARTPHONE APPLICATIONS FOR CHILDREN'S SLEEP

Talker, I. Kaar, J. L. Simon, S. L.

University of Colorado Anschutz Medical Campus, Aurora, CO.

Introduction: Empirically supported treatments for pediatric sleep problems exist but many families turn to other sources for help with their child's sleep, such as smartphone applications (apps). Sleep apps are easy for families to access but little evidence exists regarding the validity of the services and information provided. The goal of this study was to examine the features and claims of sleep apps for children.

Methods: A search of the Apple iTunes store and Google Play Store was conducted using the terms "kids sleep" and "baby sleep". 635 apps were initially identified. Apps were excluded if they were not specifically for children (n=163), not for sleep (n=152), or if they had <100,000 downloads (n=246). Content analyses were used to assess the apps functions, claims, and evidence base.

Results: A total of 74 apps were examined of which only 4% offered sleep improvement strategies. The majority were sound and light apps (77%) and 19% were bedtime games/stories. The apps were highly rated (average 4.4 out of 5) and most were free (54%); the price of paid apps ranged from \$0.99 to \$119.99 (annual

subscription). Only 2 apps were identified as containing empirical evidence, and all of the apps featuring games and stories to be used as part of the bedtime routine are in opposition to the recommendation to avoid the melatonin-suppressing effect of electronics/ bright light before bedtime. Despite this, many apps boasted claims that they will help children "fall asleep instantly," "cry less and sleep better," or improve child development.

Conclusion: A large variety of sleep applications exist aimed for use with children. Many boast claims that cannot be supported by empirical evidence, and indeed may be in opposition to research support. Collaboration between sleep researchers and technology developers may be beneficial for the creation of evidence-supported apps to help with children's sleep.

Support: N/A

0983

SLEEP ASSOCIATED WITH EXECUTIVE FUNCTIONING AMONG ADOLESCENTS ACROSS THE ADHD CONTINUUM

Lunsford-Avery, J. R.¹ Krystal, A. D.² Carskadon, M. A.³ Kollins, S. H.⁴

¹Duke University Medical Center, Durham, NC, ²University of California San Francisco, San Francisco, NC, ³Warren Alpert School of Medicine at Brown University, Providence, RI, ⁴Duke University School of Medicine, Durham, NC.

Introduction: Executive functioning (EF) deficits are a key feature of ADHD, and sleep disturbances may be an important contributor. Specifically, disturbed sleep is prevalent in ADHD and similar EF deficits are observed in ADHD and sleep disorders. Associations between disrupted sleep and EF in ADHD are poorly understood, particularly during adolescence. This study is among the first to examine relationships between sleep and EF using polysomnography (PSG) among adolescents across the ADHD symptom continuum.

Methods: In this ongoing study, 42 adolescents aged 13 to 17 (mean age = 14.86, 20 females) completed 3 nights of at-home PSG recording (total sleep time; TST) and self-reports of sleep quality, daytime sleepiness, and chronotype. Seventeen had ADHD and 25 were healthy controls (HC). Participants and parents also completed a measure of EF (BRIEF-2; global and behavioral, emotional, and cognitive subscales). Linear regressions controlling for age and sex evaluated associations between sleep and EF.

Results: Self-reported poorer sleep quality and greater daytime sleepiness and eveningness tendency (p's<.05), but not TST, were associated with poorer self-reported global EF among adolescents. Shorter TST and greater eveningness were correlated with poorer parent-reported global EF (p's<.05). Follow up analyses examine differential relationships between sleep and behavioral, emotional, and cognitive domains of EF and between ADHD and HC groups. Conclusion: This study is among the first to examine relationships between sleep and EF across the ADHD continuum in an adolescent-specific sample using PSG. Objectively-measured TST as well as subjective measures of sleep were associated with poorer EF in adolescents across the ADHD continuum. Prevention/ intervention strategies focused on sleep may support EF among adolescents, and future studies should examine this possibility. In addition, given variability in EF among individuals with ADHD, future studies should investigate whether sleep disturbances identify a phenotypic subgroup within ADHD at risk for EF deficits.

Support: This work was supported by NIMH K23 MH108704 (Dr. Lunsford-Avery)

0984

LESS CONSISTENT BEDTIMES EXPLAINS THE **RELATIONSHIP BETWEEN HOUSEHOLD POVERTY AND BMI IN TODDLERS**

Covington, L. B.¹ Armstrong, B.² Black, M. M.^{3,4} ¹University of Delaware School of Nursing, Newark, DE, ²University of South Carolina Arnold School of Public Health, Columbia, SC, ³University of Maryland School of Medicine, Baltimore, MD, ⁴RTI International, Raleigh, NC.

Introduction: Consistent bedtimes, in conjunction with physical activity and diet have been linked to healthy weight in childhood. Young children living in impoverished families are at risk for obesity, and the mechanisms of obesity etiology are not fully understood. This study compares the role of bedtime consistency, physical activity and diet quality as mediators between household poverty and toddler weight gain.

Methods: 207 toddlers participating in an obesity prevention trial wore Actical accelerometers for up to 7 consecutive days, at 3 time points over 12 months. At each assessment, gender-specific BMIfor-age z-scores (zBMI) were calculated from toddlers' weight/ length according to WHO standards. Household poverty ratio was calculated based on the number of household members and annual income. Diet quality was assessed using Healthy Eating Index (HEI-2015) from 24-hour dietary recall. Physical activity and sleep were measured using ankle accelerometry (Actical; Sadeh algorithm used for sleep). Bedtime consistency was defined as SD of sleep onset across 7 days. A multi-level mediation model was conducted in the SPSS macro MLmed examining toddler bedtime consistency, physical activity and diet quality as mediators between household poverty and toddler zBMI. The analysis adjusted for toddler age, gender, total sleep time and intervention group.

Results: Between-person effects revealed that less household poverty was associated with more consistent bedtimes. Children with less consistent bedtimes, but not poor diet quality or physical activity, had higher zBMI. Bedtime consistency indirectly explained the association between average household poverty and average toddler zBMI over 12 months.

Conclusion: Children who generally have less bedtime consistency, above and beyond physical activity and diet quality, had higher zBMI. This link uniquely indirectly explained the association between household poverty and zBMI. Inconsistent bedtimes may indicate lack of structure in other health behaviors, and therefore, continued longitudinal research examining family routines may inform obesity prevention strategies.

Support: Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), R01HD056099

0985

UTILITY OF A REST-ACTIVITY RATIO IN A PEDIATRIC **BRAIN INJURY REHABILITATION SAMPLE: A PILOT** STUDY

Lequerica, A.¹ Lengenfelder, J.¹ Genova, H.¹ Shoval, H. A.³ Marchetta, C.³ Yalamanchi, K.³ DeLuca, J.¹

¹Kessler Foundation, East Hanover, NJ, ²Kessler Foundation, East Hanover, NJ, 3Children's Specialized Hospital, New Brunswick, NJ.

Introduction: Sleep is an important element of health that can impact daytime performance in brain injury rehabilitation. Actigraphy in a pediatric inpatient setting can pose challenges when there are increased amounts of time spent in a hospital bed. A rest-activity

VII. Pediatrics

ratio (Duclos et al., 2013) has demonstrated utility in adult brain injury populations, showing sensitivity to improved regulation of the sleep-wake cycle as individuals recover during their hospitalization. The present study sought to examine the utility of this ratio in differentiating children with and without acquired brain injury (ABI).

Methods: Sixteen individuals, ages 8-16 (M=12.6, SD=2.4) admitted to an inpatient pediatric rehabilitation hospital wore an actigraph over a 7-day period. Eight inpatients were being treated for ABI whereas another 8 inpatients treated for other, nonneurological conditions served as a control. Raw activity counts across the 24-hour period were analyzed to derive a rest-activity ratio.

Results: Those with ABI had lower average ratios (0.73) compared with patients without ABI (0.84), F(1,14)=4.3, p=0.058. Individuals with ratios of 0.85 or higher were rated by their physical therapists as being more alert during therapy using a 5-point Likert scale, F(1,14)=4.1, p=0.061. While these results were marginally significant, this pilot sample was small, and the effect sizes were large (eta squared = 0.234 and 0.229 respectively).

Conclusion: The rest-activity ratio successfully distinguished those with ABI from a non-ABI sample. This preliminary evidence in a pediatric sample suggests that this ratio, shown to be sensitive to the effects of brain injury on sleep-wake regulation, may be a useful metric in the inpatient pediatric rehabilitation setting when sleep diaries may be difficult to obtain and patients may be spending more time in bed while awake. Future studies with larger sample sizes are needed to explore the correlates of this ratio with other aspects of rehabilitation after pediatric brain injury.

Support: This study was funded in part through a generous grant from the Church & Dwight Employee Giving Fund

0986

POSITIVE AIRWAY PRESSURE CARE AND CLEANING PRACTICES IN THE PEDIATRIC HOME

Pruss, K. K.¹ Willis, D.¹ Spray, B. J.¹ Jambhekar, S.² ¹Arkansas Children's, LITTLE ROCK, AR, ²University of Arkansas for Medical Sciences, LITTLE ROCK, AR.

Introduction: The Sleep Clinic at Arkansas Children's follows approximately 300 children who require positive airway pressure (PAP) at home. The clinic respiratory therapist provides oral and written cleaning instructions while some physicians choose to provide their own instructions. The home equipment company who supplies PAP in the home also gives cleaning instructions. The different routes of information given may result in inconsistent practices. It is extremely important to clean PAP equipment as directed as infection and illness may result from improper cleaning.

Methods: Caregivers of children who utilize home PAP devices were invited to complete an anonymous survey regarding cleaning practices during a Sleep clinic appointment. Data were collected electronically. Descriptive statistics were utilized to summarize results.

Results: There were 96 participants of whom 90% (87/96) were parents/caregivers. The mean age of the equipment user was 12 years and most were male (69%, 66/96). The mean length of time the equipment had been used was 2.6 years (SD 3.2). The majority of respondents, 67% (64/95), identified the parent/caregiver as responsible for cleaning. Only 25% (24/96) reported cleaning the mask daily as recommended; 43% (41/96) of participants reported cleaning tubing weekly; 27% (26/96) reported cleaning the water chamber daily by while most reported at least weekly (47%, 45/96).

The majority, 58% (56/96) reported emptying the water chamber daily and using distilled water (81%, 74/96). Most respondents did not note respiratory symptoms starting/increasing with PAP (67%, 64/96). Of those with respiratory symptoms attributed to PAP, congestion was the most common (79%, 11/14).

Conclusion: There is a discrepancy between recommended and actual practices for cleaning PAP equipment. No significant association was found between the duration of PAP use and cleaning practices. However, a moderately low correlation between age and cleaning was identified. Increased age was associated with decreased cleaning practices.

Support:

0987

CHARACTERIZING SCRATCH AND LIMB MOVEMENTS IN ATOPIC DERMATITIS DURING SLEEP

Gillow, G. M.¹ Robins, C.² Palomo, R.² Sheldon, S. S.³ Fishbein, A. B.⁴

¹Northwestern University Feinberg School of Medicine, Chicago, IL, ²Ann & Robert H. Lurie Children's Hospital, Sleep Medicine Center, Chicago, IL, ³Ann & Robert H. Lurie Children's Hospital, Division of Pediatric Pulmonary and Sleep Medicine, Chicago, IL, ⁴Ann & Robert H. Lurie Children's Hospital, Division of Pediatric Allergy & Immunology, Chicago, IL.

Introduction: Children with atopic dermatitis (AD) experience significant sleep disruption due to nocturnal scratching. Our group has found distinct patterns of limb movements in contrast to control and PLMD patients. To expand on previous findings, our objective was to characterize timing and duration of scratch v. non-scratch movement in children with AD coincidentally undergoing polysomnography (PSG).

Methods: Retrospective chart review of PSG, video footage was synchronized with the EEG and limb electrode readings using a time/date generator and was then operationally classified as either scratching or non-scratching movement. Analysis of data was done using SPSS and groups were compared using an ANOVA.

Results: We analyzed four previously completed sleep studies in children with atopic dermatitis (1 female and 3 males), mean age years±SD 11.3±1.0, mean BMI±SD 21.9±7.1, mean AHI±SD 2.3±0.8. Average scratch duration was not significantly different by sleep stage, N1v.N2v.N3v.REM (mean scratch duration in seconds \pm SD= 9.0 \pm 1.5 v 6.3 \pm 3.2 v. 11.9±11.8 v. 6.3±7.3, respectively p=0.65). However, frequency of scratching events were more common during N2v. N1v.N3v.REM (mean scratching events \pm SD= 9.3 \pm 3.9 v 3.8 \pm 1.7 v. 4.3 ± 4.3 v. 1.3 ± 1.9 , respectively p=0.02). Yet, given the duration of total time spent in sleep stages, minutes of scratching events occupied the largest percentage of N1v.N2v.N3v.REM $(\text{mean}\% \pm \text{SD}= 3.9\pm 0.9 \text{ v}. 0.6\pm 0.4 \text{ v}. 0.4\pm 0.2 \text{ v}. 0.3\pm 0.5, \text{ respect-}$ ively, p<0.01). Interestingly, non-scratch related movements were not significantly different between sleep stages (p=0.2). However, non-scratch related movements trended to occupy the largest percentage of N1v.N2v.N3v.REM (mean% ±SD= 9.3 ± 7.7 v. 2.1 ± 1.6 v. 1.5 ± 0.8 v. 1.9 ± 1.4 , respectively, p=0.05).

Conclusion: Our results suggest that scratching episodes in children with AD occur most commonly during N2 sleep, but occupy the largest % of N1 sleep. Future work will include comparing these limb movements to age and gender-matched allergic rhinitis patients.

Support: This study was unfunded.

0988

INTERACTION OF RACE/ETHNICITY AND ADVERSE CHILDHOOD EXPERIENCES: LINKS TO SUBSEQUENT CHILDHOOD SLEEP DURATION

Rojo-Wissar, D. M.¹ Sosnowski, D. W.¹ Jackson, C. L.² Maher, B. S.¹ Spira, A. P.¹

¹Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ²Epidemiology Branch, National Institute of Environmental Health Sciences, National Institutes of Health, Department of Health and Human Services, Research Triangle Park, NC.

Introduction: Adverse childhood experiences (ACEs) and sleep disturbances independently affect health and development across the lifespan. While burgeoning research suggests a link between ACEs and sleep health among adults, few studies have examined the association between ACEs and sleep in childhood or whether these associations vary by sex or race/ethnicity.

Methods: Using prospective data from 2,063 children (49% female; 20% Non-Hispanic [NH] White, 55% NH Black, 25% Hispanic/ Latino) from the Fragile Families & Child Wellbeing Study, we used multiple linear regression analyses to examine associations between primary caregiver reports of child cumulative ACEs (i.e., physical abuse; emotional abuse; neglect; household dysfunction; possible range=0 to 10) at age 5 and primary caregiver reports of average sleep duration (minutes) at age 9. We used interaction terms to examine whether these associations varied by sex (reference group: males) or race/ethnicity (reference group: NH Whites). If significant, we used plots to visually investigate other potential between-groups differences (i.e. non-overlapping 95% CIs) and tested these statistically using linear combinations of estimator tests. If there were significant group differences, analyses were stratified by sex or race/ethnicity.

Results: Associations between ACEs and sleep duration significantly varied by race/ethnicity but not sex, such that the magnitude of the association was stronger in NH Whites compared to NH Blacks (p<.0001) and in Hispanics/Latinos compared to NH Blacks (p<.0001). In analyses stratified by race/ethnicity and adjusted for both sex and age 5 sleep duration, each unit increase in ACE score was associated with a 6.66 minute shorter sleep duration in NH Whites (B=-6.66, SD=2.10, p=0.002), a non-significant 2.20 minute shorter sleep duration in NH Blacks (B=-2.20, SD=1.52, p=0.148), and a 4.36 minute shorter sleep duration in Hispanics/Latinos (B=-4.36, SD=2.03, p=0.032).

Conclusion: We found that cumulative ACEs were associated with shorter sleep duration across race/ethnicity and more strongly related in NH White and Hispanic/Latino children. Prospective studies with subjective and objective sleep measures investigating multiple sleep parameters are needed that identify factors (e.g., cumulative disadvantage) that differentially affect associations across race/ethnicity and characterize health outcomes of ACEs and sleep duration.

Support: 5T32MH014592-39, 5T32DA007292-27, Z1A ES103325-01

0989

EXAMINING THE ROLE OF TODDLER SLEEP QUALITY ON WAKE EEG AND LANGUAGE ABILITY

Page, J.¹ Walters, R.² Gould, R.³ Wakschlag, L.¹ Norton, E.¹ ¹Northwestern University, Chicago, IL, ²PhenomHealth, Philadelphia, PA, ³Johnson & Johnson Consumer Inc, New Brunswick, NJ, ⁴Northwestern University, Chicago, IL. **Introduction:** Sleep and the development of language are prominent concerns of many parents and until recently, many have examined these concerns tangentially. Children with developmental delays/disabilities have shown to have impaired sleep and poor sleep quality, and impairments or changes in sleep quality may play a prominent role in the acquisition of language and neuronal oscillatory patterns. This study examines the role of child sleep quality paired with a normed measure of language and wake electroencephalography (EEG). Examining the role of child sleep quality with language ability and wake EEG may provide nascent incremental utility to understanding the influences of sleep on healthy development.

Methods: Data from 109 toddlers (age range 24 to 30.5 m, $M = 26.83 \pm 1.58$ m, 52% male) from the Brief Infant Sleep Questionnaire (BISQ), Mullen Scales of Early Learning (MSEL), and continuous EEG were collected and analyzed. EEG was recorded (32 electrode cap BioSemi) while toddlers sat in a booster seat and watched a silent video. Data were analyzed in RStudio and Matlab to examine toddler's sleep quality (infant sleep and parent behaviors) and relations with the MSEL and EEG (controlling for child age and gender).

Results: Means and standard deviations appeared within expected limits based on the range of each variable. Toddlers with slow-developing language were associated with relatively poor sleep quality, explaining 9.75% of the variance. We find preliminary evidence to suggest a potential sleep disruption around the time when a child is undergoing a rapid expansion in their vocabulary (expressive language). Toddler's sleep quality and language acquisition were also correlated with wake EEG (alpha and beta).

Conclusion: Sleep is regarded as an essential component supporting the myriad changes observed in early development. Sleep quality fundamentally influences healthy development across domains. Here, we showed child sleep quality is highly associated with toddler's language ability, and wake EEG, providing new insights into the developing brain.

Support: National Institutes of Health R01DC016273, R01MH107652-03S1, and Johnson & Johnson Consumer Inc., Skillman, NJ, USA.

0990

EVALUATING COMPONENTS OF SLEEP QUALITY AND THE SLEEP-QUALITY OF LIFE RELATIONSHIP FOR CHILDREN WITH ACUTE LYMPHOBLASTIC LEUKEMIA

Gross, Y.¹ Barakat, L.² Daniel, L. C.¹

¹Rutgers University Camden, Camden, NJ, ²The Children's Hospital of Philadelphia, Philadelphia, PA, ³Rutgers University Camden, Camden, NJ.

Introduction: Poor sleep quality is associated with reduced health-related quality of life (HRQL) for children with Acute Lymphoblastic Leukemia (ALL). Research has yet to evaluate how components of child sleep quality uniquely contribute to HRQL beyond demographic characteristics. This study evaluates features of sleep and the relationship between sleep and HRQL for children in the maintenance phase of ALL treatment.

Methods: 89 caregivers (ages 20-52, M=35.95, SD=7.10) of children with ALL (ages 3-12, M=5.73, SD=2.21; 13.76 months from diagnosis) completed demographic questionnaires and reports of child sleep quality (Child Sleep Habits Questionnaire; CSHQ), and 64 caregivers reported on child HRQL (Pediatric Quality of Life Inventory). Descriptive statistics were calculated. T-tests compared CSHQ subscales to ratings of healthy normative values. Pearson

correlations evaluated associations between sleep and overall HRQL. Hierarchical regression assessed whether CSHQ subscales uniquely predicted HRQL beyond demographic characteristics.

Results: This sample reported greater bedtime resistance [t(88)=6.413, p<.001], sleep onset delay [t(88)=3.180, p=.002], sleep anxiety [t(88)=4.271, p<.001], night awakenings [t(88)=6.031, p<.001], parasomnias [t(88)=3.900, p<.001], and daytime sleepiness [t(87)=1.781, p=.078] than normative values, although sleep duration [t(88)=1.781, p=.078] and sleep disordered breathing (SDB) [t(88)=-.061, p=.951] did not differ. HRQL was related to SDB (r=-.289, p=.021), bedtime resistance (r=-.263, p=.036), and total sleep score (r=-.34, p=.006). The regression model with SDB and bedtime resistance explained 24.2% of variance but was not significant [F(6,31)=1.651, p=.167].

Conclusion: Caregiver ratings showed greater sleep impairments for children in this sample than of norms. Sleep subscales were associated with HRQL, but did not predict HRQL beyond demographic factors. Caregiver reports of child sleep and HRQL may vary depending on when during the monthly chemotherapy cycle questionnaires were completed. SDB did not differ from normative values but was related to HRQL, suggesting the need to screen for SDB symptoms to potentially improve child outcomes.

Support: This study was supported by funding from the American Cancer Society PF-13-238-01-PCSM (PI: Daniel).

0991

THE EFFECT OF EXTENDING TOTAL SLEEP TIME AND WEIGHTED BLANKETS ON TEENAGE SWIMMERS PERFORMANCE

Zarrouf, L. R.

TL Hanna High School, Anderson, SC.

Introduction: Effectiveness of sleep extension on performance and cognition in adult athletes has been studied extensively. Effectiveness of weighted blankets on sleep extension in children has been studied with mixed results. The effect of sleep quantity on teen competitive swimmers has not been evaluated extensively. This study investigated the effects of sleep extension and weighted blankets on performance, as well as daytime sleepiness in competitive teen swimmers. The principal investigator is a high school student and a teen-swimmer herself.

Methods: Study Design: Using an open label prospective approach, the pilot study will investigate swimmer's event time changes, actigraphy findings and daytime sleepiness with sleep extension and weighted blankets. Setting/ Participants: 12 healthy swimmers on the MAKOS swim team will maintain their habitual sleep-wake schedule for a one-week; baseline period followed by a one-week sleep extension period, combined with weighted blankets use. Procedure/Protocol: The head-coach will assign event type to each participant of the study, 2 participants of the same type of event, will do the baseline timed race and initial evaluation, followed by one week of regular sleep (control). Another timed event will be done at the end of the 1st week and followed by a 2nd week of extended sleep (one hour/day) and weighted blanket. Final timed event race will be at the end of the 2nd week. Participants will fill initial, weekly questionnaires and wear a sleep tracker during the two weeks of the study. Detailed sleep and activities analysis will be obtained.

Results: 12 swimmers were recruited, 8 females and 4 males. All participants have been consistently with the team for more than 2 years. Age range of participants is 11-17 years. Members of the team practiced 5 days every week with each practice lasting for 2

hours. The first timed race is scheduled to be done the first week in January.

Conclusion: This is the first research study to evaluate the effect of two important variables on sleep and performance in teenage swimmers.

Support: The authors report no financial support related to this study.

0992

PREVALENCE OF INSUFFICIENT SLEEP DURATION AND CONSEQUENCES ON DAYTIME SLEEPINESS, MOOD AND ACADEMIC PERFORMANCE IN THAI ADOLESCENTS

Rojrattanadumrong, R. Sudnawa, K. K. Areekul, W. Jaroenying, R. Department of pediatrics, Phramongkutklao Hospital, Bangkok, THAILAND.

Introduction: Insufficient sleep duration in adolescents is key public health concerns in many societies. This study aims to assess the prevalence of insufficient sleep duration and the association between short sleep with daytime sleepiness, depressive symptoms, and anxiety among Thai adolescents.

Methods: Thai adolescents aged 11 -18 years completed a selfadministered questionnaire including Phramongkutklao Hospital Sleep disorders center Questionnaire, Pittsburg Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), The Children depress Inventory (CDI) scale and Child Anxiety Related Emotional Disorder (SCARED).

Results: 232 adolescents with the mean age of 14.58 (range 11-18) years participated in this survey. The average total sleep time was 7.44 hours. The overall prevalence of insufficient sleep duration (sleep duration <8 hours) was 37.5% and the small difference was found between males (42.0%) and females (32.7%). Compared with those who had a sleep length of 8 hours and more, insufficient sleep duration group exhibited higher in BMI and less in Grade Point Average (GPA). Subjective sleep insufficiency among Thai adolescent also had higher percentage of subjects with ESS \geq 10 (43.2% vs 21.3%, P <0.001), depressive symptoms (56.3% vs 39.3%, P 0.012) and anxiety (54.0% vs 38.6%, P 0.022) compare to those with adequate sleep duration.

Conclusion: High amount of Thai adolescents with poor sleep duration were observed in the study, consistency with prior surveys. Insufficiency sleep duration group had significantly increased daytime sleepiness, mood and anxiety problems among Thai adolescents. These findings emphasized the development of interventions to improve sleep duration in Thai adolescents and more sample size should be achieved in the future.

Support: None

0993

IMPACT OF CHANGING SCHOOL START TIMES ON PARENT SLEEP

Meltzer, L. J.¹ Plog, A. E.² McNally, J.² Wahlstrom, K. L.³ ¹National Jewish Health, Denver, CO, ²Cherry Creek School District, Greenwood Village, CO, ³University of Minnesota, Minneapolis, MN.

Introduction: No studies have previously considered how healthy school start times impact parents. Since parent and child sleep schedules, in particular wake time, are associated, we examined whether changing school start times would impact parent sleep

(elementary school [ES] 9:00am to 8:00am, middle school [MS] 8:00am to 8:50am, high school [HS] 7:10am to 8:20am).

Methods: Parents of students (grades K-12) completed online surveys pre-change, one-year, and two-years post-change (2017 n=11,579; 2018 n=8,884; 2019 n=8,796), reporting weekday bedtime and wake time (sleep duration calculated bedtime to wake time). Four PROMIS sleep questions asked about sleep "quality", feeling "alert" upon waking, feeling "tired" during the day, and "daytime problems" because of sleep. Finally, parents were asked one-year post-change how happy they were about the new school start times.

Results: Parents with only ES students reported earlier bedtimes and wake times (10 and 13 minutes). Parents with \geq 1 HS student woke later (HS only: 22 minutes; MS and HS: 25 minutes; ES and HS: 10 minutes; all 3 levels: 14 minutes) and slept longer (HS only: 19 minutes; MS and HS: 21 minutes; ES and HS: 12 minutes; all 3 levels: 18 minutes), with changes maintained two years. Similarly, parents of only ES students reported no change in sleep quality/ impairment. Post-change, more parents with \geq 1 MS or HS student reported good sleep quality (average increase=6.9%), feeling alert (average increase=5.8%), not feeling tired (average increase=5.0%), and having few daytime problems (average increase=4.2%), with benefits maintained two-years post-change. Across levels, parents who were "very happy" about start time changes slept longer than "very unhappy" parents (12-30 minutes); however, across levels, child sleep did not differ between happy and unhappy parents.

Conclusion: This is the first study to examine the impact of changing start times on parents of students in grades K-12. Study results show no significant impact on parents of elementary students. However, similar to students, parents of middle/high school students reported later wake times and increased sleep duration. Notably, parents' happiness with start time changes was related to parent, but not student, sleep duration.

Support: Robert Wood Johnson Foundation's Evidence for Action Grant

0994

ASSESSMENT OF SLEEP-WAKE AND CIRCADIAN RHYTHM DISRUPTION IN CHILDREN AND ADOLESCENTS DIAGNOSED WITH CRANIOPHARYNGIOMA

LaRosa, K. N.¹ Crowley, S. J.² Hancock, D.¹ Caples, M.¹ Merchant, T. E.¹ Crabtree, V. M.¹ Mandrell, B.¹ ¹St. Jude Children's Research Hospital, Memphis, TN, ²Rush Medical College, Chicago, IL, ³St. Jude Children's Research Hospital, Memphis, TN.

Introduction: Patients with craniopharyngioma are at increased risk for hypersomnia/narcolepsy and circadian rhythm disruption, secondary to hypothalamic-pituitary involvement of the tumor. We assessed youth with craniopharyngioma to determine presence of the dim light melatonin onset (DLMO) and concurrent sleep disturbance.

Methods: Fifty-two patients (7-21 years; 51% female) enrolled on our institutional protocol for craniopharyngioma that included surgery, proton therapy, or both. In-home salivary melatonin was collected after surgery and hourly beginning 3 h before to 1 h after habitual bedtime to determine the DLMO, which was defined as the time that melatonin exceeded a 4 pg/mL threshold. Polysomnography and a next day multiple sleep latency test (MSLT) were also conducted.

Results: Hypersomnia/narcolepsy was indicated in 86% of patients. DLMO was detected in 29 (56%) patients and averaged 21:04 (\pm 1:14). All but 2 patients with a DLMO had a concurrent sleep diagnosis (18 hypersomnia, 8 narcolepsy, 1 insomnia). In those we could not compute a DLMO, melatonin was above the 4 pg/mL threshold in 19 (37%), suggesting that the DLMO was likely earlier than the sampling window. Two (4%) did not reach threshold, suggesting that the DLMO was later than the window. For patients in which DLMO was not computed, all but 4 had a concurrent sleep diagnosis (7 hypersomnia, 9 narcolepsy, 1 MSLT not completed). Three (6%) participants showed a pattern of melatonin decreasing before bedtime (2 hypersomnia, 1 narcolepsy). Sleep disorder diagnosis was not associated with whether a DLMO was detected or not.

Conclusion: DLMO did not occur within the sampling window in 44% of patients with the majority due to the DLMO likely occurring before sampling started. Simultaneous assessment of both sleep-wake disturbance and circadian phase could provide more informed sleep interventions for excessive sleepiness and circadian misalignment in this patient population.

Support: This study was supported by cancer center grant (CA21765) from the National Cancer Institute, and ALSAC.

0995

NIGHTCAP FOR SCHOOL-NIGHTS: ASSOCIATION BETWEEN MILK INTAKE AND SLEEP DURATION IN FIRST-GRADERS

*Chung, A.*¹ *Martinez, S.*² *Ursache, A.*³ *Chang, S.*³ *Huang, Y.*³ *Jean-Louis, G.*³ *Brotman, L.*³

¹New York University School of Medicine, New York, NY, ²University of California, San Francisco, San Francisco, CA, ³NYU Grossman School of Medicine, New York, NY.

Introduction: Insufficient sleep has been identified as an obesity risk factor due to mechanistic pathways contributing to higher carbohydrate intake, including in children. Dietary intake of macronutrients, such as fats and protein found in milk, may serve as a modifiable risk factor for adequate sleep. We hypothesize that milk intake among a sample of urban first-graders may be associated with sleep duration.

Methods: Cross-sectional analysis of parent reports of an adapted version of the Child Sleep Health Questionnaire (CSHQ) and Block Dietary Data Systems Food Frequency Questionnaire (FFQ) were analyzed among a sample of 837 Black children in Brooklyn, New York. Summary scores were created for milk type. Milk intake was classified by fat content: whole milk and 2% categorized as high-fat, and 1% and skim as low-fat. Independent t-test, correlations and regression analysis to identify associations between parent reports of child's sleep duration and milk intake were conducted.

Results: On average, children were 7.3 ± 0.6 years old and 52% female. Nearly 57% of parents were immigrants. Children's mean BMI was 17.27, approximately at the 85th BMI percentile according to CDC index-for-age percentiles. On average, FFQ data reported children consumed high-fat milk 6 days a week. Linear variable regression analysis between high-fat milk intake and sufficient sleep were significant ($\beta =$, 0.090, p < 0.05). BMI was significantly associated with high-fat milk intake ($\beta = 0.17$, p<0.05). However, high-fat milk intake was not significantly associated with (in)sufficient sleep, after controlling for BMI, sex and age. No difference was reported between immigrant parents and U.S. born parents.

Conclusion: Plausibly, high-fat milk is contributing to satiety and longer sleep duration. Future studies should include more

comprehensive measurement of milk consumption (i.e. time of day and volume) to consider possible effects on children's sleep. Actigraphy measures and sleep diaries should also be considered. **Support:** Bezos Grant and Community Service Plan grant.

0996

SCHOOL START TIMES ARE ASSOCIATED WITH YOUTH AND PARENT SLEEP DURATION.

Gunn, H. E. Eberhardt, K. R.

University of Alabama, Tuscaloosa, AL.

Introduction: Early school start times contribute to insufficient sleep in adolescents; however, we know little about the impact of school start times at a family level. Moreover, even among similar school start times, sleep opportunity varies depending on mode of transportation and travel time. Thus, the purpose of this study was to determine whether AM school departure time is associated with sleep duration in parents and young adolescents.

Methods: Parent-adolescent dyads (n=31) completed 10 days of actigraphy and sleep diaries. Adolescents were 10-14 year olds (58% male). Parents were predominately mothers (87%) and their mean age was 44 yrs (SD = 5.9). Dyads were 77% White, 11% Black, and 12% Biracial or Other. Youth leave for school time was assessed with the School Sleep Habits Survey. Actigraphy- and diary-assessed total sleep time (TST) was averaged across 10 days and on weekdays. Separate regressions models for parents and adolescents determined associations between school leave time (predictor) and two outcomes: 10-day TST and weekday TST.

Results: For adolescents, later leave for school time was associated with longer 10-day actigraphy-assessed TST ($\beta = .504$, p = .012) and diary-assessed TST ($\beta = .683$, p <.001). Later leave for school time was also associated with more weekday actigraphy and diary-assessed TST ($\beta = .661$ and .426, respectively, p's < .05). For parents, later leave for school time predicted more diary-assessed sleep across 10 days ($\beta = .481$, p = .013) and on weekdays, but this finding did not reach significance ($\beta = .373$, p = .061). Leave for school time was not associated with parents' actigraphy-assessed TST across the 10-day period or on weekdays (p's > .10).

Conclusion: The time that youth need to leave for school may more closely approximate sleep opportunity regardless of actual school start time. This is particularly relevant for urban and rural youth with long commutes. Findings add to the strong support that delayed school start times or flexible scheduling will benefit adolescent sleep and also suggest positive impacts at the family level.

Support: This material is based upon work supported by the Sleep Research Society Foundation.

0997

THE SLEEP DISTURBANCE SCALE FOR CHILDREN IN YOUTH WITH TOURETTE'S DISORDER

Montalbano, G. E.¹ Rozenman, M.² Peris, T.¹ Tan, P.⁴ Piacentini, J.¹ Ricketts, E.¹

¹University of California, Los Angeles, Los Angeles, CA, ²University of Denver, Denver, CO, ³University of California, Los Angeles, Los Angeles, CA, ⁴University of Southern California, Los Angeles, CA.

Introduction: Sleep disturbance is common in youth with Persistent Tic Disorders (PTDs), including Tourette's Disorder. However, studies elucidating the nature of sleep problems in PTDs are limited. The present study examines the types of sleep disturbance present in youth with PTDs relative to healthy controls, and

investigates the relationship between sleep disturbance and tic severity.

Methods: Participants were 56 youth ages 8 to 17 (M=11.9, SD=2.86), including individuals with PTDs (n=27), and healthy controls (n=29). An interviewer evaluated psychiatric diagnosis using the Anxiety Disorders Interview Schedule, and tic severity using the Yale Global Tic Severity Scale (YGTSS). Parents rated sleep using the Sleep Disturbance Scale for Children (SDSC), and tic severity using the Parent Tic Questionnaire (PTQ). Independent-samples t-tests and bivariate correlations were performed.

Results: Higher SDSC Total scores, t(30)=-3.74, p=.001) were found in youth with PTDs relative to healthy controls. Youth with PTDs endorsed elevated sleep disturbance with respect to: Disorders of Initiating and Maintaining Sleep, t(35)=-2.43, p=.02), Sleep-Wake Transition Disorders, t(37)=-3.04, p=.004), and Disorders of Excessive Somnolence, t(33)=-2.36, p=.02). No significant group differenceswere shown for Sleep Breathing Disorders, Disorders of Arousal, and Sleep Hyperhydrosis. There was a positive association between SDSC Total scores and YGTSS Total (p=.01, r=.56) and YGTSS Impairment scores (p=.03, r=.33). Finally, there was a positive relationship between SDSC Total and PTQ Total scores (p=.01, r=.61).

Conclusion: Findings suggest youth with PTDs are more likely to experience sleep disturbance than healthy children, particularly difficulties with sleep initiation and maintenance, abnormal movements during sleep, and daytime sleepiness. Further, there is a relationship between sleep disturbance and tic severity. Findings highlight the need for sleep screening and targeted sleep intervention in youth with PTDs.

Support: N/A

0998

HEALTH CARE UTILIZATION OF PEDIATRIC SLEEP DISORDERS IN CERNER HEALTH FACTS DATABASE

Al-Shawwa, B. Glynn, E. Hoffman, M. Ehsan, Z. Ingram, D. Children's Mercy Hospital, kansas City, KS.

Introduction: This study was aimed to identify health care utilization of sleep disorders in pediatrics and adults by using Cerner health facts database.

Methods: Health facts database has unidentified health records from all the participating facilities that have Cerner as their electronic medical records software. There are 68.7 million patients in the data warehouse with about 506.9 million encounters in about 100 healthcare systems. Sleep disorders are mostly seen in outpatient settings and therefore this study included outpatient records between the years 2010 to 2017.

Results: There were 20.5 million patients with total of 127.4 million outpatient encounters. In pediatric patients (ages 0-18 years), healthcare utilization of major sleep diagnoses per 100,000 encounters showed sleep related breathing disorders are the most commonly seen followed by parasomnia, insomnia, sleep movement disorders, hypersomnolence then circadian rhythm disorders (820.1, 258.1, 181.6, 68.3, 48.1 and 16.2 per 100,000 encounters). However, for adult patients the ranking was: sleep related breathing disorders, insomnia, sleep related movement disorders, hypersomnolence, parasomnia then circadian rhythm disorders (1352.6, 511.6, 166.3, 79.1, 25.7 and 4.2 per 100,000 encounters). Further analysis for the age groups showed bimodal pattern for sleep related breathing disorders and sleep movement disorders with the highest utilization were between the ages of 2-11 year and 40-60 years. Adolescents (age 12-18 years) showed increase utilization in the areas of circadian rhythm disorders.

Conclusion: Patients with sleep disorders have relatively low health care utilization despite high prevalence of these sleep disorders in the general population. This may highlight underrecognized sleep problem or decreased access to health care. In addition, this study highlights the effect of age on different sleep disorders which may have an impact on allocating resources. **Support:** None

0999

PREDICTORS OF NATURAL RESOLUTION OF BEHAVIORAL SLEEP PROBLEMS IN EARLY CHILDHOOD

Garrison, M. M.

Seattle Children's Research Institute, Center for Child Health, Behavior and Development, Seattle, WA.

Introduction: Primary care providers are often unsure which patients require treatment for behavioral sleep problems of early childhood vs. which are likely to improve without intervention. Given limited treatment resources as well as the time and cost burden that intervention can pose for families, it may be helpful to identify the predictors of children whose behavioral sleep problems are likely to resolve -- and stay resolved -- without active intervention.

Methods: Here, we use the control arm of a randomized trial of 2.5 to 5 year-old community-recruited children with behavioral sleep problems to examine the natural history of behavioral sleep problems and the predictors of resolution. Of 217 families in the control arm, 146 had inadequate mean sleep duration at baseline (< 10.5 hours/night per diary) and were eligible for inclusion in the analysis. Data were drawn from sleep diaries and parent report surveys conducted at baseline, 3 and 12 months. Improvement was defined as increased duration by either at least 60 min from baseline or by 30 min to at least 10.5 hours/night. We conducted an exploratory logistic regression to identify predictors of natural resolution. Results: Of the 146 eligible families, 130 had follow-up data at both 3 and 12 months and were included in this analysis. Of those 130, only 22 (17%) had substantially improved sleep by 3 months follow-up, and 10 of these continued to have improved sleep duration at 12 months. The strongest predictors of natural improvement both at 3 and 12 months were female child sex, having either infant or school-aged siblings, CBQ low-intensity pleasure score (i.e., children who tend to respond positively to cuddling, reading, and other gentle activities), mean duration of sleep onset latency per diary, parentreported parasomnias on the CSHQ, and age under 4 years.

Conclusion: These findings suggest that contrary to common beliefs of both primary care providers and parents, relatively few children will experience a clinically meaningful improvement in sleep duration without intervention.

Support: This work is supported by a grant from the National Institutes of Health, Eunice Kennedy Shriver National Institute of Child Health & Human Development (NICHD): 5R01HD071937.

1000

SOCIAL MEDIA USE AND ACTIGRAPHIC MEASURES OF SLEEP TIMING AMONG HIGH-RISK ADOLESCENTS

Hamilton, J. L.¹ Goldstein, T. R.² Sewall, C.³ Zelazny, J.⁴ Rode, N.² Gibbons, B.² Franzen, P. L.²

¹University of Pittsburgh, Pittsburgh, PA, ²Psychiatry, University of Pittsburgh, PA, ³Social Work, University of Pittsburgh, PA, ⁴Nursing, University of Pittsburgh, PA.

Introduction: Social media use is a risk factor for poor sleep among adolescents. It remains unclear whether social media use

before bed impacts later sleep timing or whether youth turn to social media because of sleep problems, which impacts sleep timing. No study to date has examined this relationship using prospective designs and objective sleep measures among high-risk adolescents, who may be particularly vulnerable to social media use.

Methods: As a preliminary test of this relationship, 25 adolescents and young adults in an intensive outpatient program for depression and suicidality completed baseline measures of social media use and wore actiwatches for up to three months. Social media use included: 1) minutes of use within 2 hours of going to sleep, and 2) frequency of social media use due to difficulty falling or staying asleep. To examine social media as a predictor of sleep timing over the next month, actigraphic measures of sleep timing (i.e., onset) were used in the first month after baseline.

Results: Multilevel modeling indicated that higher levels of social media use in the 2 hours before bed (mean = 46.94 minutes) predicted later sleep timing (B = .02; SE = .01; p= .003). Similarly, higher frequency of social media use due to perceived sleep problems predicted later sleep timing (B = .22; SE = .11; p= .04). Models covaried for age, gender, and prior-day depressed mood. When simultaneously entered, only minutes of social media use in the 2 hours before bed significantly predicted later sleep timing.

Conclusion: Findings suggest that the relationship between social media use and sleep timing among adolescents with depression and suicidality may be driven by both social media use before bed and media use due to sleep problems. Research assessing objective social media use and daily relationships are needed to further disentangle this relationship.

Support: Jessica L Hamilton is supported by a T32 fellowship from NHLBI (HL082610; PI: Buysse). This research is supported by grants from the American Foundation for Suicide Prevention and University of Pittsburgh Clinical and Translational Science Institute.

1001

INTRAVENOUS FERRIC CARBOXYMALTOSE FOR RESTLESS LEGS SYNDROME IN CHILDREN AND ADOLESCENTS

DelRosso, L. M.¹ Ferri, R.² Allen, R. P.³ Chen, M. L.¹ Kotagal, S.⁴ Picchietti, D.⁵

¹Seattle Children's Hosptial, Seattle, WA, ²Oasi Research Institute, Troina, ITALY, ³Johns Hopkins, Baltimore, MD, ⁴Mayo Clinic, Rochester, MN, ⁵Carle Illinois College of Medicine, Champaign, IL.

Introduction: Substantial scientific evidence implicates brain iron deficiency in the pathophysiology of restless legs syndrome (RLS). Current clinical guidelines recommend oral and intravenous iron (IV) in the treatment of both adult and pediatric RLS but studies using ferric carboxymaltose (FCM) are lacking in children and adolescents.

Methods: Retrospective case series of children and adolescents with RLS treated with IV FCM who had serum ferritin levels <50 µg/L. Patients were offered a single dose of IV FCM, 15 mg/ kg if weighting <50 kg or 750 mg if weighting >50 kg. Iron profile, serum ferritin, and severity assessment by the International Restless Legs Study Group severity scale (IRLS) were collected pre- and post-infusion. Clinical Global Impression Scale (CGI) was used instead of the IRLS for children. Phosphorus level and adverse effects were assessed post-infusion in all patients. Age and sex-matched children with RLS treated with oral iron supplementation (mean dosage 1.5 ± 0.62 mg/kg/day) were included as a comparison group.

Results: Twenty-eight subjects (15 females, mean age 11.5 years, SD 4.23) and 24 controls were included. Baseline ferritin levels were not significantly different from those of controls but increased significantly from 13.9±7.02 to 112.9±12.00 µg/L after 8 weeks from infusion (p<0.000001), when they were also significantly higher than control values (34.2±21.64 µg/mL, p<0.000001). Transferrin saturation increased from 22.8±9.77% to 31.7±6.81% (p<0.0001), total iron binding capacity decreased from 366.7±51.32 to $302.0\pm37.83 \ \mu g/dL \ (p<0.0000035)$. RLS was reported to be resolved or improved in all children treated with IV iron (vs. 62.5% of controls) while none of them reported no change (vs. 37.5% of controls; Chi-square test 9.84, p<0.002). IRLS Score decreased in adolescents from 30.7±22.68 to 3.2±4.21 (p<0.000008) while CGI-I was "very much improved" in six children and "much improved" in four. Side effects were reported in 17.8% of patients treated IV and 20.8% controls (Chi-square 0.0169, p=0.897). FCM side effects included lightheadedness and gastrointestinal discomfort. Post IV phosphorus levels were normal in all participants.

Conclusion: This open-label, observational and retrospective study indicates that FCM IV infusion is an effective treatment for pediatric RLS with higher efficacy than oral iron supplementation. **Support:**

1002

ENDORSEMENT OF SLEEP PROBLEMS INDEXES AUTISM SEVERITY IN CHILDREN AND ADOLESCENTS: EVIDENCE FROM A LARGE COMMUNITY SAMPLE

Saletin, J. M.¹ Koopman-Verhoeff, M.² Han, G.¹ Barker, D. H.¹ Carskadon, M. A.¹ Anders, T. F.¹ Sheinkopf, S. J.¹ ¹Brown University, Providence, RI, ²E.P. Bradley Hospital Sleep Research Laboratory, Providence, RI, ³Brown University, Providence, RI, ⁴Brown University, Providence, RI.

Introduction: Individuals with autism spectrum disorder (ASD) often experience sleep problems. A reliance on case-control studies rather than dimensional samples limit our ability to understand how sleep problems distinguish diagnosis and severity of ASD. To address this need, we present preliminary findings from a large community sample of individuals with heterogeneous autism phenotypes.

Methods: All participants (≤ 21 years) were selected from the Rhode Island Consortium for Autism Research and Treatment (RI-CART) (final n= 977; 233F; 11.27±4.13 years), a public-private-academic registry of families in Rhode Island affected by ASD-like symptoms. Participants completed the Autism Diagnostic Observation Schedule, 2nd Edition to confirm the presence of diagnosable ASD. Each caretaker also completed dimensional measures of functional impairment: Social Responsiveness Scale, 2nd edition and the Vineland Adaptive Behavior Scale (2nd/3rd editions). Caretakers were asked whether the participant suffered current/past sleep problems: yes/no. All analyses are adjusted for age, sex, race, ethnicity, caregiver education, and scale-version (e.g., 2nd/3rd ed., where applicable).

Results: Endorsement of sleep problems distinguished ASD diagnosis: a confirmed diagnosis of ASD was associated with greater prevalence of sleep problems compared to ASD diagnosis (OR: 1.58; 95% CI: 1.05,2.38; p = .028). Across the sample, endorsement of sleep problems was associated with impairments in adaptive behavior (b = -4.73; 95% CI: -7.47,-2.00045; p = .001) and social responsiveness (b = 6.72; 95% CI: 3.27,10.16; p < .001).

Conclusion: These data from a heterogenous community sample provide evidence for a link of sleep to the phenomenology of ASD.

While the search for better diagnostic indicators of ASD continues, we recommend that clinicians consider a brief assessment of sleep behaviors of patients with such neurodevelopmental conditions as autism.

Support: Simons Foundation Autism Research Initiative, Hassenfeld Child Health Innovation Institute at Brown University. K01MH109854 (JMS), NIGMS Advance CTR (JMS). KNAW Ter Meulen Grant (MEKV).

1003

SLEEP SPINDLE ABNORMALITIES IN YOUTH WITH PTSD

Peterson, B. Castelnovo, A. Riedner, B. Herringa, R. Jones, S. University of Wisconsin School of Public Health, Madison, WI.

Introduction: Sleep disturbance is central to the phenomenology of PTSD across the lifespan with up to 90% of youth with PTSD reporting sleep disturbance. Subjective sleep dysfunction has also been linked to the development, maintenance and severity of the disorder. However, to date there have been no objective EEG assessments of sleep in youth with PTSD, and little is known about how the disease impacts specific sleep features.

Methods: Ten youth with PTSD (aged 14.5 \pm 3.2; CAPS-CA score 60.5 \pm 25.3) and ten age-and sex-matched typically developing youth (TD) (aged 14.7 \pm 3.2) completed two non-consecutive overnight high-density EEG (256-channel) polysomnography sleep studies. Prior to sleep on one night, participants performed an emotion processing task. Group differences in sleep macrostructure variables were assessed with two-way ANOVA, and group differences in all-night spectral density were assessed using unpaired t-tests. An automatic algorithm was used to detect spindle amplitude, duration, and density topographically. Statistical non-parametric mapping (SnPM) cluster testing was used to determine significantly different topographic differences between groups.

Results: No significant group differences were observed in sleep macrostructure variables. All-night spectral density analysis revealed increased power in PTSD youth relative to TD youth in the sigma band on both task and baseline nights. PTSD youth showed higher spindle duration, higher integrated spindle activity, and higher spindle amplitude globally both nights relative to TD youth. The increase in spindle duration achieved significance in a robust frontal cluster on both nights (43-channel cluster (p = .044) on baseline night, 66-channel cluster (p = .019) on task night).

Conclusion: Structural and functional abnormalities of the prefrontal cortex are a prominent feature of pediatric PTSD. The observed increase in spindle duration may represent another marker of impaired cortical function in youth with PTSD reflecting a failure of cortical inhibition of the thalamically-generated spindle rhythm.

Support: K08 MH100267 to RH, Wisconsin Institute for Sleep and Consciousness Pilot Award to SJ

1004

SMITH-<MAGENIS SYNDROME (SMS) CIRCADIAN ABNORMALITIES AND BIOLOGICAL RHYTHMS

Brooks, J. Gibson, M. Kite, K. Czeisler, E. Fisher, M. Xiao, C. Polymeropoulos, C. Polymeropoulos, M. Vanda Pharmaceuticals Inc., Washington, DC.

Introduction: SMS is a rare neurodevelopmental disorder that manifests with craniofacial abnormalities, behavioral disturbances, and a severe sleep disorder. It has been reported that many SMS

patients have an inverted melatonin secretion pattern (peaking during the daytime) although a small minority have near normal patterns. The goal of this study was to better characterize the intraand inter-patient variability of melatonin secretion patterns and investigate a potential relationship with sleep behavior in SMS patients.

Methods: In this observational study, sleep behaviors of patients (N=8, 1 female, ages: 7 - 35) with SMS were characterized through caretaker surveys. On 3 separate occasions, patients had hourly serum melatonin levels sampled for 36 hours. From these data, peak serum melatonin concentration and time of peak concentration were determined. Inter- and intra-patient variability was characterized by zero lag correlation of the melatonin concentration timeseries across and within patients, respectively. The relationship between peak melatonin concentration, peak time, and sleep latency was analyzed by a generalized linear model, GLM.

Results: Peak melatonin concentrations varied across SMS patients with a range of 3.55pg/ml - 49.65pg/ml (mean $14.18 \pm 15.19pg/ml$). Time of peak melatonin concentrations ranged from 0400h-2100h (mean $1422 \pm 6h$). Correlation coefficients characterizing intra-patient variability ranged from -0.0098 to 0.89 (mean 0.55 ± 0.2533). Correlation coefficients characterizing inter-patient variability ranged from 0.18 ± 0.52). Sleep latency ranged from 8.4min - 36.35min (mean of 21.99 ± 9.77 min). GLM analysis demonstrated a significant, positive effect of peak time with sleep latency (p=0.022).

Conclusion: Consistent with previous findings, our study confirms that SMS patients have abnormal circadian rhythms. Our work extends this body of literature by demonstrating a significant degree of inter-patient variability with relatively stable intra-patient variability. Preliminary evidence suggests that the timing of melatonin peak may be related to sleep onset latency.

Support: This work was supported by Vanda Pharmaceuticals Inc.

1005

CHARACTERIZING SLEEP AND GLYCEMIA IN EMERGING ADULTS WITH TYPE 1 DIABETES

Griggs, S. Redeker, N. S. Grey, M. Yale University School of Nursing, West Haven, CT.

Introduction: Type 1 Diabetes (T1D) affects 1.25 million Americans, and only 14% of emerging adults (ages 18-30 years) achieve targets for glycemic control (A1C < 7.0%). Sleep deficiency, less than 6.5 hours total sleep time (TST), is associated with poorer glycemic control.

Methods: Emerging adults with T1D wore a wrist actigraph and their own or provided continuous glucose monitor (CGM) concurrently 24 hours/day for 6-8 days. Participants completed a 10-minute psychomotor vigilance test (PVT) on a device, 3-minute Trail Making Test on paper, and questionnaires including twice daily Pittsburgh sleep diaries in Research Electronic Data Capture (REDCap). TST, sleep onset latency (SOL), sleep efficiency (SE), wake after sleep onset (WASO), and sleep fragmentation index (SFI) were determined via actigraphy, glycemic control via A1C, and glucose variability via CGM. The purpose of this descriptive study was to explore associations between TST, sleep variability (SD of TST), neurocognitive function (psychomotor vigilance and executive function) and diabetes outcomes (glycemia and distress). Results: The sample included 36 emerging adults (mean age 22.8±3.1; 30.6% male; 91.7% White, 86.1% non-Hispanic; A1C mean 7.1±1.0%, BMI 27.3±4.8 kg/m²). Mean TST was 7.1±1.2 hours. SOL was 19.7±13.5 minutes. SE was 85.5±4.6%. WASO was 34.7±18.2 minutes, and SFI was 17.7±6.2. Shorter TST was associated with more severe sleepiness (r=-0.48,p=0.004) and more diabetes distress (r=-0.37, p=0.03). More sleep variability was associated with more severe sleepiness (r=0.36, p=0.03), longer response times (RT) \geq 500ms (*rho*=0.39, *p*=0.02) measured via PVT, more nocturnal glucose variability (r=0.38, p=0.04), greater mean daily differences in glucose levels (r=0.42, p=0.02). Shorter mean RT was associated with more time in glucose range (rho=-0.37, p=0.04).

Conclusion: Improving TST and sleep variability are potential therapeutic targets to improve glucoregulation in this high-risk population. Researchers should consider within-person multi-level modeling to inform our understanding of the true nature of the sleep-glucose association in emerging adults with T1D.

Support: T32 NR0008346, Sigma Theta Tau International, Dexcom provided continuous glucose monitors (G4) free of charge for participants who did not have their own device.

1006

SLEEP PROBLEMS AND RISK OF CANCER INCIDENCE AND MORTALITY IN THE CARDIOVASCULAR HEALTH STUDY (CHS)

Sillah, A.¹ Biggs, M.² Nieto, J.³ Watson, N.⁴ Gozal, D.⁵ Peters, U.⁶ Li, C.⁶ Thornton, T.¹ Phipps, A.¹

¹University of Washington School of Public Health, Seattle, WA, ²University of Washington, Seattle, WA, ³Oregon State University College of Public Health and Human Sciences, Corvallis, OR, ⁴University of Washington School of Medicine, Seattle, WA, ⁵The University of Missouri School of Medicine, Columbia, MO, ⁶Fred Hutchinson Cancer Center, Seattle, WA.

Introduction: Even in the absence of a formal diagnosis, sleep problems (SP) are frequently indicative of an underlying sleep disorder,

such as obstructive sleep apnea, which may be adversely associated with cancer risk and cancer outcomes.

Methods: We assessed the association of self-reported SP with incident cancer (N=4,997, excluding prevalent cancers) and cancer mortality (N=5849) among the participants of Cardiovascular Health Study (CHS), a population-based study of adults aged >=65 years recruited from 4 US communities. Participants reported SP (daytime sleepiness, observed apnea and snoring) yearly from 1989-1994; these self-reported symptoms have been validated against objective sleep measures assessed within a subset of CHS participants (n= 1240) who received a home polysomnography as part of the Sleep Heart Health Study. Cancer incidence was ascertained through linkage with state cancer registries through 2005; cancer specific death was adjudicated through 2015. We used Cox proportional hazards regression to calculate hazard ratios (HR) and 95% confidence intervals (CI) for associations of baseline SP with subsequent cancer incidence and cancer mortality, adjusting for baseline sociodemographics, lifestyle factors, and medical history.

Results: The mean age (SD) of the study population was 73 (6) years, 56% were female, and 84% were white. The prevalence of SP was 17% for daytime sleepiness, 8% for observed apnea, and 24% for snoring; 63% reported none of the 3 SP. Overall, 1,130 first incident cancers and 1,014 cancer deaths were identified over median follow-up of 12 and 13 years, respectively. Compared to participants who reported no SP, the risk of incident cancer was inversely associated with daytime sleepiness (HR 0.86 [95% CI 0.70-1.04]), observed apnea (HR 0.74 [0.56-1.00]), and snoring (HR 0.80 [0.68-0.95]). Cancer mortality HR (95% CI) estimates were 1.00 (0.82-1.21) for daytime sleepiness, 0.77 (0.57-1.04) for observed apnea, and 0.88 (0.74, 1.04) for snoring.

Conclusion: Symptoms indicating SP reported at baseline were not associated with increased cancer incidence or cancer mortality. Ongoing analyses are focused on the impact of longitudinal SP (time dependent, cumulative average) to ensure an adequate latency period is incorporated into our analysis of the association between SP and cancer risk and mortality.

Support: NIHT32CA09488017

1007

SOCIODEMOGRAPHIC, LIFESTYLE AND DIETARY CORRELATES OF ACTIGRAPHY-MEASURED IRREGULAR SLEEP SCHEDULES IN THE MULTI-ETHNIC STUDY OF ATHEROSCLEROSIS

Huang, T.¹ Chung, J.¹ Reid, M.¹ Johnson, D. A.² Billings, M. E.³ Klerman, E. B.⁴ Redline, S.¹

¹Brigham and Women's Hospital, Boston, MA, ²Emory University, Atlanta, GA, ³Harborview Medical Center, Seattle, WA, ⁴Massachusetts General Hospital, Boston, MA.

Introduction: Increasing evidence links daily variability in sleep schedules to increased cardiometabolic risk. Little is known, however, regarding sociodemographic and behavioral correlates of irregular sleep schedules that may help identify causes or consequences of irregular sleep.

Methods: Among 1,946 participants from the Multi-Ethnic Study of Atherosclerosis, we examined the cross-sectional associations of irregular sleep schedules with sociodemographic, lifestyle, dietary factors, and actigraphy-based indices of rest-wake rhythms using multiple linear regression with adjustment for age, sex, race/ethnicity, education, income, marital status and work schedules. Sleep regularity was assessed using standard deviations (SD) in actigraphy-measured sleep duration and sleep onset timing across 7 days.

Results: Compared to Whites, the 7-day sleep duration SD (95%) CI) was 17.4min (12.6, 22.2) higher in African-Americans, 10.4min (4.8, 16.0) higher in Hispanics and 7.9min (1.3, 14.4) higher in Chinese. Shift versus regular work was associated with 11.4min (5.1, 17.7) higher sleep duration SD. Irregular sleep duration was associated with lower income (p=0.006), higher depressive symptoms (p<0.0001), higher BMI (p=0.004) and current smoking (p=0.06). Higher sleep duration variability was associated with lower Alternative Healthy Eating Index (p=0.01), mainly due to suboptimal intakes of fruits, whole grains and nuts/legumes. No associations were observed for age, sex, education, marital status or number of meals per day. While sleep duration variability was not associated with self-reported physical activity level or actigraphymeasured 7-day mean activity count, sleep duration SD was inversely associated with relative amplitude (difference between the most versus the least active period; p<0.0001) and inter-daily stability (synchronization between rest-activity patterns and environmental zeitgebers; p<0.0001) of 24-h rest-activity patterns. Similar results were observed for sleep onset timing SD.

Conclusion: Substantial differences by sociodemographic factors exist regarding the consistency of day-to-day sleep schedules. Irregular sleep schedules are associated with overall circadian disruption across the day and some unhealthy lifestyle behaviors. Future studies are needed to understand temporal relationships of the observed associations.

Support: NIH grants K01HL143034, R35HL135818

1008

BRAIN AGE BASED ON SLEEP ENCEPHALOGRAPHY IS ELEVATED IN HIV+ ADULTS ON ART

Leone, M. J. Sun, H. Boutros, C. Sullivan, L. Thomas, R. J. Robbins, G. Mukerji, S. Westover, M.

Massachusetts General Hospital, Boston, MA.

Introduction: Sleep EEG is a promising tool to measure brain aging in vulnerable populations such as people with HIV, who are high risk of brain aging due to co-morbidities, increased inflammation, and antiretroviral neurotoxicity. Our lab previously developed a machine learning model that estimates age from sleep EEG (brain age, BA), which reliably predicts chronological age (CA) in healthy adults. The difference between BA and CA, the brain age index (BAI), independently predicts mortality, and is increased by cardiovascular co-morbidities. Here, we assessed BAI in HIV+ compared to matched HIV- adults.

Methods: Sleep EEGs from 43 treated HIV+ adults were gathered and matched to controls (HIV-, n=284) by age, gender, race, al-coholism, smoking and substance use history. We compared BAI between groups and used additional causal interference methods to ensure robustness. Individual EEG features that underlie BA prediction were also compared. We performed a sub-analysis of BAI between HIV+ with or without a history of AIDS.

Results: After matching, mean CA of HIV+ vs HIV- adults were 49 and 48 years, respectively (n.s.). The mean HIV+ BAI was 3.04 years higher than HIV- (4.4 vs 1.4 yr; p=0.048). We found consistent and significant results with alternative causal inference methods. Several EEG features predictive of BA were different in the HIV+ and HIV- cohorts. Most notably, non-REM stage 2 sleep (N2) delta power (1-4Hz) was decreased in HIV+ vs. HIV- adults, while theta (4-8Hz) and alpha (8-12Hz) power were increased. Those with AIDS (n=19, BAI=4.40) did not have significantly

different BAI than HIV+ without AIDS (n=23, BAI=5.22). HIV+ subjects had higher rates of insomnia (56% vs 29%, p<0.001), obstructive apnea (47% vs 30%, p=0.03), depression (49% vs 23%, p<0.001), and bipolar disorder (19% vs 4%, p<0.001).

Conclusion: HIV+ individuals on ART have excess sleep-EEG based brain age compared to matched controls. This excess brain age is partially due to reduction in delta power during N2, suggesting decreased sleep depth. These results suggest sleep EEG could be a valuable brain aging biomarker for the HIV population. **Support:** This research is supported by the Harvard Center for AIDS Research HU CFAR NIH/NIAID 5P30AI060354-16.

1009

SLEEP APNEA AND COLORECTAL ADENOMA IN THE VETERAN POPULATION: A CASE-CONTROL STUDY

Guan, L.¹ Jiao, L.^{2,3,4} Malhotra, S.^{5,6}

¹Baylor College of Medicine, Houston, TX, ²Baylor College of Medicine, Department of Medicine, Houston, TX, ³Michael E. DeBakey VA Medical Center, Center for Innovations in Quality, Effectiveness and Safety, Houston, TX, ⁴Michael E. DeBakey VA Medical Center, Section of Gastroenterology, Houston, TX, ⁵Baylor College of Medicine, Department of Pediatric Pulmonary and Sleep Medicine, Houston, TX, ⁶Texas Children's Hospital, Houston, TX.

Introduction: Colorectal cancer is the third most common cancer in the United States, with over half of colorectal cancers estimated to be the result of modifiable risk factors. Studies relating sleep apnea (SA) and colorectal adenoma (CRA) are limited and the findings are equivocal. The objective of this study was to examine the association between SA and risk of CRA.

Methods: This was a retrospective cross-sectional case-control study of data collected from 460 veterans, ages 50-79, seen in the colonoscopy clinic at the Michael E. DeBakey VA Medical Center between 2014 and 2018. Information on demographics, sleep history, and co-morbidities were obtained through lifestyle questionnaire. Self-reported SA was diagnosed by a prior sleep study. Cases consisted of 297 participants had pathologically confirmed adenoma (including 117 participants having advanced CRA with villous component or diameter of polyp > 1 cm). Controls consisted of 173 polyp-free participants. The distribution of demographics and lifestyle factors were compared between CRA and non-CRA using the Student's t or chi-square tests. Odds ratios (OR) and 95% confidence intervals (CI) of CRA in association with CRA were calculated using univariate and multivariate unconditional logistic regression models. The confounding factors included age, sex, ethnicity, obesity, smoking status, alcohol use, hypertension, and sleep duration.

Results: Compared with non-SA, the multivariable OR (95% CI) for CRA was 0.92 (0.58-1.48); for non-advanced CRA was 1.14 (0.68-1.91), and for advanced CRA was 0.61 (0.32-1.17) in SA participants. Adjustment of sleep duration in the model did not change the risk estimates.

Conclusion: Sleep-study diagnosed SA was not associated with development of CRA in this veteran population. Further studies are needed to confirm this observation and incorporate the severity and treatment of SA, and undiagnosed SA in risk assessment.

Support: This research is supported in part by the Gillson Longenbaugh Foundation, and Golfers Against Cancer organization (to LJ), the Cancer Prevention Research Institute of Texas (CPRIT) (RP#140767, to LJ).

1010

INDIVIDUALS WITH METABOLIC SYNDROME AND UNRECOGNIZED SLEEP APNEA CAN BE IDENTIFIED BY AN EMPLOYER-SPONSORED HEALTHCARE PROGRAM AND AT-HOME SLEEP STUDY

Iakoubova, O. A. Tong, C. H. Arellano, A. R. Bare, L. A. Fragala, M. S. Devlin, J. J. Birse, C. E.

Nichols Institute, Quest Diagnostics, San Juan Capistrano, CA.

Introduction: Obstructive sleep apnea (OSA) is common in individuals with metabolic syndrome (MetS) and increases risk of cardiovascular (CVD) events. Once recognized, therapeutic interventions can reduce OSA severity and associated CVD risk. Of the 25 million Americans with OSA, 80% are unaware of their disease. To facilitate and improve diagnosis of OSA, diagnostic devices for at-home OSA testing have been developed in clinical studies and approved by FDA. We evaluated an employer-sponsored healthcare outreach program and at-home OSA testing as a means of identifying individuals likely to have OSA and referring them into care.

Methods: Nine-hundred individuals with MetS, positive OSA Berlin questionnaire score and no prior diagnosis of OSA, as determined by annual workplace screening and health claims, were invited to participate in the sleep program. Those who agreed to participate (9.9%) received a diagnostic device for at-home OSA testing. Apnea-hypoapnea index (AHI) results recorded on returned diagnostic devices were evaluated by a sleep specialist. A telephone consultation with a program physician then provided each participant with an explanation of test results and referral into care. Based on AHI we identified individuals with moderate (AHI 16-30) to severe (AHI >30) OSA and referred them to care.

Results: Of the 89 participating individuals, 21% had 3 MetS components, 53% had 4 components, and 20% had 5 components; 30% were diabetic; 83% had hypertension; and >50% were obese. Moderate to severe OSA was diagnosed in 52 (58%) of participants. Of those, 50% had moderate OSA and 50%, had severe OSA. Among individuals with moderate to severe OSA, 29 (56%) had a physician consultation and were referred to treatment.

Conclusion: A personalized employer-sponsored healthcare outreach program identified individuals with unrecognized OSA and referred them into care. **Support:**

1011

SLEEP DURATION MODERATES THE ASSOCIATION BETWEEN NEXT-DAY PHYSICAL ACTIVITY AND PAIN INTENSITY AMONG WOMEN WITH FIBROMYALGIA

Whibley, D.^{1,2,3} *Williams, D. A.*² *Clauw, D. J.*² *Kratz, A. L.*³ ¹Epidemiology Group, School of Medicine, Medical Sciences and Nutrition, University of Aberdeen, Aberdeen, UNITED KINGDOM, ²Chronic Pain & Fatigue Research Center, Ann Arbor, University of Michigan, MI, ³Department of Physical Medicine and Rehabilitation, Ann Arbor, University of Michigan, MI.

Introduction: Suboptimal sleep has been consistently associated with greater next-day pain intensity among women with fibro-myalgia. In contrast, associations between physical activity and same-day pain in this population are contradictory. Given this inconsistency, we aimed to determine whether the daily physical activity-pain association is modified by sleep parameters.

Methods: This micro-longitudinal study used data collected using wrist-worn triaxial accelerometers over seven consecutive days by 44 adult women with fibromyalgia. Derived variables included sleep duration, sleep onset latency (SOL), wake after sleep onset (WASO), sleep efficiency (SE) and maximum daily physical activity count. Participants also completed digital diaries of refreshed sleep upon awakening (scale 0-100), and pain intensity (5x/day, scale 0-100). Multilevel linear regression models with interaction terms were used to investigate moderating effects of sleep on the next-day person-centered maximum physical activity-average pain intensity association.

Results: The sample mean age was 44 (SD 14). A total of 304 days of data were available for analysis. Mean sleep duration was 471min(SD 66); mean SOL 10min(SD 10); mean WASO 31min(SD 17), and mean SE 90%(SD 5). Sleep duration moderated the next-day maximum physical activity-pain association (Wald statistic p=0.01). After nights of both shorter (<7 hours) and longer sleep (>8 hours), higher levels of next-day maximum physical activity (compared to the participant's overall study average) were associated with days of greater average pain. In contrast, after nights of 7-8 hours of sleep, higher levels of next-day maximum physical activity were associated with days of lower average pain.

Conclusion: An association between higher maximum physical activity and lower levels of pain was only observed after nights of 7-8 hours sleep. Engaging in physical activity is recommended for fibromyalgia-related pain management. Optimizing sleep duration may be useful in minimizing physical activity-related pain in this clinical population.

Support: DW is supported by a Foundation Fellowship Versus Arthritis. The study that provided data was supported by the National Institute of Arthritis and Musculoskeletal and Skin Diseases of the National Institutes of Health (award number K01AR064275;PI:ALK). The Michigan Institute for Clinical & Health Research provided subject recruitment support through the UMHealthResearch.org website (MICHR:NIH award number UL1TR002240).

1012

UROLOGIC FEATURES RELATED TO THE FIRST UNINTERRUPTED SLEEP PERIOD (FUSP) IN NOCTURIA

Monaghan, T. F.¹ Agudelo, C. W.¹ Rahman, S. N.¹ Michelson, K. P.¹ Lazar, J. M.¹ Everaert, K.² Weiss, J. P.¹ Bliwise, D. L.³

¹SUNY Downstate Health Sciences University, Brooklyn, NY, ²Ghent University Hospital, Ghent, BELGIUM, ³Emory University School of Medicine, Atlanta, GA.

Introduction: In nocturia, longer FUSP (time to first void) correlates with better quality sleep (Bliwise et al, JCSM 2015;11:53-5) and, with treatment, longer FUSP is associated with decreased nightly voids (Epstein et al, Neurourol Urodyn 2018;37:186-91). We examined urologic correlates of FUSP in an outpatient nocturia population without comorbidities (CHF, OSA, ESRD, diuretics). **Methods:** Participants (n=119; men) kept a home flow/volume diary, tracking clock time and quantity of each urination across a 24-hr period. FUSP was defined as time between going to bed and time of first void. We analyzed the urine volume at first nocturnal void (FNVV) (i.e., at end of FUSP). We also analyzed all nighttime volumes and divided by reported hours of sleep to impute nocturnal urine production (NUP) (in ml/hr, classified as high [>90 ml/hr] [n=49] vs low [<90 ml/hr] [n=60])—a measure correlated with number of nocturia episodes (van Doorn et al, J Urol

2014;191:1034-9). Nocturnal maximal voided volume (NMVV) at any single nocturnal void defined maximal functional nocturnal bladder capacity. Data were analyzed non-parametrically.

Results: For 53 of 119 patients, FNVV was identical to NMVV. This was more likely in patients with NUP >90 ml/hr vs <90 ml/ hr (59% vs 40%, p=.046). High (vs low) NUP rates were also associated with higher FNVV (300 [225-420] vs 135 [100-200] ml, p<.001), as well as higher number of voids (3 vs. 2, p=.03).

Conclusion: For nearly half of these nocturia patients, the volume at first void occurred at their maximal nocturnal volume. In nocturia, higher FNVV also reflects greater overall nocturnal volume of urine produced, and excess urine volume (as opposed to insufficient bladder capacity) likely plays a central role in the pathogenesis of nocturia in these patients. The extent to which these higher initial volumes represent free-water vs solute-driven clearance is currently under investigation. **Support:** N/A

1013

CARDIOVASCULAR BIOMARKERS AND PATHOPHYSIOLOGICAL INSIGHTS INTO OBSTRUCTIVE SLEEP APNEA DURING ACUTE CORONARY SYNDROME

Cheong, C. S.¹ Aung, A. T.² Chan, S.³ Lee, C.² ¹Department of Otolaryngology - Head & Neck Surgery, National University Hospital, Singapore, SINGAPORE, ²Department of Cardiology, National University Heart Centre, Singapore, SINGAPORE, ³Department of Medicine, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, SINGAPORE.

Introduction: Obstructive sleep apnea (OSA) is prevalent and carries prognostic implication in patients with acute coronary syndrome (ACS). The relative contribution of pathophysiological mechanisms in ACS towards OSA is not well-studied. We examined the correlation between severity of OSA and myocardial necrosis, inflammation, wall stress, and fibrosis.

Methods: A total of 89 patients admitted with ACS underwent an overnight sleep study during index admission. Plasma levels of peak troponin I, high-sensitivity C-reactive protein (hs-CRP), N-terminal pro-brain natriuretic peptide (NT-proBNP), and suppression of tumorigenicity 2 (ST2) were prospectively analyzed. Two patients diagnosed with central sleep apnea were excluded.

Results: The recruited patients were divided into no (AHI <5 events/hour, 9.2%), mild (5-<15, 27.6%), moderate (15-<30, 21.8%), and severe (≥30, 41.4%) OSA. The respective Epworth Sleepiness Scale scores were 3.8±3.7, 5.3±4.9, 4.0±2.8, and 5.5±4.5 (p=0.734). Compared to the no, mild and moderate OSA groups, the severe OSA group had a higher body mass index (p=0.005). They were also more likely to present with ST-segment elevation ACS (vs non-ST-segment elevation ACS) (p=0.041), have undergone previous coronary artery bypass grafting (p=0.013), demonstrate complete coronary occlusion during baseline coronary angiography (p=0.049), and have a larger left atrium diameter measured on echocardiography (p=0.029). Likewise, the severe OSA group had higher plasma levels of troponin I (10584±13078, 11699±20130, 19280±30670, 37571±31269 µg/L; p=0.017), hs-CRP (8.1±9.2, 23.1±52.3, 9.3±17.1, 39.4±44.7 mg/L; p=0.004), and NT-proBNP (667±604, 765±856, 636±728, 1395±1220 pg/ mL; p=0.004), but not ST2 (p=0.10). After adjusting for the effects of the confounding variables, severe OSA was independently associated with troponin I (i.e., myocardial necrosis; OR 1.00003, 95%

CI 1.000013-1.000048; p=0.001) and NT-proBNP (i.e., myocardial wall stress; OR 1.00081, 95% CI 1.00021-1.00141; p=0.008). Conclusion: Severe OSA during the acute phase of ACS was associated with extensive myocardial necrosis and myocardial wall stress, but not with inflammation and myocardial fibrosis. Support: Nil

1014

RELATIONSHIP BETWEEN LUNG FUNCTION AND OBSTRUCTIVE SLEEP APNEA IN CYSTIC FIBROSIS

Shakkottai, A. Nasr, S. Z. Hassan, F. O'Brien, L. M. Chervin, R. D.

University of Michigan, Ann Arbor, MI.

Introduction: The frequency of obstructive sleep apnea (OSA) may be high among patients with cystic fibrosis (CF), a life-shortening, genetic respiratory disease that affects approximately 30,000 Americans. Yet, the potential relationship between OSA and lung function has not been thoroughly explored.

Methods: Single-center retrospective review of polysomnography (PSG) results from 2009-2017 in referred patients with CF and available pulmonary function data (PFTs) obtained at time of PSG and at 3, 6, 9, and 12-months prior.

Results: Mean ages were 11.1 ± 3.9 (sd) and 37.1 ± 14.1 years, among 18 children and 16 adults, respectively. Mean body mass index (BMI) was normal in both groups ($62.5\pm26.6\%$ in children; 25.1 ± 6.4 kg/m² in adults). Twenty-six subjects (76%) had OSA (apnea-hypopnea index >1 in children, ≥ 5 in adults). Mean forced expiratory volume in 1 second percent predicted (FEV1 PPD) was higher among subjects with vs. without OSA at PSG and at each time-point in the year prior, independent of age and BMI at PSG (longitudinal mixed effects model, β =19.0, SE=8.1, p=0.028). While FEV1 PPD remained unchanged in the non-OSA group, FEV1 PPD at PSG was lower, in comparison to the year prior in subjects with OSA, with the greatest difference observed at 9-months prior to PSG (2-sample t-test, difference of -6.6% vs 0.6% in OSA vs. non-OSA groups respectively, p=0.078).

Conclusion: The PFTs, as daytime markers of CF lung disease severity, do not seem to reliably predict risk for OSA. In our sample, CF patients with vs. without OSA had better PFTs at baseline but they also showed a greater tendency for decline in PFTs over the year prior to OSA diagnosis. Larger sample size and longer duration of assessment may help, going forward, to assess any potential adverse impact of OSA on lung function decline.

Support: NIH Training Grant (T32NS007222, F32HL145915)

1015

SLEEP DURATION AND PHYSICAL AND MENTAL HEALTH AMONG ADULT SURVIVORS OF CHILDHOOD CANCER: RESULTS FROM THE ST. JUDE LIFETIME COHORT

Lubas, M. M.¹ Mandrell, B. N.² Ehrhardt, M. J.¹ Ness, K. K.¹ Srivastava, D.³ Robison, L. L.¹ Hudson, M. M.¹ Krull, K. R.¹ Brinkman, T. M.¹

¹St. Jude Children's Research Hospital, Department of Epidemiology and Cancer Control, Memphis, TN, ²St. Jude Children's Research Hospital, Department of Pediatric Medicine, Memphis, TN, ³St. Jude Children's Research Hospital, Department of Biostatistics, Memphis, TN.

Introduction: Sleep disturbances are prevalent among adult survivors of childhood cancer, though little is known about associations between sleep and health in this vulnerable population.

Methods: Survivors recruited from the St. Jude Lifetime Cohort (n=911; 52% female; mean age 34 years; 26 years post-diagnosis) completed surveys assessing habitual sleep patterns and mental health and underwent comprehensive physical examinations. A subset of survivors (n=491) completed sleep actigraphy. Short sleep duration was defined as sleeping <7 hours per night, assessed via self-report or actigraphy. Clinically-assessed health outcomes were defined as grade ≥2 using modified CTCAE criteria for cardiac, pulmonary, and renal conditions. Anxiety and depression were defined as scores $\ge 90^{\text{th}}$ percentile on the Brief Symptom Inventory-18. Covariates included childhood cancer treatment exposures, demographics, body mass index, and physical inactivity. Separate logistic or modified Poisson (common outcomes) regression models were computed for each health category to estimate odds ratios (OR) or relative risks (RR) and 95% confidence intervals (CI).

Results: Self-report and actigraphy-assessed short sleep was identified in 44% and 42% of survivors, respectively. However, these measures were weakly correlated (r=0.23). In adjusted multivariable models, self-reported short sleep was associated with higher risk of pulmonary conditions (RR=1.3, 95% CI=1.1-1.7), depression (OR=2.6, 95% CI=1.4-5.1) and anxiety (OR=3.4, 95% CI=1.6-6.8), while associations with cardiac (RR=1.10, 95% CI=0.94-1.30) and renal conditions (OR=1.30, 95% CI=0.79-2.13) were not significant. There were no significant associations between actigraphy-assessed short sleep and any of the health outcomes.

Conclusion: Habitual self-reported short sleep was associated with clinically ascertained adverse health outcomes. Although the temporality of these associations cannot be determined in this cross-sectional study, sleep is a modifiable health behavior and improving sleep may improve health in survivors. Measures of self-reported sleep may have unique value when assessing the relationship between sleep and health.

Support: CA225590, K. Krull Principal Investigator; CA195547, M. Hudson and L. Robison Principal Investigators; CA21765, C. Roberts, Principal Investigator

1016

MULTIPLE SLEEP DISTURBANCES AND HYPERTENSION RISK AMONG WHITE, BLACK, AND HISPANIC/LATINA WOMEN

Lunyera, J.¹ Park, Y. M.² Ward, J. B.³ Gaston, S. A.² Bhavsar, N. A.¹ Muntner, P.⁴ Sandler, D. P.² Jackson, C. L.^{2,5} ¹Duke University School of Medicine, Durham, NC, ²National Institute of Environmental Health Sciences, Research Triangle Park, NC, ³Social & Scientific Systems, Durham, NC, ⁴University of Alabama at Birmingham, Birmingham, AL, ⁵National Institute on Minority Health and Health Disparities, Bethesda, MD.

Introduction: Poor sleep has been associated with a higher risk of hypertension, but few prospective studies have included multiple sleep dimensions and few have investigated age differences or racial/ethnic disparities in this relationship among pre- and postmenopausal women.

Methods: To investigate the association between sleep disturbances and hypertension risk, we used data from women in the United States enrolled in the Sister Study who were aged 35 to 74 years at baseline (2003 to 2009) and did not have hypertension

at enrollment. Participants were followed through September 2017. Sleep duration, inconsistent weekly sleep patterns, sleep debt, frequent napping, and insomnia symptoms were reported at baseline. During follow-up, participants reported whether they were diagnosed by a healthcare provider with hypertension. Adjusting for sociodemographic characteristics, health behaviors, and health conditions including diabetes and depression, we used Cox Proportional Hazards regression to estimate hypertension risk among women with vs. without unfavorable sleep characteristics. We also investigated potential modification by race/ethnicity, age,and menopausal status.

Results: Of 33,175 women without hypertension at baseline (mean age \pm standard deviation: 53.9 \pm 8.8 years; 88.8% White, 6.4% Black, and 4.9% Hispanic/Latina), 19.9% developed hypertension over a median follow-up of 9.2 years (interquartile range: 7.6 to 10.9). After adjustment, insomnia symptoms (hazard ratio[HR]=1.08 (95% Confidence Interval [CI]: 1.03-1.15)) and insomnia symptoms combined with short sleep (HR=1.14 (95% CI: 1.06-1.23)) were associated with incident hypertension. While similar across race/ethnicity, these associations were stronger in younger (age <54 vs. \geq 54 years) and premenopausal vs. postmenopausal women (p-values for interaction <0.05).

Conclusion: Sleep disturbances related to insomnia were associated with an increased risk of hypertension, especially among younger and premenopausal women.

Support: This work was funded by the Intramural Program at the National Institutes of Health (NIH), National Institute of Environmental Health Sciences (NIEHS, Z1AES103325-01 [CLJ] and Z01 ES044005 [DPS]).

1017

AN INTEGRATED BEHAVIORAL THERAPY FOR POSITIVE AIRWAY PRESSURE ADHERENCE AND INSOMNIA REDUCES NOCTURIA FREQUENCY

*Fung, C. H.*¹ *Martin, J. L.*¹ *Dzierzewski, J. M.*² *Fiorentino, L.*³ *Stepnowsky, C.*³ *Song, Y.*¹ *Zeidler, M.*¹ *Mitchell, M.*⁴ *Vaughan, E. C.*⁵ *Huang, A.*⁶ *Markland, A.*⁷ *Josephson, K.*⁴ *Alessi, C.*¹

¹UCLA/VA Greater Los Angeles, North Hills, CA, ²Virgina Commonwealth University, Richmond, VA, ³University of California, San Diego, La Jolla, CA, ⁴VA Greater Los Angeles, North Hills, CA, ⁵Emory University, Atlanta, GA, ⁶University of California, San Francisco, San Francisco, CA, ⁷University of Alabama at Birmingham, Birmingham, AL.

Introduction: Nocturia is common among patients with both insomnia disorder and obstructive sleep apnea (OSA) and adversely affects quality of life. Within a randomized controlled trial testing an integrated behavioral therapy for positive airway pressure (PAP) adherence and insomnia for patients with coexisting OSA and insomnia disorder, we examined the impact of the integrated therapy on nocturia frequency.

Methods: Patients aged \geq 50 years with untreated OSA (apnea hypopnea index \geq 15) and chronic insomnia disorder were recruited from a VA medical center and randomized to 5 weekly individual integrated behavioral treatment sessions versus control (general sleep education). Nocturia frequency (self-reported average number of nocturia events per night) was assessed at baseline, 3 months, and 6 months. Linear regression models were used to examine relationships between objective PAP adherence (# nights used \geq 4hrs over the last 30 days) and Insomnia Severity Index (ISI) score (0 [none] - 28 [severe]) and nocturia frequency

(0 - 5+/night), and change in nocturia frequency associated with integrated therapy.

Results: Nocturia data were available for 112 participants (treatment=56, control=56; mean age 63 [SD 7], 95% male). Mean nocturia frequency (episodes/night) was 2.1 (SD 1.3; baseline), 1.6 (SD 1.1; 3 months), and 1.7 (SD 1.2; 6 months). Overall, higher PAP adherence (B=.029, p=.008) and ISI score improvement (B=0.05, p=.004) were associated with decreased nocturia frequency at 3-month follow-up. No differences were observed in nocturia frequency between treatment and control participants at baseline (p=.429). Integrated therapy reduced nocturia frequency at 3 months (B=-0.56, p=.020) but not at 6 months follow-up (B=-0.4, p=.081) compared to control.

Conclusion: Nocturia frequency improved with integrated behavioral therapy for PAP adherence and insomnia in veterans (primarily male) with co-existing OSA and insomnia. Additional studies are needed to examine the mechanisms underlying the relationship between the behavioral therapy for PAP adherence and insomnia and nocturia. **Support:** VA HSR&D; NIA; VA GRECC

1018

SLEEP AND FATIGUE: EXAMINING THE IMPACT ON COGNITIVE FUNCTION IN OLDER ADULTS LIVING WITH HIV

Frain, J. A.¹ Chen, L.²

¹Goldfarb School of Nursing, St. Louis, MO, ²Washington University, St. Louis, MO.

Introduction: Poor sleep affects 75% of older adults living with HIV, negatively impacting health. The purpose of this study was to examine the associations between sleep, fatigue and cognitive function in older adults living with HIV with well-controlled HIV virus. **Methods:** Forty-three adults aged 50 years and older living with HIV were recruited for this study. Participants provided demographic and health information. Participants wore actigraph watches continuously for one week, while completing a daily sleep diary, fatigue instrument, and Epworth Sleepiness Scale. After one week participants returned and completed the Pittsburgh Sleep Quality Index (PSQI) and performed cognitive testing including the NIH Toolbox Cognition Battery and the Montreal Cognitive Assessment (MoCA).

Results: Fluid cognition (measured with the Cognition Battery) positively correlated with hours of sleep measured via actigraph the night immediately prior to testing (p = .008), but not by average hours slept over the week. Average daily fatigue and daytime sleepiness were also correlated with fluid cognition (p = .012, p = .032 respectively). Similar results were found when cognition was measured using the MoCA, with sleep (p = .001), average fatigue (p = .017), and daytime sleepiness (p = .028) all correlated with cognition. When sleep was measured subjectively, Pearson correlation indicated that there was a statistically significant negative relationship of moderate strength between global sleep and cognitive function (r = .47, p = .015).

Conclusion: The study provides evidence that poor sleep, measured objectively or subjectively, is associated with cognitive impairment. Despite we-controlled HIV virus, 86% of study participants had global sleep scores indicating poor sleep. Sleep measured objectively resulted in less nightly sleep than by subjective measure, 4.5 vs 6.07 average hours per night. Studying effective interventions to improve sleep should be a next step as a way of improving cognitive function for this population.

Support: This study was supported through a grant funded by Sigma Theta Tau International and the National Gerontological Nurses Association.

1019

SUBJECTIVE SLEEP QUALITY AND SLEEP RECOMMENDATIONS RECEIVED BY PATIENTS WITH CANCER AND DEPRESSION

Price, S. N. Trejo, J. I. Halaby, L. M. Guzman, D. Liu, Y. Verlaque, R. Hamann, H. A. Weihs, K. L. University of Arizona, Tucson, AZ.

Introduction: Diagnoses of cancer and depression are independent predictors of poor sleep, but less is known about subjective sleep quality among patients with both of these potential risk factors or about recommendations made by physicians for improving sleep among this population. This study examines correlates of poor subjective sleep quality and sleep recommendations received by patients with cancer enrolled in the Collaborative Oncology Project to Enhance Depression Care (COPE-D), a collaborative care intervention to treat depression among patients with cancer.

Methods: Participants were 74 adult cancer survivors. Demographic and clinical characteristics, subjective sleep quality, and provider sleep recommendations were obtained by patient self-report prior to intervention. Sleep quality was evaluated using the Pittsburgh Sleep Quality Index (PSQI), general health status was measured using the PROMIS Global-10, and depressive symptoms were measured using the PHQ-9.

Results: 81% of patients reported significantly poor sleep quality (PSQI global scores >8) and 75.3% reported poor sleep efficiency (<85%). The strongest correlates of poor sleep quality were worse global mental (r=-.431, p<.01) and physical health (r=-.40, p=<.01). 63% reported pain interference with sleep at least once per week. Cancer type and stage, current cancer treatment, and depressive symptoms were not significantly associated with poor sleep quality (p's>.05). 12% of those reporting sleep disturbances since their cancer diagnosis had not discussed these problems with a medical provider. Among those who talked to their provider, 41.8% reported receiving sleep hygiene recommendations, 40.5% anti-depressants, 14.9% sedative-hypnotic medication (e.g. zolpidem, benzodiazepines), 10.8% cognitive behavioral therapy, 9.6% antihistamines, 6.8% melatonin, and 4.1% were recommended meditation or hypnosis.

port very high rates of poor subjective sleep quality, which was most strongly associated with global mental and physical health. Improved screening and patient-provider communication about sleep may be especially beneficial for this at-risk population. **Support:** Merck Foundation Alliance to Advance Patient-Centered

1020

Cancer Care

SLEEP AND GLYCEMIC CONTROL IN ADULTS WITH LONG-STANDING TYPE 1 DIABETES AND HYPOGLYCEMIA UNAWARENESS

Malone, S. K.¹ Peleckis, A. J.² Pack, A. I.³ Perez, N.¹ Yu, G.¹ Rickels, M. R.² Goel, N.⁴

¹Rory Meyers College of Nursing, New York University, New York, NY, ²Division of Endocrinology, Diabetes, and Metabolism, Department of Medicine, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, ³Division of Sleep Medicine, Department of Medicine, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, ⁴Biological Rhythms Research Laboratory, Department of Psychiatry and Behavioral Sciences, Rush University Medical Center, Chicago, IL. **Introduction:** Nocturnal hypoglycemia is life threatening for individuals with type 1 diabetes (T1D) due to loss of hypoglycemia symptom recognition (hypoglycemia unawareness) and impaired glucose counterregulation. These individuals also show disturbed sleep, which may result from glycemic dysregulation. Whether use of a hybrid closed loop (HCL) insulin delivery system with integrated continuous glucose monitoring (CGM) designed for improving glycemic control, relates to better sleep across time in this population remains unknown.

Methods: Six adults (median age=58y,T1D duration=41y) participated in an 18-month ongoing clinical trial assessing the effectiveness of an HCL system. Sleep and glycemic control were measured concurrently using wrist actigraphs and CGM at baseline (1 week) and months 3 and 6 (3 weeks) following HCL initiation. BMI and hemoglobin A1c (HbA1c) were collected at all timepoints. Spearman's correlations modeled associations between sleep, BMI, and glycemic control at each time point. Repeated ANOVAs modeled sleep and glycemic control changes from baseline to 3 months and to 6 months.

Results: Sleep and glycemic control indices showed significant associations at baseline and 3 months. More time-in-bed and later sleep offset related to higher HbA1c levels at baseline. Later sleep onset, midpoint and offset, and greater sleep efficiency associated with greater %time with hyperglycemia (glucose >180 mg/dL) or hypoglycemia (glucose <70 mg/dL) at baseline and 3 months. Longer sleep duration and greater sleep efficiency related to greater %time with hyperglycemia at 3 months. At 3 months, more wake after sleep onset associated with lower HbA1c levels and longer nocturnal awakenings and more sleep fragmentation associated with less glycemic variability. While both sleep and glycemic control improved from baseline to 3 and 6 months, these were not statistically significant.

Conclusion: Various dimensions of actigraphic sleep related to concurrently estimated glycemic indices indicative of poorer glycemic control and HbA1c across time in adults with long-standing T1D and hypoglycemia unawareness.

Support: This work was supported by NIH R01DK117488 (NG), R01DK091331 (MRR), and K99NR017416 (SKM).

1021

EFFECT OF DIETARY NITRATE SUPPLEMENTATION ON SLEEP IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS

Wisor, J. P.¹ Holmedahl, N.² West Saxvig, I.³ Fjeldstad, O.⁴ Weitzberg, E.⁵ Gronli, J.³ Engen, H.⁶

¹Washington State University, Spokane, WA, ²LHL Hospital Gardermoen, Gardermoen, NORWAY, ³University of Bergen, Bergen, NORWAY, ⁴LHL-hospital Gardermoen, Gardermoen, NORWAY, ⁵Karolinska Institute, Stockholm, SWEDEN, ⁶Unicare Rehabilitation and Health, Oslo, NORWAY.

Introduction: Chronic obstructive pulmonary disease (COPD) requires the use of accessory muscles to overcome inadequate ventilation. The activity of these voluntary muscles is compromised during sleep, resulting in insufficient ventilation, oxygen desaturation and disruption of sleep. Nitrate supplementation with dietary beetroot juice (DBJ) is known to increase the efficiency of oxygen utilization in non-COPD individuals; its therapeutic effect in COPD is uncertain.

Methods: In a repeated measured experiment involving 15 COPD patients, subjects consumed either 70 mL of beetroot juice containing nitrate (~ 6.2 mmol NO_3^-) or placebo (NO₃⁻-depleted juice)

immediately before bedtime. Sleep states were defined based on F4-O2 electroencephalogram and masseter electromyogram. All subjects spent at least 6.2 hrs in bed; the data analysis was therefore restricted to the first 6 hrs in bed.

Results: Standard polysomnography indicated no changes in the amount of time spent in any sleep stages. Wake-to-N2 transitions were greater than two-fold more frequent after placebo (a total of 21 observed) than DBJ (9 observed), resulting in a significant main effect of treatment ($F_{1,14}$ =7.3, P=0.017). N2-to-wake transitions were nearly 3-fold more frequent after placebo (a total of 35 observed) than DBJ (12 observed), resulting in a significant main effect of treatment ($F_{1,14}$ =2.52, P=0.024). Direct wake-to-REMS transitions were observed four times after placebo and never after DBJ ($F_{1,14}$ =2.26, P=0.041). DBJ also resulted in sustained elevation of peripheral oxygen saturation (SpO₂), measured by pulse oximetry, during episodes of wake after sleep onset (WASO). Two minutes into WASO after DBJ SpO₂ was elevated by 1.09 + 0.31% relative to pre-WASO; two minutes into WASO after placebo SpO₂ was elevated by 0.08 + 0.54% relative to pre-WASO (P=0.012).

Conclusion: Collectively, the reduced frequency of atypical transitions after DBJ are indicative of an improvement of sleep quality. DBJ is thus a potential adjunct therapy for disordered sleep in COPD.

Support: N/A

1022

ASSOCIATION BETWEEN HOMOCYSTEINE AND SLEEP IN POSTMENOPAUSAL WOMEN

BANZOLI, C. V.¹ Bezerra, A. G.² D'Almeida, V.² Andersen, M. L.² Tufik, S. V.² Hachul, H. V.²

¹Universidade Federal de São Paulo, SAO PAULO, BRAZIL, ²Universidade Federal de São Paulo, São Paulo, BRAZIL.

Introduction: Homocysteine (Hcy) is a sulfur amino acid, considered an independent risk factor for cardiovascular disease. Excessive Hcy directly harms the endothelium and can lead to premature atherosclerosis, with progression to stroke and acute myocardial infarction. One of the causes of hyperhomocysteinemia (Hhcy) is known to be hypoestrogenism. Hypoestrogenism increases the cardiovascular risk as well as the occurrence of sleep disorders. Hhcy prevalence varies by population and its value in postmenopausal women in Brazil is unknown. Objective: To evaluate the prevalence of hyperhomocysteinemia in postmenopausal women in the city of São Paulo. Check if there is an association between the variables: Hcy and lipid profile; Hcy and hot flushes; Hcy and subjective sleep parameters

Methods: A population-based cross-sectional study was conducted that included a total of 1,042 volunteers living in the city of São Paulo in 2007. This research is part of the São Paulo Sleep Epidemiological Study (EPISONO). Study approved by the Ethics Committee (CEP # 0593/06) and registered with ClinicalTrials. gov (NCT00596713). Hcy, total cholesterol, HDL, LDL, triglycerides were measured. Sleep questionnaires PSQI, IGI, Epworth Sleepiness Scale were used. The GLzM (Generalized Linear Model) was used to verify the association between the different variables. Dependent variables were used in binominal and gama distribution when needed

Results: The sample consisted of 193 postmenopausal women, with a mean age of 58 years (SD \pm 9). The prevalence of Hhcy in this sample was 4.7%, while 14.7% had dyslipidemia 22.8% with hypertriglyceridemia and 29% with low HDL levels. There was an association between Hcy and HDL. There was no association

between Hcy and hot flushes, Hcy and LDL, nor with Hcy and sleep parameters.

Conclusion: The studied population presented low prevalence of Hhcy and there was no association between Hcy and sleep parameters.

Support: This research was support by fellowships from Associação Fundo de Incentivo à Pesquisa (AFIP) and Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES) - Finance Code 001.

1023

ONCOLOGISTS' BELIEFS ABOUT MEDICAL MARIJUANA'S EFFECTIVENESS FOR SLEEP IN CANCER PATIENTS: A NATIONALLY REPRESENTATIVE SURVEY

Zhou, E. S.^{1,2} Nayak, M. M.² Braun, I. M.²

¹Harvard Medical School, Boston, MA, ²Dana-Farber Cancer Institute, Boston, MA.

Introduction: Cancer is a qualifying condition for medical marijuana (MM) access in almost every state which has legalized medical use. Over 20% of patients report recent cannabis use to manage cancer treatment side effects, including poor sleep. However, there have been no clinical trials of MM for sleep in oncology populations. This study explores oncologists' characteristics associated with their views on MM for sleep.

Methods: N=237 medical oncologists completed a mailed survey querying demographics, training history and current practice, and their knowledge and views on MM. In these analyses, we compared oncologists who viewed MM as being equally or more effective than standard treatment approaches to manage poor sleep with those who viewed MM as less effective.

Results: Sixty-four percent of oncologists believed MM to be equally or more effective than standard treatments for poor sleep. They were significantly less likely than peers to have a medical school faculty appointment (53% vs 72%) or self-report sufficient knowledge about MM to make clinical recommendations (42% vs 57%). Oncologists who viewed MM favorably compared to standard treatments for poor sleep were more likely to have recommended its use and to view it as beneficial during end of life care. We identified no differences between groups by age, sex, ethnicity, US vs foreign medical training, patient volume, or whether the state where they practiced had legalized MM.

Conclusion: Oncologists affiliated with a medical school report less knowledge about MM and are more cautious in their views of its effectiveness for treating poor sleep. This may be due to their institutions' reluctance to draft MM policies for fear of losing federal licensure or their awareness of the limitations of existing data. There are important practice implications related to the likelihood of an oncologist recommending MM for poor sleep. **Support:** Hans and Mavis Lopater Foundation.

Support: Hans and Mavis Lopater Foundat

1024

ASSOCIATIONS OF LOW BACK PAIN AND SLEEP AMONG NURSING STAFF

Zhang, Y.¹ Thind, H.¹ Kim, S.¹ Nunes, R.¹ Reidy, J.¹ Punnett, L.¹ Duffy, J.²

¹University of Massachusetts Lowell, Lowell, MA, ²Brigham and Women's Hospital, Boston, MA.

Introduction: Nursing is one of the top occupations suffering musculoskeletal disorders, especially low back pain (LBP). Nursing staff also experience short and disturbed sleep. Although there is a

known relationship between pain and sleep, the specific associations between different aspects of LBP (e.g., duration, frequency, intensity) and sleep have not been studied. The objective of this study is to examine different aspects of LBP and their cross-sectional associations with sleep among nursing staff.

Methods: Online Qualtrics surveys were distributed among nurses and nursing assistants at a community hospital in the northeast U.S. LBP was assessed in terms of duration, frequency, intensity, and intensity change from before to after the work shift. Sleep duration and disturbances were assessed with validated scales.

Results: Among the 541 participants (94% female; age 43±13y), more than a third reported short sleep duration (\leq 6hrs/day; 38%) or sleep disturbances (38%), and more than half (60%) reported LBP in the past 6 months. Among those with LBP, 82% had ongoing pain for at least 6 months; 44% had ongoing pain for at least half the days in the past 6 months; 39% had LBP intensity \geq 4 out of 10; and 79% reported post-shift LBP intensity increased of at least 1 level. Short sleep duration was associated with ongoing LBP for at least half the days in the past 6 months, intensity \geq 4, and post-shift LBP intensity increase. Sleep disturbances were associated with prevalent LBP and intensity \geq 4.

Conclusion: Nursing staff reported a high prevalence of LBP as well as short and disturbed sleep. Overall, poor sleep was associated with higher LBP prevalence, frequency, intensity, and postshift increase. Future longitudinal studies are needed to clarify the causal directions of these relationships. Workplace interventions should address the widespread problems of LBP and sleep deficiency of nursing staff.

Support: Drs. Yuan Zhang and Jeanne F. Duffy were supported by NIH grant R01 AG044416.

1025

SLEEP PATTERNS IN HEAD NECK CANCER PATIENTS DURING RADIOTHERAPY

Gu, F.¹ Jungquist, C.² Sonia, A.³ Liu, L.³ Repasky, E.¹ Schlecht, N.¹ Reid, M.¹ Ambrosone, C.¹ Andrew, R.¹ Singh, A.¹ ¹Roswell Park Comprehensive Cancer Center, Buffalo, NY, ²SUNY-Buffalo, School of Nursing, Buffalo, NY, ³Moores Cancer Center, La Jolla, CA.

Introduction: Sleep disturbances are reported to be highly prevalent in head and neck cancer (HNC) patients, but no carefully assessed sleep data exists in patients with HNC undergoing concurrent chemoradiotherapy (CRT).

Methods: To objectively assess sleep patterns in this study population, we conducted a pilot study in 15 patients and 13 non-cancer healthy volunteers. Patients wore the wrist Actiwatch Spectrum (Philips Respironics) at week 1, 3, and 6/7 during the 7-week treatment period. Volunteers wore the Actiwatch for one week. We used the Actiware software to calculate sleep parameters. A sleep log was used as a complement to define participants' bedtime and rise-up time. Any sleep episode scored by the software during daytime was considered as a nap.

Results: Compared to healthy volunteers, patients had lower overnight sleep efficiency, longer sleep onset latency and more waking time after sleep onset (WASO), indicating more difficulty falling asleep and maintaining sleep. During CRT, patients' sleep efficiency decreased whereas latency and WASO increased, indicating possible the decrease of sleep quality. Sleep efficiency of <85% has been used previously as a cut-off for poor sleep; based on this criteria, 45% of HNC patients had poor sleep at treatment baseline, compared to 31% in non-cancer volunteers, and this proportion

increased to 51% by the end of treatment. Patients had longer napping time: compared to healthy volunteers, the napping time was on average 2 hours longer at baseline, and 3 hours longer at the end of treatment, indicating unhealthy sleep habits of these patients.

Conclusion: Our data suggested HNC patients had severe sleep disturbances and unhealthy sleep habits, which were aggravated during CRT treatment.

Support: This study was supported by UL1TR001412-04, a Clinical and Translational Research Award under SUNY-Buffalo.

1026

ASSOCIATIONS BETWEEN PHYSIOLOGICAL AROUSAL AND EXECUTIVE FUNCTION IN ADULTS WITH CHRONIC WIDESPREAD PAIN AND INSOMNIA COMPLAINTS

Curtis, A. F. McGovney, K. McCrae, C. S. University of Missouri, Columbia, MO.

Introduction: Individuals with chronic widespread pain (CWP) commonly experience increased physiological arousal, insomnia symptoms, and cognitive disturbance. Higher arousal (as measured by heart rate variability, HRV) is associated with worse executive function in healthy adults, suggesting that hyperarousal selectively disrupts prefrontal cortical activity. However, the HRV/cognition relationship in CWP, the moderating impact of insomnia severity, as well as the components of executive function affected, are unclear. The present study assessed independent associations between HRV and components of executive functioning in patients with CWP and insomnia complaints, and evaluated whether these associations depend on insomnia severity.

Methods: Forty-two adults (M_{age} =46.2, SD=13.7) with comorbid CWP and insomnia complaints (difficulty falling/staying asleep plus daytime dysfunction) underwent 5-minutes of Holter monitoring to assess resting HRV. The root mean standard deviation of successive normal to normal heartbeats (RMSDNN) was computed as the HRV index. Participants completed the Insomnia Severity Index (ISI). Participants also completed three computerized tasks measuring executive function: Stroop task (inhibition), Sternberg task (working memory), and the Balloon Analogue Risk Task (risk taking behavior). Multiple regressions examined whether RMSDNN independently predicted or interacted with ISI to predict cognition, controlling for age.

Results: RMSDNN independently predicted Stroop performance, with higher RMSDNN (lower arousal) associated with lower interference scores (better inhibitory function), B=-.003, SE=.001, p=.029, Full Model R²=.31. RMSDNN and ISI did not independently predict or interact to predict Sternberg/ BART performance.

Conclusion: In patients with CWP and insomnia symptoms, reduced physiological arousal was associated with better inhibition, and this did not depend on insomnia severity. Findings highlight the potential underlying role of hyperarousal in a specific component of executive disruption in CWP. Results suggest that treatments aimed at reducing physiological arousal in comorbid CWP and insomnia (e.g., relaxation, HRV biofeedback) may selectively improve inhibitory function. Findings warrant further consideration in larger samples and prospective analyses.

Support: Research was supported by the National Institute of Nursing Research (NR017168; PI: McCrae). Data collected as part of clinical trial NCT02001077 Sleep and Pain Interventions (SPIN2) at the University of Missouri (PI: McCrae).

1027

DIFFERENCES IN POLYSOMNOGRAPHY-BASED SLEEP DISORDERS BETWEEN HIV-INFECTED PERSONS AND MATCHED CONTROLS

Chen, Y.¹ Chen, C.² Strollo, P. J.³ Li, C.² Ko, W.² Lin, C.² Ko, N.² ¹National Cheng Kung University Hospital, Tainan, TAIWAN, ²National Cheng Kung University, Tainan, TAIWAN, ³University of Pittsburgh, Pittsburgh, PA.

Introduction: Sleep disturbance is a prevalent problem among HIV-infected persons. The recognition of comorbid sleep disorders in patients with HIV is currently hampered by limited knowledge of sleep-related symptoms, sleep architecture, and types of sleep disorders. We aimed to compare the differences in sleep-related symptoms and polysomnography-based sleep disorders between HIV-infected persons and controls.

Methods: The study included 170 men with a Pittsburgh sleep quality index (PSQI) greater than 5, composed of 44 HIV-infected men and 126 male controls who were frequency-matched by sex, age (-/+ 3.0 years) and BMI (-/+ 3.0 kg/m2). For all participants an overnight sleep study using a Somte V1 monitor was conducted. Differences in sleep-related symptoms and sleep disorders between HIV-infected patients and controls were examined using t-tests or Chi-square tests.

Results: HIV-infected persons with sleep disturbances more often had psychological disturbances (72.7% vs. 40.5%, p<0.001) and suspected rapid eye movement (REM) behavior disorder (RBD) (25.0% vs. 4.8%, p<0.01) than that of controls. The sleep-disordered breathing (SDB) in HIV-infected persons was less common than that in controls (56.8% vs. 87.3%, p<0.001). The mean percentage of REM sleep among HIV-infected patients was higher than that among the controls (20.6% vs. 16.6%, p<0.001). Enuresis was more common in HIV-infected persons than controls (40.9% vs. 22.2%, p=0.02).

Conclusion: Psychological disturbances and SDB can be the possible explanations of sleep disturbances in HIV-infected persons, in which suspected RBD is notable. Further studies are warranted to examine underlying factors of suspected RBD among HIV-infected persons with sleep disturbances.

Support: This work was supported by the Ministry of Science and Technology, Executive Yuan of Taiwan [MOST 105-3011-E-006-002], and National Cheng Kung University Hospital [NCKUH-10702022]

1028

SLEEP AND ENTERIC DISEASE: SLEEP NOW FOR LESS DIARRHEA LATER

Mantua, J.¹ Gutierrez, R. L.² Isidean, S. D.² Alaca, A. N.² Testa, K. J.² Talaat, K.³ Doty, T. J.¹ Capaldi, V. F.¹ Porter, C.² ¹Walter Reed Army Institute of Research, Silver Spring, MD, ²Naval Medical Research Center, Silver Spring, MD, ³Johns Hopkins Medicine, Baltimore, MD, ⁴Walter Reed Army Institute of Research, Silver Spring, MD.

Introduction: The bi-directional relationship between sleep and immune function is well-established. Sufficient sleep supports immune health and can increase vaccine efficacy. Conversely, sickness can disturb sleep quality, which can delay recovery and waking functioning. However, the bidirectional relationship between sleep and infectious diarrhea, the leading infectious disease threat to deployed military populations, has not been studied. We assessed the bi-directional relationship between sleep and enteric disease

utilizing data from a recently-completed controlled human infection model (CHIM) with enterotoxigenic Escherichia coli (ETEC). Methods: During a CHIM to assess the efficacy of an immunoprophylactic targeting ETEC (NCT03040687), we measured sleep via actigraphy over an 8-day inpatient period. Participants ingested prophylaxis 3 times/day during days -2 and -1 and ingested ETEC on day 0. The primary outcome was moderatesevere diarrhea following the ETEC challenge. We hypothesized better sleep pre-challenge would reduce risk of disease after the challenge (assessed using linear regression). We also hypothesized total sleep time (TST) and sleep efficiency (SE) after the challenge would be lower/poorer than baseline (assessed using paired t-test). Results: Among 59 participants (aged 34.4±8.1yrs, 64% female), longer TST the night preceding ETEC challenge was associated with lower total diarrhea volume (B=-3.13,p=.001). SE was slightly but significantly poorer after the challenge (78 vs. 76%; t(55)=2.2,p=.03), but there was no significant change in TST, potentially due to low TST pre-challenge (316 vs. 329 minutes; p=0.12).

Conclusion: These results - in aggregation with previous work on sleep and vaccines - suggest military sleep regulations should be put in place to increase sleep prior to traveling to an area of responsibility with high risk for enteric disease. These minor behavioral changes could provide lasting benefits to readiness of military servicemembers.

Support: This work was supported by Joint Warfighter Medical Research Program (JWMRP) and the Military Operational Medicine Research Program (MOMRP). The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the US Army or of the US Department of Defense. This abstract has been approved for public release with unlimited distribution.

1029

OBJECTIVELY-MEASURED SLEEP FOLLOWING A TIME RESTRICTED EATING INTERVENTION IN ADULTS WITH OBESITY

Simon, S. L.¹ Fleischer, J. G.² Manoogian, E. N.³ Panda, S.³ Mashek, D. G.⁴ Chow, L.⁴

¹University of Colorado Anschutz Medical Campus, Aurora, CO, ²Salt Institute for Biological Studies, La Jolla, CA, ³Salk Institute for Biological Studies, La Jolla, CA, ⁴University of Minnesota, Minneapolis, MN.

Introduction: Time-restricted eating (TRE), limiting food intake to a consistent daily window, is emerging as a novel weight loss intervention but impact of TRE on sleep remains unclear. Prior studies reported mixed results but are limited by subjective sleep measurements and lack of a randomized control group. Thus, we examined changes in actigraphy-measured sleep following a 12-week TRE protocol.

Methods: Participants were 20 adults ages 18-65 years with BMI $\geq 24 \text{kg/m}^2$. Participants were randomized to either TRE (8-hour eating window) or non-TRE (typical eating). At baseline and follow-up, all participants had anthropometric measurements, oral glucose tolerance test, logged eating occasions in a smartphone application, and wore an ActiGraph Link for two weeks. Independent samples t-tests compared groups on actigraphy-estimated sleep variables. Pearson correlations examined associations between sleep variables with health outcomes.

Results: The TRE (N=11) and non-TRE groups (N=9) were predominantly female and had a baseline eating window of

approximately 15 hours. There were no differences in actigraphyassessed sleep variables at baseline or follow-up between groups. Participants did not significantly change their sleep from baseline to follow-up. Median weekday sleep duration was 6.2 hours at follow-up for all participants, suggesting insufficient sleep compared to the recommended 7-9 hours of sleep. Participants who obtained greater than the median weekday sleep duration at follow-up had significantly lower BMI, better insulin sensitivity (HOMA and Matsuda Index), and greater percent improvement in insulin sensitivity.

Conclusion: Our data show that TRE does not significantly alter sleep behaviors in participants with obesity. However, longer sleep duration at follow-up was associated with lower BMI, better insulin sensitivity, and greater improvement in insulin sensitivity, indicating that sleep may be an important variable to consider in dietary interventions. Future research examining behavioral sleep strategies in combination with TRE is needed to evaluate whether improved sleep leads to better weight loss and glycemic outcomes for individuals with obesity.

Support: This work was support by the Healthy Foods Healthy Lives program (17SFR-2YR50LC to LC) and the National Institutes of Health (NIH National Center for Advancing Translational Sciences, UL1TR002494).

1030

AN EXPLORATION OF THE IMPACT OF COGNITIVE BEHAVIOURAL THERAPY OF INSOMNIA (CBT-I) ON PERCEIVED COGNITIVE IMPAIRMENT IN BREAST CANCER SURVIVORS

Walsh, N^{1,2} *Garland*, S.^{1,2,3} *Lester*, R.³ *McCarthy*, J.³ *Laing*, K.³ ¹Department of Psychology, Memorial University, St. John's, NL, CANADA, ²Beatrice Hunter Cancer Research Institute, Halifax, NS, CANADA, ³Discipline of Oncology, Memorial University, St. John's, NL, CANADA.

Introduction: Insomnia and cognitive impairment are prevalent and persistent symptoms in cancer survivors. Cognitive Behavior Therapy is effective for improving insomnia and comorbid symptoms in cancer survivors but there are very few empirically supported treatments that can improve cognitive impairment. This feasibility study explored the impact of CBT-I on perceived cognitive impairment in breast cancer survivors.

Methods: We enrolled 10 early stage breast cancer survivors with insomnia disorder and perceived cognitive impairment. Participants received 7 individual sessions of CBT-I over the course of 8 weeks and completed the Insomnia Severity Index (ISI), the Functional Assessment of Cancer Therapy - Cognitive Function (FACT-Cog) questionnaires and The Hospital Anxiety and Depression Scale (HADS) at baseline and post-treatment. Paired samples t-tests were used to assess change over time.

Results: The sample was predominantly diagnosed with stage II breast cancer (60%). Women were an average age of 50.8 (SD 6.84) and 18.2 (SD 3.62) years of education. CBT-I significantly reduced insomnia severity [19.4 to 7.1; t(9)= 6.56, p < .001] and improved perceived cognitive impairment [t(9)= -3.55, p < .01], perceived cognitive ability [t(9)= -2.87, p < .05], quality of life [t(9)= -3.14, p < .05], and overall subjective cognitive function [t(9)= -3.67, p < .01]. Although participants began treatment with low levels of mood disturbance, CBT-I further decreased symptoms of anxiety (baseline: M= 10.10, SD= 4.34; post-treatment M= 8.20, SD= 3.91) and depression (baseline: M= 7.90, SD= 3.45; post-treatment M= 5.30, SD= 2.83), although not statistically significant.

Conclusion: This study suggests CBT-I may improve perceived cognitive impairment in cancer survivors, in addition to insomnia and mood. Future randomized controlled trials with larger samples and objective measurements of cognition are needed.

Support: Nyissa Walsh is a trainee in the Cancer Research Training Program of the Beatrice Hunter Cancer Research Institute (BHCRI). Dr. Sheila Garland is supported by a Scotiabank New Investigator Award from BHCRI.

1031

EXPLORING THE IMPACT OF COGNITIVE BEHAVIORAL THERAPY FOR INSOMNIA (CBT-I) ON DAYTIME PRODUCTIVITY IN SURVIVORS OF BREAST CANCER

Kieley, J.¹ Walsh, N.^{1,2} McCarthy, J.³ Powell, E.³ Garland, S. N.^{1,3,2} ¹Department of Psychology, Memorial University, St. John's, NL, CANADA, ²Beatrice Hunter Cancer Research Institute, Halifax, NS, CANADA, ³Discipline of Oncology, Memorial University, St. John's, NL, CANADA.

Introduction: Post-treatment insomnia disorder and fatigue symptoms can impair work and daytime productivity in breast cancer survivors. Cognitive Behavioral Therapy for Insomnia (CBT-I) significantly improves insomnia and daytime fatigue. This feasibility study examined whether improving insomnia and fatigue using CBT-I is associated with improved work and activity productivity in breast cancer survivors.

Methods: 10 survivors of early stage breast cancer participated in 7 weekly individual CBT-I sessions. The primary outcome was the Work Productivity and Activity Impairment Questionnaire-General Health (WPAIQ-GH) questionnaire. Secondary outcomes were the Insomnia Severity Index (ISI) and the Multidimensional Fatigue Symptom Inventory-Short Form (MFSI-SF). Assessments were conducted at baseline and post-treatment. Paired samples t-tests examined the impact of CBT-I on productivity and fatigue. Linear regression assessed whether change in fatigue was associated with change in productivity.

Results: Participants had a mean age of 50.8 (range 42-63) and the majority were diagnosed with stage II (60%) cancer. There was a significant reduction in fatigue [t(9)= 2.43, p=.04] and activity impairment due to insomnia [t(9)= 3.105, p<.05] following treatment. Insomnia affected 52% of work productivity at baseline with a non-significant decrease to 15% following treatment [t(3)= 2.25 p=.110]. Reductions in fatigue were significantly associated with reductions in activity impairment [F(1,8)= 7.25, p=.03], accounting for 47.5% of the variability.

Conclusion: Treating insomnia with CBT-I significantly improved daytime productivity, activity impairment, and fatigue. Controlled research with larger sample sizes is warranted to confirm these preliminary results.

Support: Nyissa Walsh is a trainee in the Cancer Research Training Program of the Beatrice Hunter Cancer Research Institute (BHCRI). Dr. Sheila Garland is supported by a Scotiabank New Investigator Award from BHCRI.

1032

AURICULAR POINT ACUPRESSURE FOR SLEEP DISTURBANCE IN WOMEN WITH BREAST CANCER: A PILOT RANDOMIZED CONTROLLED TRIAL

Wang, Y.¹ Wu, J.¹ Li, J.² Zhou, J.¹

¹Shanghai University of Traditional Chinese Medicine, shanghai, CHINA, ²Johns Hopkins University, Baltimore, MD. **Introduction:** Sleep disturbance is reported in up to 60% of cancer patient. In traditional Chinese medicine, evidence suggests that auricular point acupressure (APA) improves sleep. However, little is known about APA's effect on sleep disturbance in patients with breast cancer (BC). We tested the preliminary efficacy of APA on sleep in BC women undergoing chemotherapy.

Methods: A pilot randomized controlled trial was conducted in 41 BC patients (mean age= 50 ± 14) with self-reported poor sleep [Pittsburgh Sleep Quality Index (PSQI) \geq 7]. Participants were randomly assigned to an APA group (n=22) and a control group (n=19). All patients received sleep hygiene education. Additionally, for the APA group, magnetic pellets were attached to selected auricular points once a week for 3 weeks at the clinic, and the participants were instructed to self-press the pellets 4 times a day. Sleep were objectively measured by Actiwatch Spectrum and subjectively using PSQI at baseline and post-intervention. Paired t-tests and analyses of covariance using the variable baseline values were used to examine changes in sleep parameters.

Results: Twenty-one participants from the APA and sixteen from the control groups completed the study. Within the APA group, PSQI [mean difference (MD)=3.85, 95% Confidence Interval (C)= $3.12\sim4.60$] and sleep onset latency (MD=18.02, 95%CI= $5.96\sim30.09$) were significantly decreased, and the sleep duration (MD=-0.53, 95%CI=-0.99 \sim -2.35) and sleep efficacy (MD=-5.00, 95%CI= $-8.72\sim-1.28$) were significant increased at post-intervention. Compared to the control group, participants in the APA group had significantly lower PSQI (F=30.77, p<0.001) and greater sleep efficacy (F=5.25, p=0.028) at post-intervention. Conclusion: APA may be an inexpensive and effective approach to

improve sleep in patients with BC. More rigorous research with larger samples is needed to further test the efficacy of APA on promoting sleep in BC patients.

Support: None

1033

SLEEP AND PATIENT-REPORTED OUTCOMES IN PERSONS WITH HEART FAILURE WITH PRESERVED EJECTION FRACTION

Baniak, L. M. Atwood, C. W. Strollo, P. J. Forman, D. E. Chasens, E. R.

University of Pittsburgh, University of Pittsburgh, PA.

Introduction: Sleep quality has a significant bearing on disease. A better understanding of sleep quality may help identify opportunities to improve patient-reported outcomes (PROs) in persons with heart failure with preserved ejection fraction (HFpEF). We aimed to explore the association between sleep and PROs in patients clinically diagnosed with HFpEF.

Methods: Cross-sectional study of 22 participants (71.2±7.2 years, 95% male, 86.4% white) with HFpEF, recruited from a heart failure (n=14) and sleep clinic (n=8). Sleep disordered breathing was measured objectively using one-night in-home obstructive sleep apnea (OSA) testing (ApneaLink). Actigraphy (7 days) was used to assess sleep duration, efficiency, and wake after sleep onset (WASO). Subjective sleep measures included the Insomnia Severity Index (ISI), Epworth Sleepiness scale (ESS), and Pittsburgh Sleep Quality Index (PSQI). PROs included functional status (Functional Outcomes Sleep Questionnaire [FOSQ]), depression (PROMIS Depression), fatigue (PROMIS Fatigue), and heart failure specific quality of life (Kansas City Cardiomyopathy Questionnaire [KCCQ]; overall summary score [KCCQ-os] and clinical summary

score [KCCQ-cs]). The KCCQ-cs includes physical function and symptom scores to corresponds with NYHA Classification.

Results: Fifty percent of the participants had poor sleep quality (PSQI >5) and 2 (9.1%) had ISI scores >14. The majority (64%; n=14) had OSA; 10 currently on OSA therapy. Mean oxygen desaturation index (ODI) was 20.8 ± 17.8 . Mean actigraphy data indicated poor sleep (sleep duration 302 ± 116 minutes, sleep efficiency $70.0\pm18.6\%$, and WASO 52 ± 28 minutes) despite only 5 (22.7%) participants reporting excessive daytime sleepiness (ESS>10). Greater insomnia symptom severity was associated with lower heart failure specific quality of life (KCCQ-os) and functional status, and, greater fatigue and depression (all p-values <.05). FOSQ was negatively associated with PSQI (r= -.710, p= <.001) and positively with sleep efficiency (r=.496, p=.026). The KCCQ-cs was positively associated with sleep duration (r=.496, p=.026) and negatively but not significantly associated with ODI (r= -.453, p=.07).

Conclusion: Impaired sleep and OSA are highly prevalent in patients with HFpEF and both are adversely associated with PROs. Goals to improve sleep is important for effective symptom management and for potential improvements in PROs.

Support: American Nurses Foundation, Preventative Cardiovascular Nurses Association

1034

SHOULD WE RECOMMEND MORE SLEEP TO PREVENT OBESITY?

Kim, D. S.¹ Foster, B. E.² Collen, J. F.² Eliasson, A. H.²

¹Department of Internal Medicine, Walter Reed National Military Medical Center, Bethesda, MD, ²Sleep Disorders Center, Walter Reed National Military Medical Center, Bethesda, MD.

Introduction: According to the 2015-2016 National Health and Nutrition Examination Survey (NHANES), the national adult obesity rate was 40% with the incidence of adult obesity having increased by 70% over the last 30 years. Paralleling the obesity epidemic have been worsening sleep deprivation and eroding sleep quality. We analyzed data from a Cardiovascular Health Registry to explore a link between total sleep time and obesity.

Methods: Registry participants underwent anthropometrics and completed validated questionnaires assessing health behaviors and symptoms including total sleep time (TST), Epworth Sleepiness Scale (ESS), Fatigue Severity Scale (FSS), Pittsburgh Sleep Quality Index (PSQI), Perceived Stress Scale (PSS), and exercise time. Differences between subjects with sufficient (\geq 7 hours) and insufficient (\leq 7 hours) sleep were analyzed using T-tests.

Results: Registry participants (n=630) had mean age 55.3 ± 9.9 years (45% men, 391W, 182B, 26H, 12A, 19O). The subgroup with sufficient sleep (n=261, 48% men), had mean BMI 29.3 \pm 5.6 while the subgroup with insufficient sleep (n=369, 44% men) had mean BMI of 30.5 ± 5.3 , p=0.008. The insufficient sleep group was noted to be sleepier (ESS 9.7 \pm 4.9 vs 7.4 \pm 4.6, p<0.001), more fatigued (FSS 4.9 \pm 2.3 vs 3.5 \pm 2.4, p<0.001) and have worse sleep quality (PSQI 8.6 \pm 3.7 vs 4.7 \pm 2.8, p<0.001). Insufficient sleepers also perceived greater stress levels (PSS 22.2 \pm 8.4 vs 18.9 \pm 6.2, p<0.001), and showed a trend toward less exercise per week (143 \pm 134 vs 163 \pm 106 minutes, p=0.13).

Conclusion: Participants with insufficient sleep were significantly more overweight on average and were more symptomatic for insufficient sleep. While current approaches to weight management focus largely on diet and physical activity, the data from this study suggest that insufficient sleep should also be considered as a risk

factor for obesity and should be incorporated into management plans for obesity. **Support:**

1035

SLEEP DURATION AND METABOLIC SYNDROME: AN UPDATED SYSTEMATIC REVIEW AND META-ANALYSIS

Li, Y.¹ Xie, J.¹ Chen, B.¹ Basta, M.² Vgontzas, A.² ¹Sleep Medicine Center, Mental Health Center, Shantou University Medical College, China, Shantou, CHINA, ²Sleep Research & Treatment Center, Department of Psychiatry, Pennsylvania State University, College of Medicine, Penn State Health Milton S. Hershey Medical Center, Hershey, PA.

Introduction: To systematically examine the association between sleep duration and metabolic syndrome (Mets) risk in cross-sectional and prospective cohort studies.

Methods: Data were collected from 36 cross-sectional and 9 longitudinal studies with a total of 164799 MetS subjects and 430895 controls. Odds ratios (ORs) for MetS in cross-sectional studies and risk ratios (RRs) for incident MetS were calculated through metaanalyses of adjusted data from individual studies. Subgroup analyses were performed to investigate the association between MetS and the duration of short-and-long sleep.

Results: Short sleep duration was significantly associated with increased prevalent MetS (OR= 1.11, 95% CI =1.05-1.18) and incident MetS (RR= 1.28, 95% CI =1.07-1.53,) in cross-sectional and longitudinal studies, respectively. Furthermore, long sleep duration was significantly associated with increased prevalent MetS in cross-sectional studies (OR= 1.14, 95% CI =1.05-1.23), rather than incident MetS (RR= 1.16, 95% CI =0.95-1.41) in longitudinal studies. Interestingly, the association between long sleep and prevalent MetS was found in sleep duration defined by 24-hour sleep (including naps) rather than nighttime sleep. In cross-sectional studies, pooled odds for MetS were 1.36 (95% CI=1.04-1.78, $I^2=83.3\%$) in \leq 5 hours, 1.09 (95% CI=1.02-1.16, $I^2=67.8\%$) in \leq 6 hours, 1.01 (95% CI=0.93-1.10, I²=24.9%) in <7 hours, 1.11 (95% CI=1.02-1.21, I²=67.0%) in ≥9 hours and 1.31 (95% CI=1.22-1.40, $I^2=0\%$) in ≥ 10 hours, respectively. The association of short sleep and MetS was stronger in young and middle age adults, but lost in adults age >60 years.

Conclusion: Our findings suggest 1) a "U-shape" relationship between sleep duration and MetS in cross-sectional studies and 2) association between short sleep duration, but not long sleep duration with incident MetS. Future studies should shed light on the underlying mechanisms related to the association between sleep duration and MetS and examine if normalizing sleep duration reduces MetS risk in the general population.

Support: This study was supported by National Natural Science Foundation of China (No. 81600068 & 81970087), the Young Elite Scientists Sponsorship Program by CAST (No. YESS20160072), Medical Science Foundation of Guangdong Provence (A2018296) and Grant for Key Disciplinary Project of Clinical Medicine under the Guangdong High-level University Development Program.

1036

DOES DIARY AND ACTIGRAPHY MEASURED SLEEP DIFFER BETWEEN GOOD AND POOR SLEEPERS DURING BREAST CANCER TREATMENT?

Tulk, J.¹ Garland, S. N.^{1,2,3} Rash, J.¹ Lester, R.² Laing, K.²

¹Department of Psychology, Memorial University, St. John's, NL, CANADA, ²Discipline of Oncology, Memorial University, St. John's, NL, CANADA, ³Beatrice Hunter Cancer Research Institute, Halifax, NS, CANADA.

Introduction: Women may enter in breast cancer (BCa) treatment with poor sleep, or it may begin during treatment. We assessed how subjective and objective sleep changes during the first year of treatment for women with BCa. Further, we examined whether this differs between previously good and poor sleepers and whether there was agreement between subjective and objective measures of sleep. Methods: Sleep onset latency (SOL), wake after sleep onset (WASO), total sleep time (TST), and sleep efficiency (SE) were measured among 100 patients with newly diagnosed, nonmetastatic BCa using 7 days of diary and actigraphy collected at 4 time points: pre-treatment, 4, 8, and 12 months. Women with a score ≥ 5 on the Pittsburgh Sleep Quality Index at treatment onset were classified as poor sleepers. A 4 (time: 0-, 4-, 8-, 12-months) by 2 (sleep measure: sleep diary, actigraphy) by 2 (group: good, poor sleepers) mixed model ANOVAs was performed for each sleep parameter.

Results: There was a time by sleep measure by group interaction for TST, [F(3,294)=3.014, p=.03). Good sleepers reported greater TST on diaries- than actigraphy at pre-treatment and 12 months, whereas there were no differences in poor sleepers. There was a group by time effect for good vs. poor sleepers [F(3,294)=2.909, p=.035]. Good sleepers experienced decreased TST and SE from pre-treatment through 4-mo, followed by increases. Poor sleepers showed the opposite pattern. Neither group returned to pretreatment levels. Sleep diaries and actigraphy are concordant over time for TST, but not SOL, WASO, or SE.

Conclusion: Sleep parameters worsen during the first year following onset of BCa and concordance between sleep diaries and actigraphy differ between good or poor sleepers.

Support: Dr. Garland is supported by a Scotiabank New Investigator Award and seed funding from the Beatrice Hunter Cancer Research Institute (BHCRI).

1037

ONE YEAR TRAJECTORY OF INSOMNIA AND COMORBID SYMPTOMS IN WOMEN WITH EARLY STAGE BREAST CANCER

Mahon, K.¹ Garland, S. N.^{1,2,3} Tulk, J.¹ Rash, J.¹ Seal, M.² Laing, K.²

¹Department of Psychology, Memorial University, St. John's, NL, CANADA, ²Discipline of Oncology, Memorial University, St. John's, NL, CANADA, ³Beatrice Hunter Cancer Research Institute, Halifax, NS, CANADA.

Introduction: Insomnia symptoms are a common problem and are often comorbid with hot flashes, fatigue, anxiety, and depression following a breast cancer diagnosis. The present study examined changes in insomnia severity and comorbid symptoms in the year following diagnosis.

Methods: This study is part of a larger prospective observational cohort study of 100 women with early stage breast cancer. Insomnia symptoms were measured using the Insomnia Severity Index, fatigue was measured using the Multidimensional Fatigue Symptom Inventory-Short Form, anxiety and depression were assessed using the Hospital Anxiety and Depression Scale, and hot flashes were assessed using the Hot Flash Related Daily Interference Scale. Assessments were performed shortly after diagnosis, 4, 8, and

12 months. A series of repeated measures within subjects ANOVAs were performed to assess changes in symptoms over time.

Results: Among 100 women with breast cancer, 45% reported at least mild insomnia symptoms. There were significant quadratic effects of time on insomnia severity, F(3, 297)= 12.776, $p \le .001$, depression (F[3, 297]= 4.409, p = .005), and fatigue (F[3, 297]= 7.995, $p \le .001$). These symptoms initially worsen and then improve throughout the year, but they do not rebound to pre-treatment levels. Interference from hot flashes worsens and endures for longer than other symptoms but does begin to show improvement one year post-diagnosis (F[3, 297]= 12.110, $p \le .001$). The were no time effects for anxiety (F[3, 297] = 1.4, p = .243).

Conclusion: In general, women treated for breast cancer can expect insomnia and comorbid symptoms to worsen then improve, but not recover, during the first year after a breast cancer diagnosis. Early efforts to educate women and manage symptoms could prevent insomnia and other issues from becoming persistent problems. **Support:** Dr. Garland is supported by a Scotiabank New Investigator Award and seed funding from the Beatrice Hunter Cancer Research Institute (BHCRI).

1038

SLEEP ARCHITECTURE IN INDIVIDUALS RECEIVING METHADONE FOR MEDICATION-ASSISTED TREATMENT OF OPIOID USE DISORDER

Erwin, J. A.^{1,2} Wilson, M.^{1,3} Finlay, M.^{1,2} Hansen, D. A.^{1,2} Little-Gott, A.³ Reynolds, D.³ Quock, R. M.⁴ Layton, M. E.^{1,2} Van Dongen, H.^{1,2}

¹Sleep and Performance Research Center, Washington State University, Spokane, WA, ²Elson S. Floyd College of Medicine, Washington State University, Spokane, WA, ³College of Nursing, Washington State University, Spokane, WA, ⁴Department of Psychology, Washington State University, Pullman, WA.

Introduction: It has been established that the use of opioids suppresses stage N3 sleep. For individuals with opioid use disorder (OUD), medication-assisted treatment (MAT) is a widely employed opioid replacement therapy used to mitigate withdrawal effects and drug cravings. We investigated sleep architecture in individuals receiving methadone-based MAT.

Methods: N=6 individuals (aged 43.8 ± 12.8 y; 5 females), who were within 90 days of methadone initiation, underwent in-laboratory overnight polysomnography (8h TIB; 22:00-06:00). Prior to bedtime, pain intensity and opioid withdrawal symptoms were assessed using the Numeric Pain Rating Scale (0-10) and the Clinical Opiate Withdrawal Scale (0-48). Sleep recordings were scored visually according to AASM guidelines.

Results: In this sample, subjects exhibited 87.4-93.0% (M: 92.2%) sleep efficiency (SE), 8.0-16.2min (M: 12.1min) sleep latency (SL), 5.5-7.5% (M: 6.5%) N1, 46.4-52.7% (M: 49.6%) N2, 20.7-30.6% (M: 25.6%) N3, 17.5-19.1% (M: 18.3%) REM, 28.0-38.5min (M: 33.3min) N3 latency, and 84.1-125.9min (M: 105.0min) REM latency. Subjects reported moderate pain intensity scores of 5-6 (M: 5.3) and mild to moderate withdrawal symptoms of 1-15 (M: 7.8). **Conclusion:** Relative to published healthy sleeper norms, subjects showed more N1 and N3 and less REM sleep. The increased N3 was unexpected given that opioids (such as methadone) typically suppress N3; it may reflect subjects carrying a substantial sleep debt. Pain and withdrawal symptoms may be a factor increasing N1 and reducing REM sleep. Such potential sleep deficiencies may interfere with subjects achieving OUD recovery goals and are worthy of further investigation.

VIII. Sleep and Medical Disorders

Support: Supported in part by a seed grant from the Washington State University Office of Research Advancement and Partnerships.

1039

SLEEP AND DAYTIME ACTIVITY AMONG MECHANICALLY VENTILATED ADULTS DURING EARLY CRITICAL ILLNESS

Liang, Z.¹ Elias, M. N.¹ Ji, M.² Munro, C. L.¹ ¹University of Miami School of Nursing and Health Studies, Coral Gables, FL, ²University of South Florida College of Nursing, Tampa, FL.

Introduction: The purpose of this study is to report 5 consecutive days' descriptive data for sleep efficiency (SE), total sleep time (TST), and daytime activity ratio (DAR) among critically ill mechanically ventilated adults from 9 intensive care units (ICU) across two hospitals. To our knowledge, this is the first study to describe sleep and activity patterns among mechanically ventilated adults during the early critical illness period.

Methods: We enrolled 31 critically ill mechanically ventilated subjects within 48 hours of ICU admission. Daytime periods were defined as 06:00-21:59; nighttime periods were defined as 22:00-05:59. Actigraphy estimated nighttime SE, TST, and the DAR. We calculated mean DARs [DAR = (daytime activity count per minute / 24-hour activity count per minute) x 100], which may be an indicator of altered rest/activity cycles. In our study, a DAR of >80% was used to define normal rest/activity patterns. Descriptive analyses were used for this sub-analysis of our parent randomized controlled trial.

Results: Among the 31 subjects included, the mean age was 59.6 \pm 17.3 years, 41.9% were male, 80.6% were White, and 67.7% were Hispanic/Latino. The mean nighttime SE and TST over the 5-day ICU period were 83.1% \pm 16.1 and 6.6 \pm 1.3 hours, respectively. The mean DAR over the 5-day ICU period was 66.5% \pm 19.2. Only 17.5% of subject days (14 days out of a total of 80 recorded days) met the definition of normal rest/activity patterns (DAR >80%).

Conclusion: Throughout the early ICU period, among mechanically ventilated patients, both the sleep/wake as well as the rest/ activity cycle were disturbed. Intervention studies targeting the optimization of nighttime sleep consolidation and daytime activity should be investigated.

Support: This project was supported by a federal grant from the National Institutes of Health/National Institute of Nursing Research (R01NR016702). This clinical trial is registered with ClinicalTrials.gov (NCT03128671).

1040

POST-ICU DAYTIME SLEEP AND ACTIVITY IMPACT HOSPITAL DISCHARGE OUTCOMES FOR OLDER ICU SURVIVORS

Elias, M. N. Liang, Z. Munro, C. L.

University of Miami School of Nursing and Health Studies, Coral Gables, FL.

Introduction: The aims of this study were to: describe post-ICU daytime sleep and activity among older ICU survivors within 24-48 hours post-ICU discharge, and to examine differences in post-ICU daytime sleep and activity by prospective acuity of discharge disposition at time of hospital discharge.

Methods: Within 24-48 hours of ICU discharge, we enrolled 30 ICU survivors who were at least 65 years old, functionally independent prior to hospitalization, and mechanically ventilated while

in ICU. Actigraphy was used to estimate daytime total sleep time (TST, hours) and daytime activity (mean activity counts/minute) for one daytime period (06:00 AM to 22:00 PM) within 24-48 hours post-ICU discharge. Independent samples t-tests examined differences in mean daytime TST and daytime activity counts, between subjects who were discharged home versus those who were discharged to a facility (inpatient rehabilitation facility, skilled nursing facility, or long-term acute care hospital).

Results: The mean age was 71.37 ± 5.35 years; 63.3% were male and 76.7% were White non-Hispanic/Latino. The mean daytime TST was 7.61 ± 4.31 hours; the mean daytime activity count was 41.2 ± 28.24 counts/minute. Subjects who were discharged to a facility (8.88 ± 3.69 hours) slept significantly longer than those discharged to home (5.54 ± 4.21 hours; t(26) = 2.083, p = .047). Additionally, subjects who were discharged to a facility ($32.96 \pm$ 24.26 counts/minute) were significantly less active during the daytime than those discharged to home (54.42 ± 29.30 counts/minute; t(25) = -2.157, p = .041).

Conclusion: Older ICU survivors who were ultimately discharged to a facility slept significantly longer and were less active during the daytime hours immediately following transition out of ICU, compared to subjects who were discharged home. Intervention studies should investigate whether increasing daytime activity while promoting nighttime sleep consolidation following transfer out of ICU improves discharge outcomes.

Support: N/A

1041

SLEEP QUALITY MEDIATES THE RELATIONSHIP BETWEEN FEAR OF CANCER RECURRENCE AND PSYCHOLOGICAL DISTRESS IN YOUNG ADULTS WITH CANCER

Daniel, L. C.¹ Garland, S.² Zhou, E.³ Chalifour, K.⁴ Eaton, G.⁵ Dunmyer, L.⁶

¹Rutgers University Camden, Camden, NJ, ²Memorial University, St. John's, NL, CANADA, ³Dana Farber Cancer Institute, Boston, MA, ⁴Young Adult Cancer Candada, St. John's, NL, CANADA, ⁵Young Adult Cancer Canada, St. John's, NL, CANADA, ⁶Rutgers University Camden, Rutgers University Camden, NJ.

Introduction: Fear of cancer recurrence is common in young adults with cancer and also related to poorer psychological outcomes. Sleep may be disrupted by anxious thoughts about cancer, causing long-term psychological distress. Thus, the current study tests sleep as a putative mediator of the association between fear of cancer recurrence and overall psychological distress in young adult cancer survivors.

Methods: In a national cross-sectional survey of Canadians, 436 young adults diagnosed with cancer between the ages of 15-39 (current age range 20-39, m=32.39, *SD*=4.70; 88% female) completed the Pittsburgh Sleep Quality Index, the Fear of Cancer Recurrence Inventory—Short Form, and the Kessler 10 Distress Inventory. Mediation was estimated using PROCESS. Age, sex, and on/off treatment status were entered into models as covariates.

Results: In the current sample, average fear of cancer recurrence was above the clinical cut-point (m=22.92, *SD*=6.84), psychological distress was high (m=25.18, *SD*=7.81), and sleep quality was poor (m=9.11, *SD*=3.95). Females reported significantly higher fear of cancer recurrence than males [F(1, 435)=15.49, p <.001]. Patients on treatment reported significantly higher fear of cancer recurrence [F(1,435)=11.43, p=.001], poorer sleep quality [F(1,435)=6.48,

p=.011], and greater psychological distress [F(1,435)=4.73, p <.001] than patients off treatment. Using a bootstrapping model with covariates, higher fear of cancer recurrence was related to poorer sleep quality and, in turn, higher psychological distress as indicated by the indirect effect's confidence interval not containing 0 (indirect effect=.13; 95%CI=0.081, 0.189).

Conclusion: Sleep quality may play an important role in connecting the common experience of fear of cancer recurrence to psychological distress in young adult cancer survivors. Future longitudinal research is needed to examine this possible mediator of young adult cancer patients' psychological distress outcomes over time.

Support: This research was supported by a grant from the Newfoundland and Labrador Support for People and Patient-Oriented Research and Trials (NL-SUPPORT) Unit. Sheila Garland is supported by a Scotiabank New Investigator Award from the Beatrice Hunter Cancer Research Institute (BHCRI).

1042

SLEEP CHARACTERISTICS IN EHLERS DANLOS PATIENTS WITH HYPERMOBILE TYPE: A POLYSOMNOGRAPHIC STUDY

Metlaine, A.¹ LEGER, D.² SAUVET, F.³ GOMEZ-MERINO, D.³ CHENNAOUI, M.³ HAMONET, C.⁴

¹APHP Hôtel Dieu Centre du Sommeil et de la Vigilance, Paris, FRANCE, ²Université de Paris, Paris Descartes, PARIS, FRANCE, ³Université de Paris, EA 7330 VIFASOM, Paris, FRANCE, ⁴ELLASANTE, Paris, FRANCE.

Introduction: Ehlers-Danlos syndrome (EDS) is an heritable collagen disorder with various multisystemic clinical manifestations affecting primarily skin, ligaments and joints, blood vessels and internal organs. The clinical spectrum is very large from mild skin and joint hypermobility to severe physical disability. Patients with Ehlers-Danlos syndrome often complain of poor sleep quality and fatigue with impaired quality of life. The purpose of this study was to assess any objective sleep disturbances in EDS by polysomnography.

Methods: In this case-control study, we included 47 patients EDS type III (hypermobile type) (29 F et 18 M) which were one to one strictly matched to 47(29 F et 18 M) controls according to sex, age, and BMI. Participants underwent level-1 polysomnography for a complete sleep study.

Results: The two group were strictly similar for age and BMI (mean age 29.3 \pm 9.2 years, BMI 23.3 \pm 4.4 kg/m²). Total Sleep time (TST) was significantly reduced in EDS (343.7 \pm 69.3 min versus 395 ± 74.8 min; F= 11.9; p< 0.01). Sleep quality was significantly impaired, with a decreased Sleep efficiency (SE): 74.4 ± 10.5 versus 90.2 ± 7.8 F= 68.5; p< 0.001), an increased wake after sleep onset (WASO) time (116.5 \pm 45.7 min versus 43.3 \pm 36.8 min; F= 73.2; p <0.01) and micro arousal index (14.8 \pm 7.8 versus 6.5 \pm 4.3; F= 41.2; p<0.01). We also found a significant reduction of slow wave sleep length, but REM sleep was not affected. The apnea-hyponea index (IAH) and periodic leg movement index (PLMI) were higher in EDS patients (IAH: 10.8±4.8 versus 5.8±3.9; F= 30.4; p<0.01 et PLMI: 4.5 ± 4.6 versus 2.6 ± 2.5 ; F= 6; p<0.01). In patients with EDS type III, the prevalence of OSA (AHI>10/hour) was 75% versus 7.1 % in the control group (OR 5.1 (95% CI 2.3 to 14.7); p<0.001).

Conclusion: PSG may help in better understanding the diagnosis and treatments of EDS patients. **Support:**

1043

THE RELATIONSHIP BETWEEN NIGHTTIME EATING AND BODY MASS INDEX

Low, D.¹ Leroux, A.² Urbanek, J.¹ Crainiceanu, C.² ¹Johns Hopkins School of Medicine, Baltimore, MD, ²Johns Hopkins Bloomberg School of Public Health, Baltimore, MD.

Introduction: Late night eating has been associated with higher odds of being overweight or obese. This study aims to evaluate the relationship between late night eating and body mass index in a nationally representative sample.

Methods: Actigraphy was used to estimate the average bedtime, waketime, duration and midpoint of sleep in the National Health and Nutrition Examination Survey 2003-04 and 2005-06 cohorts. Given the circular nature of clock time, the average was calculated to be the point that minimized the sum of squares of differences between time points. Dietary data was collected through two detailed interviews of the participants. Nighttime calories were defined as the average amount of calories consumed between the average bedtime and the average midpoint of time-in-bed, based on the data recorded during the dietary interviews.

Results: Higher average nighttime caloric consumption (in units of 100 kcal) was associated with higher BMI [B(95% CI): 0.062 (0.003, 0.121)]; this remained significant after adjustment for age, gender, and race [B(95% CI): 0.084 (0.026, 0.142)]. Higher nighttime caloric consumption (as a percentage of total average daily calories consumption) was associated with higher BMI [B(95% CI): 1.522 (0.312, 2.733)]. This remained significant after adjustment for age, gender, and race [B(95% CI): 1.718 (0.505, 2.931)].

Conclusion: Higher nighttime caloric consumption, both in average amount (in units of 100 kcal) and as a percentage of average daily calories consumption, was associated with higher BMI. Additional study is needed to further elucidate the relationship between nighttime eating habits and body mass index. **Support:** NHLBI T32HL110952

1044

ADEQUATE SLEEP IS ASSOCIATED WITH IMPROVED DIABETES KNOWLEDGE AND HBA1C

Pandey, A.¹ Cooper, L.¹ Zrebiec, J.¹ Spadola, C.² Bennett, R. L.³ Rosenthal, M. M.⁴ Littlewood, K.¹

¹University of South Florida, Tampa, FL, ²Florida Atlantic University, Boca Raton, FL, ³Collaborative solutions, Tampa, FL, ⁴Data with Purpose, San Francisco Bay Area, CA.

Introduction: Although 55 % of the people with T2DM have low quality of sleep that may affect their physical and emotional wellbeing, and present challenges to the management of their condition, many Diabetes Self-Management Education Programs (DSME) that aim to improve knowledge of T2DM, don't include information on healthy sleep. This study will examine the relationship between adequate sleep on improved T2DM knowledge and diabetes maintenance (HbA1c).

Methods: The Sleep Integrated with Diabetes Education (SLIDE) Trial tests whether including four brief healthy sleep hygiene sessions within an existing traditional Diabetes Self-Management Education Program improves healthy sleep, motivation for change, and biopsychosocial outcomes for 50 patients with DM who are under and uninsured. This study uses descriptive and ANOVAs to examine the relationship between adequate sleep and change in diabetes knowledge (Diabetes Knowledge Test) using self-report. EMR was used to link HbA1c and other biological measures. **Results:** Fifty patients with T2DM (mean HbA1c = 8.79 ± 2.42) participating in a DSME Program at a southern urban community nonprofit hospital were randomly assigned to DSME classes or DSME classes + four 15-minute presentations (video and powerpoint) highlighting healthy sleep hygiene practices. The majority of these patients were obese (mean BMI=38.56±8.20). Only 11% reported normal sleep, with 41% reporting short sleep (<6 hours) and 7% long sleep (>8 hours). Patients who reported adequate sleep were more likely to improve diabetes knowledge (81% score vs. 68% score, p<.001) and HbA1c (1.1 vs. -.03, p<.001).

Conclusion: There is a relationship between adequate sleep and improving diabetes knowledge and maintenance for patients with T2DM. Future research could further explore this relationship and determine barriers and facilitators to adequate sleep and what role adequate sleep plays in improving T2DM knowledge and maintenance. **Support:** Bon Sequor Foundation

1045

IMPACT OF PRE-TREATMENT SLEEP AND MENOPAUSAL STATUS ON SLEEP QUALITY IN THE 12 MONTHS FOLLOWING A BREAST CANCER DIAGNOSIS

Squires, L.¹ Mahon, K.¹ Rash, J.¹ Powell, E.² Seal, M.² Garland, S. N.^{1,2,3}

¹Department of Psychology, Memorial University, St. John's, NL, CANADA, ²Discipline of Oncology, Memorial University, St. John's, NL, CANADA, ³Beatrice Hunter Cancer Research Institute, Halifax, NS, CANADA.

Introduction: Sleep disturbances are a prevalent and enduring problem in women who have completed treatment for breast cancer. Less is known about whether sleep during and after cancer treatment is influenced by pre-treatment sleep quality and menopausal status. The present study aims to examine the trajectory of sleep quality in the 12 months following a cancer diagnosis and assess whether trajectory is influenced by pre-treatment sleep quality and menopausal status.

Methods: Newly-diagnosed women (N=88) with non-metastatic BCa were recruited before beginning treatment. They completed the Pittsburgh Sleep Quality Index (PSQI) before treatment and 4, 8, and 12 months later. Women with a score ≥ 5 on the Pittsburgh Sleep Quality Index at treatment onset were classified as poor sleepers. Menopausal status (pre- or post-menopausal) was chart abstracted. A mixed ANOVA assessed the impact of pre-treatment sleep quality and menopausal status on sleep quality trajectory.

Results: The mean age of the sample was 60 years, 70% were classified as poor sleepers, and 72% were post-menopausal. There was a significant linear time by sleep quality interaction, F(1, 83)= 5.79, p =.02. Good sleepers experienced a greater initial worsening of sleep quality than poor sleepers. At 12 months, poor sleepers had returned to baseline levels whereas scores in good sleepers remained higher than baseline. The 3-way time x sleep quality x menopausal status and the 2-way time by menopausal status interactions were not significant.

Conclusion: Baseline sleep quality is a more powerful determinant of sleep trajectory during treatment than menopausal status. Early intervention is necessary to treat existing sleep problems and prevent the development of sleep problems in women with a history of good sleep.

Support: Dr. Garland is supported by a New Investigator Award and seed funding from the Beatrice Hunter Cancer Research Institute (BHCRI).

1046

ASSESSING SLEEP-RELATED HYPERTENSION RISKS USING JNC 8 GUIDELINES: ANALYSIS OF THE NATIONAL HEALTH AND NUTRITION EXAMINATION SURVEY DATA

Lough, L.¹ Seixas, A.¹ Avirappattu, G.² Robbins, R.³ Rogers, A.⁴ Williams, S.¹ Jean-Louis, G.¹

¹NYU Grossman School of Medicine, New York, NY, ²Kean University, Union, NJ, ³Harvard University, Cambridge, MA, ⁴St. John's University, Queens, NY.

Introduction: Associations between self-reported sleep duration and risk of hypertension (HTN) are well established. The level of sleep-related HTN risk based on the new JNC 8 classification guidelines requires further research. In this study, we modeled the associations of insufficient sleep with HTN using the National Health and Nutrition Examination Survey (NHANES).

Methods: Data were extracted from the 2006-2016 NHANES (n=38,540), a nationally representative study of the US civilian population. Self-reported demographic and sleep duration were determined from household interview questions. Insufficient sleep was categorized as sleeping <7hrs. Using 2017 ACC/AHA guide-lines, HTN was classified as elevated (SBP:120-129mmHg and DBP <80mmHg, Stage I (SBP:130-139mmHg and DBP:80-89mmHg), or Stage II (SBP ≥140mmHg and DBP ≥90mmHg). Logistic regression modeling was performed using R.

Results: Participants' ages ranged from 18-85 years. Of the sample, 51% were female, 41% white, 22% black, 26% Hispanic, 8% others; 46% were married, and 25% completed < high school. The model showed strong age and BMI-adjusted associations of insufficient sleep with HTN at all levels: (elevated: OR=1.079, CI=1.03-1.13; Stage I: OR=1.127, CI=1.07-1.18, and Stage II: OR=1.334, CI=1.17-1.52). Important sex and race/ethnicity differences in sleep-related HTN risks were observed: males (elevated: OR=1.024, CI=0.95-1.10; Stage I: OR=1.077, CI=1.01-1.15, and Stage II: OR=1.254, CI=1.06-1.48); females (elevated: OR=1.125, CI=1.05-1.21; Stage I: OR=1.170, CI=1.08-1.26, and Stage II: OR=1.445, CI=1.17-1.79); whites (elevated: OR=1.007, CI=0.93-1.08; Stage I: OR=1.030, CI=0.95-1.12, and Stage II: OR=1.131, CI=0.90-1.43); blacks (elevated: OR=1.047, CI=0.94-1.16; Stage I: OR=1.080, CI=0.97-1.20, and Stage II: OR=1.179, CI=0.95-1.46); and Hispanics (elevated: OR=1.066, CI=0.94-1.21; State I: OR=1.089, CI=0.96-1.24, and Stage II: OR=1.337, CI=0.92-1.92).

Conclusion: Our analyses showed that sleep-related HTN risks vary as a function of individual's sex and race/ethnicity. Increasing sleep duration at all HTN severity level is important and males and Hispanics at Stage II HTN might benefit the most.

Support: This study was supported by funding from the NIH: R01MD007716,R01HL142066, R01AG056531, T32HL129953, K01HL135452 and K07AG052685

1047

SOCIAL RHYTHM INSTABILITY IS ASSOCIATED WITH ABDOMINAL ADIPOSITY AFTER INVOLUNTARY JOB LOSS

Haynes, P. L.¹ Apolinar, G.¹ Thomson, C. A.¹ Quan, S.² Silva, G. E.³ Kobayashi, U.¹ Glickenstein, D. A.⁴ ¹Department of Health Promotion Sciences, Mel & Enid Zuckerman College of Public Health, University of Arizona, Tucson, AZ, ²Department of Medicine, College of Medicine, University of Arizona, Tucson, AZ, ³Biobehavioral Health Science Division, College of Nursing, University of Arizona, Tucson, AZ, ⁴Department of Mathematics, College of Science, University of Arizona, Tucson, AZ.

Introduction: Involuntary job loss is an acute stressor that disrupts daily time structure and activity and exacerbates economic hardship and psychological distress. Studies show that unemployment is associated with negative obesity-related health outcomes, such as metabolic syndrome. However, very little is known about daily routine, depression, and obesity in individuals who have recently experienced involuntary job loss. We hypothesized that individuals with less consistent daily routines, or unstable social rhythms, after job-loss would have more abdominal adiposity than individuals with more consistent social rhythms. We also hypothesized that this relationship would vary as a function of depressive symptoms. Methods: Cross-sectional baseline data (n = 186) from the ongoing ADAPT study (Assessing Daily Activity Patterns through occupational Transitions) were analyzed using linear regression techniques. Participants were predominantly female (62%) with a mean age of 41.12 years (SD = 10.16 years); 31% were Hispanic or Latino. Over two weeks, participants completed the daily Social Rhythm Metric-17 (SRM), Beck Depression Inventory II (BDI), and waist circumference (adiposity) measurements (cm).

Results: A significant BDI x SRM interaction was detected in the prediction of waist circumference, B = .36, SE = .18, <i>p </i> < .05, 95% CI [.002, .709], $R^2 = .07$). The SRM was inversely associated with waist circumference, B = -5.57, SE = 2.25, <i>p </i> < .05, 95% CI [-9.98, -1.13], only at lower levels of BDI (-1 SD below the mean). Results from the Johnson-Neyman technique identified that the conditional effect of SRM on waist circumference was statistically significant at a BDI raw score of 8.33 (0-13 points is minimal depression) with ~45% of cases within this region.

Conclusion: A less consistent daily routine was associated with a larger waist circumference among individuals with minimal depressive symptoms. These findings are the first to demonstrate a relationship between social rhythm stability and abdominal adiposity in adults at high risk for central obesity. Results highlight the moderating role of depression in obesity prevention. Future prospective analysis is necessary to examine causal pathways.

Support: #1R01HL117995-01A1

1048

PREVALENCE OF POSITIONAL OBSTRUCTIVE SLEEP APNEA (OSA) IN PATIENTS WITH OSA-COPD OVERLAP SYNDROME

Rani, S.¹ Rengan, R.¹ Mandal, M.¹ Ramzy, J.¹ VegaSanchez, M.¹ Jaffe, F.¹ Solar, X.² D'Alonzo, G.¹ Criner, G. J.¹ Chatila, W.¹ Shariff, T.¹ Weaver, S.¹ Krachman, S.¹

¹Lewis Katz School of Medicine at Temple University, Philadelphia, PA, ²University of California at San Diego, San Diego, CA.

Introduction: Positional OSA (non-supine apnea-hypopnea index [AHI] < 5 events/hr) is present in 30% of patients with OSA. We demonstrated that in patients with OSA- COPD overlap syndrome the AHI inversely correlated with the degree of gas trapping, suggesting a stabilizing effect on the upper airway. We hypothesized

that sleep position would be less important, resulting in a lower prevalence of positional OSA.

Methods: Patients underwent a polysomnogram that demonstrated OSA (AHI \geq 5 events/hr). To confirm COPD, patients had spirometry performed and a chest computed tomography for measurements of percent gas trapping.

Results: Sixteen patients [6 (38%) males, 55±7 years/old, FEV, 1.2±0.5 L, FEV, % Predicted 45±19%, FVC 2.3±0.8 L, FVC % Predicted 69±20%, FEV,/FVC 51±12%, BMI 33±9 kg/m²)] were diagnosed with OSA (AHI 15±12 events/hour). Four patients (25%) had positional OSA (AHI 13±6 events/hr, non-supine AHI 1±1 event/hr) compared to 12 patients who were non-positional [AHI 16±13 events/hr (p=0.95)]. There was no difference in age $[52\pm8 \text{ and } 56\pm7 \text{ yrs } (p=0.3)]$ or severity of obstruction in those with and without positional OSA [FEV, 1.4±4 L and 1.1±0.5 L, (p=0.3), FEV, % predicted 50±17% and 44±20%, (p=0.7), FVC 2.9±0.8 L and 2.1±0.8 L (p=0.1), FVC % predicted 78±21% and 66±20%, (p=0.3), and FEV₁/FVC 50±11% and 51±12%, (p=0.8), respectively]. However, patients with positional OSA were less heavy than those with non-positional OSA [BMI 23±3 and 37 ± 8 kg/m², respectively (p=0.005)]. Finally, there was no difference in the CT-Derived % Gas Trapping in those with and without positional OSA [$48\pm37\%$ and $36\pm25\%$, (p=0.6), respectively].

Conclusion: The prevalence of positional OSA in patients with OSA-COPD overlap is similar to OSA patients without COPD. Despite the presence of obstructive disease and gas trapping that may affect upper airway stability, other factors including body position and BMI remain important determinants for developing OSA in patients with COPD.

Support: R01-HL089856, R01-HL089897

1049

SLEEP FRAGMENTATION AND SLEEP RESTRICTION ARE ASSOCIATED WITH INCREASED ENERGY INTAKE AMONG INDIVIDUALS WHO HAVE INVOLUNTARILY LOST THEIR JOBS

Mayer, C. M.¹ Liu, Y.¹ Thomson, C. A.¹ Glickenstein, D. A.² Havnes, P. L.¹

¹Department of Health Promotion Sciences, Mel & Enid Zuckerman College of Public Health, University of Arizona, Tucson, AZ, ²Department of Mathematics, College of Science, University of Arizona, Tucson, AZ.

Introduction: Obesity is a major public health concern disproportionately affecting people of lower socioeconomic status. Involuntary job loss is a predictor of economic hardship and unemployment has been associated with poor sleep quality. Little is known about daily sleep and energy intake in this high-risk population. We hypothesized that disrupted, short sleep would be associated with increased energy intake among individuals who experienced involuntary job loss within the last 90 days.

Methods: Complete baseline data were analyzed from the ongoing Assessing Daily Activity Patterns through occupational Transitions (ADAPT) study. Over the same two-week period, participants (n = 191; 117 female; 63 Hispanic) were instructed to complete 14 days of daily sleep diaries and up to three 24-hour dietary recalls, conducted by trained interviewers using the Nutrition Data System for Research. The primary sleep variable was a composite score summarizing standardized number of awakenings and reversed-scored total sleep time; higher scores represented worse sleep. Energy intake was estimated as average 24 hour reported

kcals/day. Linear regression was employed with age, gender, and body mass index as covariates.

Results: Higher sleep composite scores were associated with higher self-reported intake in kilocalories (kcal), B = 84.83, SE = 38.01, t = 2.23, $\langle i \rangle p \langle /i \rangle \langle .05$. Wake time after sleep onset, sleep onset latency, and sleep efficiency were not associated with energy intake. Interestingly, higher subjective sleep quality was associated with consumption of more average daily kcals.

Conclusion: In a sample population of adults experiencing stress and economic hardship related to job loss, sleep continuity and quantity were associated with higher energy intake. With further prospective support, these findings suggest that public health interventions for obesity may benefit from behavioral sleep intervention components targeting both sleep fragmentation and sleep restriction. **Support:** #1R01HL117995-01A1

1050

SLEEP, THE MISSING DOMAIN FOR CARDIOVASCULAR HEALTH?

Foster, B. E.^{1,2} Collen, J. F.^{1,2} Eliasson, A. H.^{1,2} ¹Sleep Disorders Center, Walter Reed National Military Medical Center, Bethesda, MD, ²Uniformed Services University, Bethesda, MD.

Introduction: In developed countries, cardiovascular disease (CVD) is the leading cause of death. Identification and mitigation of CVD risk factors has been the chief strategy for prevention of CVD. To this end, the American Heart Association (AHA) and American College of Cardiology (ACC) suggest metrics of ideal cardiovascular (CV) health in seven domains and have published guidelines encouraging use of the "Simple Seven" to improve CV health. Sleep is notably absent from the Simple Seven despite substantial epidemiological evidence of its importance for CV health. We sought to measure the correlation of sleep parameters with CVD risk estimate by Framingham risk score (FRS).

Methods: Subjects prospectively enrolled in a CV Health Registry provided data for FRS, the seven domains of the "Simple Seven" including blood pressure, physical activity, total cholesterol, diet, weight, smoking, and glucose level, and completed validated questionnaires: Pittsburgh Sleep Quality Index (PSQI) and Berlin Questionnaire (BQ) for sleep apnea. Pearson correlations were analyzed between FRS and the "Simple Seven" as well as for the sleep questionnaires for comparison.

Results: Among the 646 subjects (mean age 55.4 ± 9.9 y, 43% men, 399W, 190B, 26H, 12A, 19other), FRS correlated substantially with blood glucose (R=0.398, p<0.00001), moderately with blood pressure (R=0.220, p<0.00001), and mildly with weight (r=0.145, p-0.0002) and physical activity (R=0.110, p=0.01). A negative (paradoxical) correlation was identified between total cholesterol and diet when compared to FRS. In comparison, FRS did correlate mildly with BQ (R=0.114, p=0.0002) and PSQI (r=-0.103, p=0.009).

Conclusion: This study suggests that sleep parameters, specifically BQ and PSQI, may contribute to risk assessment of CVD while also offering actionable information for improving CV health. **Support:**

1051

LEVEL OF AGREEMENT BETWEEN OBJECTIVE AND SUBJECTIVE SLEEP MEASURES IN LUNG CANCER SURVIVORS WITH INSOMNIA

Weiss, C.¹ Kwon, M.² Dickerson, S.² Dean, G.²

¹Jacobs School of Medicine and Biomedical Sciences, State University of New York at Buffalo, NY, ²School of Nursing, State University of New York at Buffalo, NY.

Introduction: Lung cancer survivors (LCS) have the secondhighest incidence of impaired sleep among cancer population. Clinical studies use self-reported and objective measures to assess insomnia in LCS. However, negative beliefs of sleep patterns may impair sleep perception, impact psychological state, and influence insomnia treatment and survivorship outcomes. This study aims to assess the level of agreement between subjective and objective sleep measures in LCS.

Methods: Forty-four non-small cell lung cancer survivors, stage I-III, at least 6 weeks after treatment completion, with Insomnia Severity Index >7, were recruited from two sites from 2014-2016. Individuals with sleep apnea, menopause, rotating shift work, uncontrolled substance abuse, or unstable medical/psychiatric illnesses were excluded from the study. Agreement between total sleep time (TST), sleep latency (SL) and sleep efficiency (SE) from Sleep Diary and wrist-actigraphy (i.e. CamNtech) were assessed with Intraclass Correlation (ICC) and Bland-Altman plots. Analysis of ICC, mean difference, standard deviation (SD) and 95% confidence interval (CI) were conducted using SPSS 24.

Results: LCS were male (63.6%), 60-69 years old (52.3%), married or living with a partner (50%). Self-reported measurements underestimated TST (-68.2 minutes, ICC 0.45, CI 95%, SD 108.1) and SL (-25.9 minutes, ICC 95%, ICC -0.54, DS 17.5). Objective and self-reported measurements largely overlapped for SE, with a small actigraphy-based overestimation (9.2, ICC 0.2, CI95%, SD 7.3). Bland-Altman plots revealed that the agreement increased with lower SE values.

Conclusion: LCS tended to underestimate TST and SL. An apparent agreement between objective and self-reported measures for SE may indicate sleep state misperception. Differences in various sleep parameters and potential covariates should be investigated for contribution to the unexplained considerable individual variability in behavioral treatment response among cancer survivors in larger sample sizes.

Support: 1R01NR018215-01 (GED); T32GM099607(CW).

1052

CONTINUOUS BLOOD PRESSURE MONITORING IN SLEEP

Roth, R. H.¹ Bonner, H.² Logan, J.³ Baruch, M.⁴ Calhoun, D.⁵ Berry, R.⁶ Cho, Y.¹ Kwon, Y.¹

¹University of Virginia School of Medicine, Charlottesville, VA, ²University of Virginia Sleep Disorder Center, Charlottesville, VA, ³University of Virginia School of Nursing, Charlottesville, VA, ⁴Caretaker Medical, Charlottesville, VA, ⁵University of Alabama School of Medicine, Birmingham, AL, ⁶University of Florida College of Medicine, Gainesville, FL.

Introduction: Abnormal nocturnal blood pressure(BP) such as non-dipping or nocturnal hypertension(reverse-dipping) represents a potent marker for cardiovascular risks. Standard cuff-based ambulatory nocturnal BP measurement yields limited data points potentially resulting in imprecise results, especially compared to continuously recorded BP. We hypothesized nocturnal BP based on periodic measurement would be different from true average beat-to-beat based BP.

Methods: We prospectively enrolled patients undergoing clinically indicated in-lab polysomnography, both CPAP and non-CPAP

studies, for sleep apnea evaluation. Nocturnal BP was continuously monitored beat-to-beat by a noninvasive CaretakerTM device, which uses the Pulse Decomposition Analysis(PDA) algorithm. We compared BP recorded at 30-minute intervals with average BP continuously recorded over 30 minutes, both recorded by CaretakerTM. We also looked at the differences between recording spot and continuous BP from an awake or sleeping state and BP variability(SD) based on continuously recorded BP. Using first 30 min as a reference, we determined dipping status (dipping: 10-20% reduction, level: 0-10% reduction, riser: any increase) by the two methods.

Results: A total of 18 patients were recruited(male:11, mean age:52.2). Among a total of 261 periodic BP measurements, 60 (30.0%) were obtained while awake. Mean nocturnal SBP by periodic BP measurement was higher compared with beat-to-beat-derived average BP(135.6mmHg[24.2] vs. 131.5[20.3], p<0.0001). The difference between the two methods remained similar when continuous BP was derived from sleep vs. awake period(4.5mmHg[3.1] vs. 7.7[9.9], p=0.202). BP variability was more pronounced during awake compared with sleep period(6.7mmHg[8.1] versus 3.95[7.5], p=0.047). 8 patients were dippers by spot check measurement, but 11 were dippers by continuous BP.

Conclusion: Standard ambulatory periodic nocturnal BP recording may not yield true sleep BP patterns due to its spot-check nature and lack of sleep-awake information, which leads to inaccurate dipping measurements. Incorporation of beat-to-beat continuous BP measurement can provide more accurate and meaningful nocturnal BP information.

Support: N/A

1053

SLEEP DURATION AND TIMING ASSOCIATED WITH HISTORY OF BREAST PROSTATE AND SKIN CANCER: DATA FROM A NATIONALLY-REPRESENTATIVE SAMPLE

Piro, B.¹ Garland, S.² Jean-Pierre, P.³ Gonzalez, B.⁴ Seixas, A.⁵ Muench, A.⁶ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹ ¹University of Arizona, Tucson, AZ, ²Memorial University of Newfoundland, St. John's, NL, CANADA, ³Florida State University, Tallahassee, FL, ⁴Moffit Cancer Center, Tampa, FL, ⁵New York University, New York, NY, ⁶University of Pennsylvania, Philadelphia, PA.

Introduction: Sleep disturbances are a common problem among cancer survivors. Also, cancer patients can have altered circadian rhythms and these changes can continue to affect the patient long after the conclusion of their treatment. This analysis aims to investigate how the sleep and wake times of cancer survivors differ from the rest of the population, depending on the type of cancer.

Methods: Data from the 2015-2016 National Health and Nutrition Examination Survey were used. Population-weighted data on N=5,581 individuals provided complete data. History of breast, prostate, and skin cancer (melanoma or other) was self-reported. Sleep duration was self-reported in half-hour increments, and typical bedtime and waketime was self-reported. Covariates included age, sex, and race/ethnicity. Weighted linear regressions with sleep duration, bedtime and waketime were examined, with each cancer type as predictor.

Results: Prevalence was 1.7% for prostate cancer, 1.5% for breast cancer, 2.3% for non-melanoma skin cancer, and 0.8% for melanoma. In adjusted analyses, prostate cancer was associated with an additional 26.5 minutes of average total sleep (95%CI 2.2,50.9, p=0.03), a 23.1 bedtime minutes earlier (95%CI -40.4,-5.8,

p=0.009), and no difference in waketime. Breast cancer was associated with a bedtime that was 41.1 minutes later (95%CI 10.3,72.0, p=0.009) and a waketime that was 48.7 minutes later (95%CI 12.5,84.9, p=0.008), but no difference in sleep duration. No statistically significant effects were seen for either type of skin cancer, melanoma or non-melanoma.

Conclusion: Prostate cancer was associated with an earlier bedtime and associated increased sleep time. Breast cancer, on the other hand, was associated with a phase delay of the sleep period but no change in sleep duration. Skin cancer was not associated with differences in sleep duration or timing. These findings may have implications for not only treatment of sleep problems in different types of cancer, but also possible circadian mechanisms. **Support:** Dr. Grandner is supported by R01MD011600

1054

IMPROVING SLEEP BY REDUCING ALLERGY SYMPTOMS

Rus, H. Danoff-Burg, S. Cruz Martir, L. Raymann, R. J. SleepScore Labs, Carlsbad, CA.

Introduction: Allergies can impact sleep quality and, in turn, quality of life. Sleep is affected because allergens irritate nasal passages, which can make breathing more difficult, start an immune response, and directly interfere with sleep. Nasal congestion often is worse at night, and during the night some anti-allergic control processes of the body dip, which can lead to increased symptoms. We tested if an air purifier in the bedroom would improve sleep and reduce allergy symptoms.

Methods: 29 adults with self-reported allergy complaints participated in an 8-week field study, using a non-counterbalanced pre-post intervention design. Intervention consisted of the use of an Alen BreatheSmart 45i air purifier at home for 24 hours a day during the duration of the test period. Sleep was measured with self-report and objectively with ResMed S+ every night. Allergy symptoms were measured using self-report. Paired t-tests and multilevel regression were used to test for statistical significance.

Results: 28 individuals reported a reduction in allergy symptoms and 27 reported better sleep during the intervention. Overall, participants reported a 38% improvement in allergy symptoms and a 59% improvement in sleep continuity. Moreover, participants felt they fell asleep faster (20% faster), experienced better sleep quality (14% better), and felt more rested in the morning (14% more rested). Objective data showed an 11% improvement of Wake After Sleep Onset and a 1.1% increase in sleep efficiency during intervention (all ps < 0.05), but only in the subgroup (n = 11) of participants with the poorest sleep at baseline.

Conclusion: Using an air purifier in the bedroom can reduce allergy symptoms and improve sleep. Objectively measured sleep improvements supported the self-reported improvements, but only in the subset of participants who showed compromised sleep at baseline.

Support: Alen Corporation

1055

CARDIOMETABOLIC DISEASE RISK AND SLEEP DIFFICULTY: ASSOCIATIONS WITH DIET, PHYSICIAN SUPPORT, PROACTIVE HEALTH BEHAVIORS, AND FAMILY RISK KNOWLEDGE

Gilles, A. Delgado, G. University of Hawaii - West Oahu, Kapolei, HI. **Introduction:** Research relating sleep impairment and cardiometabolic disease supports exercise and diet in improving both conditions. The role of self- and other-informed knowledge on self-care behaviors is unknown. This study investigated how proactive health behaviors, physician support, and familial disease awareness are related in this comorbid population.

Methods: National Health and Nutrition Examination Survey (NHANES) data from 2015-2016 was used. For this study, U.S. adults (N=9,971;49.3% female) with dietary, sleep, cardiometabolic disease risk, proactive health behavior, physicianinformed, and familial knowledge of cardiometabolic disease data were selected. Self-reported sleep difficulty was defined as ever telling a doctor/health professional of trouble sleeping. Kendall's tau-b correlations and multinomial regression were performed. Covariates included race, gender, weight control, dietary efforts, and exercise.

Results: Statistically significant associations between comorbid cardiometabolic disease/sleep difficulty (CMD/SD) and efforts to control/lose weight($X^2(6)=63.956, p>0.0001$), reduce dietary $salt(X^{2}(6)=69.702, p>0.0001)$, and reduce dietary $fat(X^{2}(6)=70)$.666,p>0.0001) were found. Despite having comorbid CMD/ SD, most participants (51%) reported no history of receiving physician-informed health improvement methods. However, statistically significant associations between comorbid CMD/SD and physician-informed methods and weight loss efforts($X^2(4)=76.87$ 3,p>0.0001), increased exercise($X^{2}(4)=72.713,p>0.0001$), reduced dietary salt($X^2(4)=96.892, p>0.0001$), and reduced dietary fat($X^2(4)=96.892, p>0.0001$) 4)=104.231,p>0.0001) were found. Statistically significant associations between comorbid CMD/SD and knowledge of close relative with heart attack($X^{2}(9)=23.905, p=0.004$) or diabetes($X^{2}(9)=1$ 29.705,p>0.0001) were found. Participants with comorbid CMD/ SD were more likely to reduce dietary fat($X^2(1)=4.575, p < 0.032$) than participants with comorbid sleep difficulty/cardiovascular disease or comorbid sleep difficulty/metabolic disease. Although no association between age of cardiometabolic diagnosis onset and proactive health behaviors was found, the regression model showed that male gender(p=0.008) and reducing dietary fat was predictive of comorbid CMD/SD(p=0.032).

Conclusion: Participants with comorbid CMD/SD directed proactive health efforts towards eating behavior (less food/decreasing salt and fat). With physician-informed support, participants additionally increased exercise level. Further exploring the role of familial disease knowledge, gender-specific support, and innovative efforts by health professionals in treating/preventing comorbid CMD/SD is warranted.

Support: None

1056

PERCEIVED FINANCIAL DIFFICULTY PREDICTS SLEEP QUALITY IN PARTICIPANTS WITH TYPE 2 DIABETES AND OBSTRUCTIVE SLEEP APNEA

Morris, J. L.¹ Baniak, L.² Belcher, S. M.³ Imes, C.¹ Luyster, F.¹ Scott, P. W.¹ Sereika, S.¹ Chasens, E. R.¹

¹University of Pittsburgh, Pittsburgh, PA, ²VA Pittsburgh Healthcare System, Pittsburgh, PA, ³Emory University, Atlanta, GA.

Introduction: People with multiple chronic conditions such as type 2 diabetes (T2D) and obstructive sleep apnea (OSA) are at increased risk for poor sleep quality. It is unclear if social determinants of health (SDoH) such as race, perceived financial difficulty, education, gender, and marital status are associated with

sleep quality in this population. The purpose of this cross-sectional secondary analysis of data from the Diabetes Sleep Treatment Trial was to explore SDoH and disease severity as predictors of sleep quality in persons with both OSA and T2D.

Methods: Disease severity was measured by Apnea-Hypopnea Index [(AHI) \geq 5] and A1C for glycemic control. SDoH included perceived financial difficulty (none/moderate-severe), race (White/ African American), sex (f/m), marital status (no/yes), education (\leq or > 2 years post high school), and age. Sleep quality was measured by Pittsburgh Sleep Quality Index (PSQI). Correlations and linear regression modeling investigated associations between SDoH and disease severity on sleep quality. Post-hoc correlations were explored for significant relations among SDoH.

Results: The sample (N = 229) was middle-aged (57.6 ± 10.0; 66 % White and 34% African American; and 54 % men vs. 46% women. Participants carried a high burden of disease (mean AHI = 20.7±18.1, mean A1C = 7.9 %±1.7%). Disease severity was not significantly associated with sleep quality (all p > .05). The perception of worse financial difficulty was the only SDoH that predicted worse sleep quality (b=-1.54, p=.015). Characteristics significantly associated with worse financial difficulty were being African American, female, ≤ 2 years post high school, and younger (all p < .01).

Conclusion: Financial difficulty may be a more important predictor of subjective measures of sleep quality than disease severity in patients with OSA and T2D. Researchers and clinicians should be aware of these characteristics as potential markers of vulnerability to poor sleep quality in this population.

Support: The National Institute of Diabetes and Digestive and Kidney Diseases (R01DK096028) and through the Clinical +Translational Research Institute grants UL1TR001857 and UL1TR000005.

1057

EVALUATING DIFFERENCES IN UPPER AIRWAY ANATOMY BETWEEN DIABETIC AND NON-DIABETIC OSA PATIENTS

Ihemeremadu, N. Lavi-Romer, N. Zang, Y. Keenan, B. Schwab, R. Penn Sleep Medicine, Philadelphia, PA.

Introduction: Studies show that OSA is linked to impaired glucose tolerance, insulin resistance, and the onset of diabetes. We hypothesized that diabetic OSA patients will have higher apnea-hypopnea index (AHI) values than OSA patients without diabetes after adjusting for age and body mass index (BMI) and that this difference can be explained through increases in upper airway structures between diabetic and non-diabetic OSA patients.

Methods: This study evaluated differences in upper airway and craniofacial dimensions and volume of the pharyngeal soft tissues between diabetic and non-diabetic patients with obstructive sleep apnea (OSA) using magnetic resonance imaging (MRI). Airway sizes, soft tissue volumes and craniofacial dimensions were quantified using three-dimensional MRI in OSA patients without diabetes (n=237) and OSA patients with diabetes (n=64). Comparisons in upper airway measures among diabetics and non-diabetics were performed using linear regression models controlling for age, sex, BMI, race, and AHI.

Results: Among study participants, diabetic OSA patients were older than non-diabetic OSA patients (54.2 ± 10.1 vs. 47.3 ± 11.1 years; p<0.0001). No significant differences were found between diabetic and non-diabetic OSA patients with respect to BMI (39.8 ± 7.0 vs. 38.4 ± 8.8 kg/m²; p=0.207) or AHI (45.0 ± 31.0

vs. 38.8±27.8 events/hour; p=0.154). In covariate adjusted models, non-diabetic OSA patients also had smaller RP minimum airway area (adjusted difference [95% CI] = -3119 [-5359, 879] mm²; p=0.0066) and RP minimum AP distance (-16.0 mm [-29.6, -2.5]; p=0.021) compared to diabetic OSA patients. No differences were observed in soft tissue volumes or craniofacial dimensions.

Conclusion: While diabetics had higher average AHI, we observed no significant differences in AHI between diabetic and non-diabetic patients with sleep apnea. In general, upper airway anatomy was similar between diabetic and non-diabetics apneics, controlling for demographic factors and AHI. Future studies should examine dynamic changes, in addition to static upper airway anatomy, in diabetic and non-diabetics apneics. **Support:**

1058

MODELING SELF-REPORTED SLEEP DURATION AND HYPERTENSION USING DEEP LEARNING NETWORK: ANALYSIS OF THE NATIONAL HEALTH AND NUTRITION EXAMINATION SURVEY DATA

*Williams, S.*¹ *Seixas, A.*¹ *Avirappattu, G.*² *Robbins, R.*³ *Lough, L.*¹ *Rogers, A.*⁴ *Beaugris, L.*² *Bernard, M.*⁵ *Jean-Louis, G.*¹

¹NYU Grossman School of Medicine, New York, NY, ²Kean University, Union, NJ, ³Harvard University, Cambridge, MA, ⁴St.

John's University, Queens, NY, $^5\mathrm{New}$ York University, New York, NY.

Introduction: Epidemiologic data show strong associations between self-reported sleep duration and hypertension (HTN). Modeling these associations is suboptimal when utilizing traditional logistic regressions. In this study, we modeled the associations of sleep duration and HTN using Deep Learning Network. Methods: Data were extracted from participants (n=38,540) in the National Health and Nutrition Examination Survey (2006-2016), a nationally representative study of the US civilian noninstitutionalized population. Self-reported demographic, medical history and sleep duration were determined from household interview questions. HTN was determined as SBP ≥ 130 mmHg and $DBP \ge 80 \text{ mmHg}$. We used a deep neural network architecture with three hidden layers with two input features and one binary output to model associations of sleep duration with HTN. The input features are the hours of sleep (limited to between 4 and 10 hours) and its square; and the output variable HTN. Probability predictions were generated 100 times from resampled (with replacement) data and averaged.

Results: Participants ranged from 18 to 85 years old; 51% Female, 41% white, 22% black, 26% Hispanic, 46% married, and 25% < high school. The model showed that sleeping 7 hours habitually was associated with the least observed HTN probabilities (P=0.023%). HTN probabilities increased as sleep duration decreased (6hrs=0.05%; 5hrs=0.110%; 4hrs=0.16%); HTN probabilities for long sleepers were: (8hrs=0.027; 9hrs=0.024; 10hrs=0.022). Whites showed sleeping 7hrs or 9hrs was associated with lowest HTN probabilities (0.008 vs. 0.005); blacks showed the lowest HTN probabilities associated with sleeping 8hrs (0.07), and Hispanics showed the lowest HTN probabilities sleeping 7hrs (0.04).

Conclusion: We found that sleeping 7 hours habitually confers the least amount of risk for HTN. Probability of HTN varies as a function of individual's sex and race/ethnicity. Likewise, the finding that blacks experience the lowest HTN probability when they sleep habitually 8 hours is of great public health importance. **Support:** This study was supported by funding from the NIH: R01MD007716, R01HL142066, R01AG056531, T32HL129953, K01HL135452, and K07AG052685.

1059

SLEEP PARAMETERS IN PEOPLE WITH TYPE 2 DIABETES WITH AND WITHOUT INSOMNIA SYMPTOMS

Alshehri, M.¹ Alkathiry, A.² Alenazi, A.³ Alothman, S.⁴ Rucker, J.⁵ Phadnis, M.⁶ Miles, J.¹ Kluding, P.¹ Siengsukon, C.¹ ¹University of Kansas Medical Center, Kansas City, KS, ²Physical Therapy department, Majmaah University, Almajmaah, Central Region, Saudi Arabia, SAUDI ARABIA, ³Physical Therapy department, Prince Sattam Bin Abdulaziz University, Alkharj City, Central Region, Saudi Arabia, SAUDI ARABIA, ⁴Physical Therapy and Rehabilitation Science Depart, Kansas City, Kansas, KS, ⁵University of Kansas Medical Center, Kansas City, Kansas, KS, ⁶University of Kansas Medical Center, Kansas City, Kansas, KS.

Introduction: There is an increasing awareness of the high prevalence of insomnia symptoms in people with type 2 diabetes (T2D). Past studies have demonstrated the importance of measuring sleep parameters in both averages and variabilities using subjective and objective methods. Thus, we aimed to compare the averages and variability of sleep parameters in people with T2D with and without insomnia symptoms.

Methods: Actigraph measurements and sleep diaries were used in 59 participants to assess sleep parameters, including sleep efficiency (SE), sleep latency, total sleep time, and wake after sleep onset over seven nights. Validated instruments were used to assess the symptoms of depression, anxiety, and pain. Circular data were used to describe the distribution of bed distribution with SE as a magnitude for both groups. Mann Whitney U test was utilized to compare averages and variability of sleep parameters between the two groups. Multivariable general linear model to control for demographic and clinical variables. For the secondary aim, multiple linear regression tests were utilized to assess the association between averages and variability values for both groups.

Results: SE was found to be lower in average and higher in variability for participants with T2D and insomnia symptoms, than those with T2D only subjectively and objectively. SE variability was also the only sleep parameter higher in people with T2D and insomnia symptoms, with psychological symptoms potentially playing a role in this difference. We observed that people in T2D+Insomnia tend to go to bed earlier compared to the T2D only group based on objective measures, but no difference was observed between groups in subjective measures. The only significant relationship in both objective and subjective measures was between the averages and variability of SE.

Conclusion: Our findings suggest a discrepancy between subjective and objective measures in only average of total sleep time, as well as agreement in measures of variability in sleep parameters. Also, the relationship between averages and variabilities suggested the importance of improving SE to minimize its variability. Further research is warranted to investigate the complex relationship between sleep parameters and psychological factors in people with T2D and insomnia symptoms.

Support: None

1060

SCREENING FOR OBSTRUCTIVE SLEEP APNEA IN SEVERE GASTROESOPHAGEAL REFLUX DISEASE PATIENTS: A FORGOTTEN ASSOCIATION?

De, A. Gharib, H. Frenia, D. Velpari, S. Saint Peter's University Hospital, New Brunswick, NJ.

Introduction: Obstructive sleep apnea (OSA) is an emerging epidemic in the USA and remains underdiagnosed. Investigations of Gastroesophageal reflux disease (GERD) poses a substantial burden on patient-welfare and costs to the health system. Current literature has highlighted the association between severe GERD and OSA, and other sleep disorders. We conducted a retrospective analysis of patient records undergoing Bravo pH monitoring for refractory GERD to measure the prevalence of OSA and screening. **Methods:** Records of patients who underwent outpatient Bravo pH monitoring at a teaching hospital were reviewed from August 2018 to May 2019. 72 records were reviewed in our analysis. Analysis variables included age, gender, body mass index, history of hypertension and OSA. Outpatient records were reviewed for documentation for OSA or screening and demographics were obtained for calculation of a partial STOP-BANG score (a validated OSA screening tool).

Results: 8 out of 72 (11%) were excluded due to incomplete documentation regarding their history. Of the remaining 64, 2 had a known diagnosis of OSA (3%) and 1 was due a sleep study for maintenance insomnia. Of the remaining 61 patients, none had documentation of a history pertaining to sleep complaints or full screening for OSA. 4 of the 8 components to the STOP-BANG criteria were documented and used to measure risk of OSA in these patients. 23 (39%) patients had a score of 3 or above characterizing them as intermediate risk. The other 4 components were not used due to a lack of clinical information. 13 of these patients had a positive Bravo test, 2 had an inconclusive result and 8 had a negative result. Of the 61 patients in total, 31 had a positive Bravo result and 9 had an inconclusive result.

Conclusion: In our study, we found that 39% of patients based on demographic data were of intermediate risk of OSA. Over half of these patients had a positive result for GERD. Despite the increased awareness of sleep disorders it is still neglected despite prevalent associated comorbid conditions. This study highlights the failure to screen for this modifiable risk factor within a teaching environment. **Support:** None

1061

PROBLEMS DURING DAYTIME DUE TO POOR SLEEP ARE ASSOCIATED WITH INDICATORS OF POOR DENTAL HEALTH IN A LARGELY NON-SMOKING POPULATION

Postolache, T. T.^{1,2,3} Wadhawan, A.^{1,4} Daue, M. L.^{5,6,7} Dagdag, A.¹ Reynolds, M. A.⁸

¹Mood and Anxiety Program, University of Maryland School of Medicine, Baltimore, MD, ²Rocky Mountain Mental Illness Research Education and Clinical Center (MIRECC), Veterans Integrated Service Network (VISN) 19, Military and Veteran Microbiome Consortium for Research and Education (MVM-CoRE), Denver, CO, ³Mental Illness Research, Education and Clinical Center (MIRECC), Veterans Integrated Service Network (VISN) 5, VA Capitol Health Care Network, Baltimore, MD, ⁴Saint Elizabeth's Hospital, Department of Psychiatry, Washington, DC, ⁵Division of Endocrinology, Diabetes and Nutrition, Department of Medicine, University of Maryland School of Medicine, Baltimore, MD, ⁶Program for Personalized and Genomic Medicine, University of Maryland School of Medicine, Baltimore, MD, ⁷Geriatrics Research and Education Clinical Center, Veterans Affairs Medical Center, Baltimore, MD, ⁸Department of Advanced Oral Sciences & Therapeutics, University of Maryland School of Dentistry, Baltimore, MD.

Introduction: Growing evidence connects periodontal disease, a major and modifiable cause of local and systemic inflammation, with metabolic and cardiovascular morbidity and mortality, as well as mental illness. Sleep has been previously predictively associated with metabolic and psychiatric morbidity and mortality, and has recently been linked with periodontal disease. We are now evaluating associations between self-reported insomnia measures and surrogate indicators of periodontal disease in a population with a very low prevalence of smoking — a major confounder in previous studies.

Methods: Dental and sleep questionnaires (Pittsburgh Sleep Quality Index) were obtained from 3881 Old Order Amish from Lancaster county. Difficulty falling or staying asleep, sleep quality, problems during the day due to poor sleep, and sleepiness during daytime were related to self-reported loose teeth, partial dentures, full dentures and any dentures using linear models with adjustment for age and sex.

Results: Significant associations emerged between problems falling asleep and loose teeth (p<0.05), problems staying asleep and any dentures (p<0.05), sleep quality with loose teeth and partial, as well as complete dentures (p<0.05 for both). Problems during daytime due to poor sleep were associated with loose teeth (p<0.05), any dentures (p<0.003) and full dentures (p<0.0001 — the only associations resisting Bonferroni correction). Sleepiness during daytime, which is the most important marker associated with sleep apnea was not associated with any dental health measures.

Conclusion: Limitations include not accounting for family aggregation, limited generalizability, not fully differentiating between respiratory versus non-respiratory sleep impairment, and periodontal versus traumatic dental pathology. Yet, the results of our study, which minimizes the strong potential confounding by smoking, confirm associations between sleep-related problems and periodontal disease, and justify future longitudinal and interventional research to dissect causality and identify multi-target treatment modalities.

Support: This work was supported by the Mid-Atlantic Nutrition Obesity Research Center Preliminary Developmental NORC grant (Postolache, PI), a sub-award of the parent grant P30 DK072488 (Mitchell, PI), and intramural funds from the University of Maryland, Baltimore.

1062

THE ROLE OF SLEEP IN SEX AND RACIAL/ETHNIC DIFFERENCES IN 10-YEAR CVD RISK IN THE SLEEP HEART HEALTH STUDY: THE USE OF MACHINE-LEARNT AND PRECISION INSIGHTS TO UNDERSTAND RACIAL/ETHNIC AND SEX DIFFERENCES IN SLEEP-CVD DISPARITY

Seixas, A.¹ Jin, P.¹ Liu, M.¹ Nunes, J.² Grandner, M.³ Rogers, A.⁴ McFarlane, S.⁵ Jean-Louis, G.¹

¹NYU Grossman School of Medicine, New York, NY, ²City University of New York School of Medicine, New York, NY, ³University of Arizona College of Medicine, Tuscon, AZ, ⁴St. John's University, Queens, NY, ⁵State University of New York, Downstate Health Science University, New York, NY.

Introduction: The current study investigated whether insufficient sleep (<7 hrs.) explains differences in 10-year CVD risk, using

Framingham risk (FRS) and Reynolds risk (RRS) scores, between blacks and whites and characterized risk and protective CVD risk profiles.

Methods: Using the Sleep Heart Health Study (SHHS) (N=6,441) data, we investigated the independent role of insufficient sleep in explaining differences in 10-years CVD between blacks and whites via a proportional odds model of four 10-year CVD risk groups: low (<5%), low-medium (5% to <10%), medium-high (10% to <20%) and high (\geq 20%), adjusting for age, sex, and apnea-hypopnea index (AHI). We performed two levels of cluster analyses; via hierarchical cluster algorithm with entire sample (Level 1), and latent profiles in the low (protective profiles) and high (risk profiles) CVD risk groups (Level 2) to determine overall CVD risk, and risk and protective CVD profiles.

Results: Blacks had a higher prevalence of smoking behavior, diabetes, mean systolic blood pressure, body mass index, total cholesterol compared to whites. Conversely, whites had a higher mean HDL cholesterol, sleep hours, and sleep efficiency compared to blacks. Men had higher 10-year CVD risk than women. AHI and race/ethnicity-sleep interaction were positively associated, while sleep was negatively associated with FRS and RRS. Across all CVD risk groups, whites who slept less than 5.5 hrs. had a higher CVD risk and those who slept more than 6.5 hrs. had a lower CVD risk compared to blacks. In Level 1 cluster analyses, we found two clusters: Cluster 1 (n= 3233): 6.17 sleep hours, apnea-index 11.84, age 59, SBP 125.43, total cholesterol 209, HDL 51.39, BMI 29.03, and slightly more than 50% female; and Cluster 2 (n=1657): 5.61 sleep hours, apnea-index 13.41, age 74, SBP 131, total cholesterol 204, HDL 50.30, BMI 26.45, and slightly less than 50% female. In Level 2 cluster analyses, we found two profiles within the low and high CVD risk groups.

Conclusion: These findings suggest that blacks may not receive full protection from long-term CVD risk with longer sleep duration, as their white counterparts.

Support: K01HL135452, R01MD007716, R01HL142066, K07AG052685

1063

DEPRESSION MODERATES THE ASSOCIATION BETWEEN POSTTRAUMATIC STRESS DISORDER AND NIGHTMARE SEVERITY IN NURSES

Shapiro, T.¹ Messman, B.¹ Slavish, D. C.¹ Alkire, C.¹ Wardle-Pinkston, S.² Dietch, J.³ Kelly, K.¹ Ruggero, C.¹ Taylor, D.² ¹University of North Texas, Denton, TX, ²University of Arizona, Tuscon, AZ, ³Palo Alto Veterans Affairs Health Care System, Palo Alto, CA.

Introduction: Nurses report a higher prevalence of posttraumatic stress disorder (PTSD) than the general population, and approximately 18% of nurses report having depression. Nightmares are a common symptom of PTSD, and both nightmares and PTSD are correlated with depression. Nightmares may represent a possible clinical target for improving outcomes in both disorders. This study assessed associations between PTSD and depressive symptoms with nightmare severity, and whether depressive symptoms moderated associations between PTSD and nightmare severity.

Methods: Participants were 461 nurses (91% female; 77% white, mean age = 38.39 years) recruited from two hospitals for a parent study. Participants completed the Patient Health Questionnaire (PHQ-9), Post-traumatic Stress Disorder Checklist (PCL-5), and 14 days of sleep diaries to assess daily nightmare frequency and severity (on a scale of 0 = not at all severe to 3 = very severe).

Results: 22.1% of participants reported at least one nightmare across the 14 days, with a mean daily nightmare frequency of 1.17 (SD = 2.15) and a mean severity of 0.11 (SD = 0.18). PCL-5 and PHQ-9 scores were significantly correlated with nightmare severity (r = 0.27; r = 0.24, respectively) and each other (r = 0.69). PHQ-9 scores moderated the association between PCL-5 scores and nightmare severity ($\beta = -.01$, SE = <0.01, p = 0.015). For individuals 1 SD below the PHQ-9 mean, higher PCL-5 scores were associated with higher nightmare severity. For individuals 1 SD above the PHQ-9 mean, higher PCL-5 scores were associated with higher nightmare severity, but to a lesser degree.

Conclusion: Both depressive and PTSD symptoms were associated with more severe nightmares. Surprisingly, the association between PTSD symptoms and nightmare severity was stronger for those with lower depressive symptoms. Results suggest depression, PTSD, and nightmares may represent a partially overlapping symptom cluster. Research should investigate how nightmare treatment may reduce PTSD and depressive symptoms. Support: NIAID R01AI128359-01

1064 SLEEP PATTERNS AMONG URBAN HAITIAN EARTHQUAKE SURVIVORS WHO EXPERIENCED THE TRAUMA OF NONPARTNER SEXUAL VIOLENCE: A LATENT CLASS ANALYSIS APPROACH

Rahill, G. J.¹ Joshi, M. J.¹ Blanc, J.² Rice, C.³

¹University of South Florida, Tampa, FL, ²NYU Langone Medical Center, New York, NY, ³Florida International University, Miami, FL.

Introduction: Sleep health is crucial to recovery from trauma. Haiti's Cité Soleil residents (approximately 350,000) live in extreme poverty and regularly experience or witness life-threatening events, including gang and non-partner sexual violence (NPSV). Differences in levels of sleep disturbance among men and women in resource-limited settings who survive disasters as well as NPSV are understudied. In a larger study in which we investigated trauma

symptoms among 2010 Haiti earthquake survivors via the Traumasymptom checklist -40 (N=526; 290 males, 236 females), we also assessed self-reported frequency of sleep disturbance symptoms using the measure's sleep disturbance subscale, comparing the latter by NPSV victim status and by gender.

Methods: SAS enabled 3-Class Latent Class Analysis (LCA): Class 1 ("No symptoms"), Class 2 ("Some symptoms"), Class 3 ("All Symptoms)".

Results: Distribution of class membership differed by gender $(\chi 2 = 23.9, df = 2, p < .0001)$. Proportions of respondents assigned to the three classes differed between genders (Females: Class 1, 29.2%; Class 2, 35.5%; Class 3, 35.3%); Males: Class 1, 25.7%; Class 2, 54.4%; Class 3, 19.9%). NPSV status influenced levels of sleep disturbance symptoms, and membership distribution differed across classes by gender ($\chi 2 = 23.9$, df = 2, p < .0001). Class 2 membership was greater for men (65.9%), but class 3 membership was greater for women (59.3%). Women who experienced NPSV were statistically more likely members of sleep disturbance symptom classes (Class 2 or Class 3) than Class 1 ($\chi 2$ = 14.9, df = 2, p = 0.0006). No difference was found in Class membership for men reporting NPSV ($\chi 2= 1.6$, df = 2, p = 0.45).

Conclusion: Investigating the sleep health of Cité Soleil residents adds to the body of literature on sleep health, sleep equity and gendered vulnerability. Findings suggest women in post-disaster settings, especially in LMICs, are at even greater risk for a variety of adverse health outcomes and for suboptimal sleep, even when local men have similar traumatogenic experiences, such as NPSV. Girls and women in post-disaster LMIC settings need trauma-informed sleep health promotion and NPSV-prevention.

Support: N/A

1065

A BOUT OF SLEEP APNEA OR A POSTTRAUMA NIGHTMARE OCCURRENCE?

Youngren, W. A.¹ Miller, K.²

¹The University of Kansas, Lawrence, KS, ²Veterans Affairs, Philadelphia, PA.

Introduction: The enigmatic nature of Posttrauma Nightmares (PTNs) has left research without an agreed upon operational definition. This is partially due to PTNs often containing well remembered content that is similar to the triggering trauma, but also manifesting as severe nighttime awakenings without a concise or remembered dream narrative. Given that recent research has linked episodes of Obstructive Sleep Apnea (OSA) to PTNs, this study aimed to examine if OSA could explain why some distressed awakenings occur without memory of nightmare content.

Methods: Participants included 36 trauma survivors who reported experiencing PTNs, recruited from a clinical referral or at a Veterans Affairs Hospital. Presence of OSA was captured from self-reports of previous polysomnography-based sleep study results. PTNs were measured via a self-report measure that assessed past month nightmare frequency and if the content was remembered upon awakening. Analysis included descriptive statistics and chi-square tests.

Results: Out of the group with a reported diagnosis of OSA (N = 8), 75% (n = 6) reported they did not remember the content of their nightmares upon awakening, whereas out of the group without a reported OSA diagnosis (N = 28), only 4% of participants (n = 1) reported not remembering the content of their nightmares. There was a significant difference between OSA diagnosis and remembering nightmare content ($X^2 = 57.83, p < 0.001$).

Conclusion: Individuals with diagnosed OSA commonly experienced nightmares that were often not remembered upon awakening, while the group without OSA most often remembered the content of their nightmares. Due to this relationship, it is possible that some PTNs experienced by the OSA group may instead be misinterpreted respiratory events. Understanding the relationship between OSA and PTNs is crucial for developing the most effective treatment course. **Support:** None.

1066

RAPID EYE MOVEMENT (REM) SLEEP DURATION IN POSTTRAUMATIC STRESS DISORDER (PTSD) VARIES DYNAMICALLY AND DIRECTLY WITHIN THE INDIVIDUAL WITH INDICES OF SLEEP CONSOLIDATION AND PARASYMPATHETIC TONE *Gupta, M. A.*

Schulich School of Medicine and Dentistry, Department of Psychiatry, University of Western Ontario, London, ON, CANADA.

Introduction: Autonomic arousal in posttraumatic stress disorder (PTSD) has been associated with functional hypoactivation of the medial prefrontal cortex and hyperactivity of the amygdala which can directly affect sleep physiology including REM sleep. REM sleep has been associated with reduced fear conditioning; and PTSD has been associated with REM sleep fragmentation. A case report of a drug-free PTSD patient (Gupta MA,2019) who underwent 10 home sleep apnea tests (HSATs) observed a dynamic and inverse relation between REM sleep fragmentation. This study has examined the relationship between REM sleep duration and sleep parameters related to sleep consolidation and parasympathetic tone in 17 PTSD patients who had completed at least 10 HSATs each.

Methods: 17 civilian PTSD patients (all female; mean±SD age: 47.59±10.52 years; 16 white) each completed 10 HSATs (WatchPAT200, Itamar)(over 1 to 45 months). The mean±SD initial PTSD Checklist for DSM-5 score was 49.24±13.08 (n=17), and Clinician Administered PTSD Scale for DSM-5 (CAPS-5) score was \geq 55. Patients using benzodiazepines and/or narcotics were excluded. **Results:** The overall mean±SD REM duration for all 10 visits (for 17x10 HSATs) was 84.40±8.65 minutes (range 69.13-96.97 min); the mean REM duration over the 10 HSATs correlated with other sleep indices as follows: sleep onset latency (Pearson r= -0.667, p=0.035); sleep efficiency (r=0.636, p=0.048); light sleep (NI+N2) percentage (r= -0.754, p=0.012); light sleep duration (r=0.692, p=0.027); deep sleep (N3) duration (r=0.635, p=0.048).

Conclusion: Over the 10 HSATs the average (n=17) REM sleep duration was directly related to indices of sleep consolidation (decreased sleep latency, increased sleep efficiency, increase in both light and deep sleep duration). The direct relation of REM sleep duration to duration of deep sleep, and inverse relation with light sleep percentage suggests REM sleep- related promotion of increased parasympathetic tone within the individual.

Support: None

1067

REPLICATIVE AND NON-REPLICATIVE NIGHTMARES IN THE DEVELOPMENT OF POSTTRAUMATIC STRESS DISORDER

Maeder, T.¹ Daffre, C.² Oliver, K. I.² Lasko, N. B.² Seo, J.² Ulsa, C.² Kleim, B.¹ Pace-Schott, E. F.²

¹University of Zurich, Zurich, SWITZERLAND, ²Massachusetts General Hospital, Harvard Medical School, Boston, MA.

Introduction: Nightmares are a frequent and disturbing symptom of posttraumatic stress disorder (PTSD). They are associated with sleep disruption and increased psychopathology. There is growing evidence that different types of nightmares may differ in their effects on psychopathology. Previous findings suggest that nightmares that are close replications of the experienced traumatic event might be especially important in the development of PTSD. This study investigated trauma-related (replicative) and non-trauma-related (non-replicative) nightmares as predictors of PTSD in a civilian sample.

Methods: Participants were recruited from the general public of the greater Boston area. The sample consisted of 108 participants who had experienced a psychological trauma in the past 2 years (e.g. sexual or physical assaults and accidents). The criteria for PTSD were met by 49% of participants. PTSD diagnosis was assessed using the Structured Clinical Interview for DSM-IV-TR Axis I Disorders-Non-Patient Edition. Participants received an Actiwatch 2 (Philips Respironics, Bend, OR) and a sleep diary for sleep measurements over an average of 14 consecutive nights. The diary included a prospective nightmare assessment and an item assessing the relatedness of each nightmare to traumatic events. Logistic regression analyses were performed with PTSD as the categorical outcome variable.

Results: Our analyses showed that replicative nightmares were the only statistically significant predictor of PTSD (OR = 1.2, p = .027), while controlling for age, sex, time since the traumatic event, and actigraphy total sleep time and minutes awake after sleep onset. All of these variables, including non-replicative nightmares, did not significantly predict PTSD in our analyses.

Conclusion: This study confirms and adds to the existing knowledge of nightmares and the importance of the degree to which they replicate the trauma in the development of PTSD. These findings underline the potential role of specific nightmare treatments after traumatic events, with a special focus on replicative nightmares. **Support:** R01MH109638

1068

USING SLEEP TO AVOID INPATIENT PTSD TREATMENT

Woodward, S. H.¹ Jamison, A. L.¹ Souter, T.² Shin, H. J.² Loew, D. E.² Armontrout, J. A.²

¹National Center for PTSD, Palo Alto, CA, ²VA Palo Alto Health Care System, Palo Alto, CA.

Introduction: During inpatient psychiatric treatment, mattress actigraphy can be used to track nightly sleep in a zero-burden fashion to examine its relationships with other markers of treatment progress. We report here on associations between actigraphic sleep parameters and treatment markers in combat veteran engaged in inpatient treatment for posttraumatic stress disorder. We focus first on sleep scheduling, the best-validated outcome available from actigraphic data.

Methods: 140 combat veteran inpatients provided between 5 and 164 nights of mattress actigraphy over the course of their inpatient psychiatric hospitalizations. The sample was characterized by multiple traumas and a high prevalence of comorbid mood and substance use disorders. Approximately one-half of the sample had undergone objective screening for OSA and for this subsample AHI < 20 was used as an inclusion criterion. Unscreened participants were required to have a BMI < 35. Inclusion also required

mean actigraphic SE > 60%. The PTSD Checklist and Combat Exposure Scale were obtained at admission, from which a trauma load index was created by adding z-scores obtained from standardizing these scales.

Results: In a multiple regression analysis, after accounting for secular change in LOS, higher trauma loads (p < 0.001) and earlier bed times (p = 0.001) were both significantly associated with shorter lengths of stay (LOS; F(3,137) = 10.4, p < 0.001; adj. R-squared = 0.17). Trauma load and bed time did not interact (p = 0.7).

Conclusion: Avoidance is a criterial symptom of PTSD. Early bed times and early exits from treatment may both reflect the impact of syndromal avoidance on treatment, limiting its dosage and benefit. If so, prescribing later bed times may have utility in inpatient treatment programming.

Support: Award W81XWH-15-2-0005 from the Department of Defense, Military Operational Medicine Research Program and the Department of Veterans Affairs. This material is the result of work supported with resources and the use of facilities at the Palo Alto VA Medical Center.

1069

POSTTRAUMATIC STRESS DISORDER IS ASSOCIATED WITH POORER SLEEP SPECIFIC QUALITY OF LIFE, BUT NOT WITH SLEEP APNEA

Holty, J. C.^{1,2} Pandey, A.¹ Ho, J. Q.³

¹VAPAHCS, Palo Alto, CA, ²Stanford University School of Medicine, Palo Alto, CA, ³Albert Einstein College of Medicine, Bronx, NY.

Introduction: The degree that posttraumatic stress disorder (PTSD) contributes to obstructive sleep apnea (OSA) or sleep specific quality of life (QOL) remains uncertain.

Methods: We evaluated consecutive military veterans (n=3,155) with suspected OSA using multivariable regressions to test associations between sleep and QOL measures including the apneahypopnea index (AHI), Pittsburgh Sleep Quality Index (PSQI) and Short-Form-12 mental component score (MCS). A mental health expert determined PTSD presence with severity measured utilizing the PTSD Checklist (PCL). Subjects were evaluated with prospectively collected questionnaires, sleep studies, and detailed electronic medical record reviews.

Results: Current-PTSD (n=1,172, 37%) were younger, more likely single, unemployed on disability, report non-white ethnicity or have current alcohol or drug dependence, report past suicide attempt, have current insomnia, restless sleep or nightmares, report lower MCS or higher Epworth Sleepiness, Fatigue Severity, Beck Depression Inventory-II (BDI) and PSQI scores than non-PTSD (n=1,880) or past-PTSD (n=103) veterans. Among current-PTSD, instigating trauma was 75% combat and 13% sexually related. In multivariable regressions, male gender (OR 4.5, p<0.001), age >65 years (OR 2.3, p<0.001), BMI $\ge 35 \text{ kg/m}^2$ (OR 3.5, p<0.001), prior stroke (OR 1.8, p<0.006), current hypertension (OR 1.4, p<0.001), neck circumference >40 cm (OR 1.3, p=0.032), and non-white ethnicity (OR 1.2, p=0.034) were associated with moderate-severe OSA (AHI \geq 15/h), however current (OR 0.9, p=0.06) or past-PTSD (OR 1.2, p=0.41) were not. PCL (p=0.937) was not associated with AHI. Factors most associated with lower MCS included current-PTSD (scaled standardized beta[SSB]=0.09, p=0.001), depression (SSB=0.09, p=0.001), age <50 years (SSB=0.09, p<0.001), non-white ethnicity (SSB=0.07, p=0.004), female gender (SSB=0.06, p=0.007) or single/no-partner (SSB=0.05, p=0.03). Likewise, factors most

associated with a higher PSQI included depression (SSB=0.19, p<0.001), current-PTSD (SSB=0.15, p<0.001), unemployed on disability (SSB=0.14, p<0.001), non-white ethnicity (SSB=0.13, p<0.001) or age <50 years (SSB=0.10, p=0.001). Among current-PTSD, higher PSQI was associated with BDI \geq 20 (SSB=0.31, p<0.001), PCL \geq 50 (SSB=0.24, p<0.001) and non-white ethnicity (SSB=0.11, p=0.034), but not with moderate-severe OSA (SSB=-0.09, p=0.095).

Conclusion: In the largest PTSD sleep cohort to date, PTSD is associated with insomnia, restless sleep, poorer sleep specific QOL, and greater daytime sleepiness and fatigue, but is not associated with OSA.

Support: None

1070

AN EXAMINATION OF THE RELATIONSHIP BETWEEN LANGUAGE USE IN POST-TRAUMA NIGHTMARES AND PSYCHOLOGICAL SEQUELAE IN A TREATMENT SEEKING POPULATION

Paquet, C. Davis, J. University of Tulsa, Tulsa, OK.

Introduction: Studying language use in dreams and nightmares has become an increasingly used tool to understand underlying emotional and cognitive processes. Specifically, in regards to post-trauma nightmares (PTNMs), nightmare transcriptions can offer a lens to understand a survivor's interpretation of their trauma. The current study will utilize a method of quantitative text analysis to analyze the relationship between specific psychological constructs and symptoms of posttraumatic stress disorder (PTSD) and nightmare qualities. It is hypothesized that there will be a positive correlation between words related to perceptual processes and negative emotions in nightmares and PTSD symptom and nightmare severity. There will be a negative correlation between cognitive processes and positive emotion words, and PTSD symptom and nightmare severity.

Methods: Fifty-three nightmares were collected from participants that were recruited from the community in a Midwestern city as part of an ongoing investigation of the effectiveness of a brief cognitive-behavioral intervention for PTNM, Exposure, Relaxation, and Rescripting Therapy (ERRT). All participants were over the age of 18, have experienced a criterion A trauma, and have nightmares at least once weekly. Linguistic Inquiry and Word Count (LIWC) was utilized to analyze the nightmare transcriptions. The Posttraumatic Stress Disorder Checklist for the DSM-5 (PCL-5) and the Trauma-related Nightmare Survey (TRNS) were used to measure symptom severity. A Pearson's correlation analysis was used for this exploratory study.

Results: Words related to perceptual processes were significantly positively correlated with PTSD symptom and nightmare severity (p<.05) Neither negative nor positive emotion words were significantly related to PTSD and nightmare symptoms (p>.05). Cognitive processing words were significantly negatively correlated with PTSD and nightmare symptoms (p<.05).

Conclusion: The results of this study support the hypothesis that language use in nightmares reveals important information about underlying cognitive and emotional functioning. The results of this study may have an important impact on treatment considerations for those who have experienced trauma. Analyzing language use in PTNM may help to understand the etiology and maintenance of PTSD symptoms.

Support: Support for this study comes from the University of Tulsa Institute of Trauma, Adversity, and Injustice.

1071

SUBJECTIVE MEASURES OF HYPERAROUSAL PREDICT SUBJECTIVE LONGITUDINAL AND RETROSPECTIVE MEASURES OF SLEEP QUALITY IN INDIVIDUALS EXPOSED TO TRAUMA

Kram Mendelsohn, A. Daffre, C. Oliver, K. I. Seo, J. Lasko, N. B. Pace-Schott, E. F.

Massachusetts General Hospital, Charlestown, MA.

Introduction: Hyperarousal and disturbed sleep are intrinsic symptoms of posttraumatic stress disorder (PTSD). We explored whether self-reported indices of hyperarousal predict longitudinally measured objective, subjective, and retrospective evaluations of sleep quality in trauma-exposed individuals.

Methods: Individuals exposed to a DSM-5 PTSD Criterion-A traumatic event within the past two years (N=130, 91 females), aged 18-40 (mean 24.43, SD 5.30), 51.54% of whom met DSM-5 criteria for PTSD, completed 14 days of actigraphy and sleep diaries. Participants also completed the PTSD Checklist for DSM-5 (PCL-5), the Clinician-Administered PTSD Scale (CAPS-5), published Hyperarousal (HAS) and Hypervigilance (HVQ) scales, and the Pittsburgh Sleep Quality Index (PSQI) (N=108-125 for different scales). Mean total sleep time (TST), sleep onset latency (SOL), sleep efficiency (SE) and sleep midpoint were calculated from actigraphy and subjective SOL, SE, number of awakenings, and time spent awake from diaries. Simple regressions were used to predict associations of the PCL-5, HAS, and HVQ scores with measures of sleep quality.

Results: Hyperarousal indices predicted diary but not actigraphy measures of sleep quality. Longer diary-reported SOL was predicted by higher scores for: PCL-5 total score (R=0.290, p=0.001), PCL-5 hyperarousal items without the sleep item (R=0.261, p=0.004), and HAS without sleep items (R=0.220, p=0.016). Diary-reported number of awakenings and wake time after sleep onset were predicted by higher HAS scores without the sleep question: (R=0.373, p<0.001; r=0.352, p<0.001). Similarly, all hyperarousal indices significantly predicted PSQI global score (PCL-5: R=0.482, p<0.001; PCL-5 hyperarousal: R=0.389, p<0.001; HVQ: R=0.214, p=0.017; HAS without sleep question: R=0.415, p<0.001).

Conclusion: Self-reported hyperarousal measures predict subjective longitudinal (especially SOL) and retrospective measures, but not objective measurements of sleep quality. Similar discrepancies between self-reported and objective measures of sleep quality have been reported in patients with insomnia disorder. Cognitive-behavioral therapy for insomnia may be especially effective in treating post-traumatic sleep disturbances. **Support:** R01MH109638

1072

POST-TRAUMATIC STRESS DISORDER IS ASSOCIATED WITH INCREASED OSCILLATORY FREQUENCY OF SLEEP SPINDLES

Wang, C.^{1,2} Laxminarayan, S.^{1,2} Ramakrishnan, S.^{1,2} Dovzhenok, A.^{1,2} Cashmere, D.³ Germain, A.³ Reifman, J.¹ ¹DoD Biotechnology High Performance Computing Software Applications Institute, Fort Detrick, MD, ²The Henry M. Jackson Foundation for the Advancement of Military Medicine, Inc., Bethesda, MD, ³Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, PA.

Introduction: Patients with post-traumatic stress disorder (PTSD) often suffer from sleep disturbances. Sleep spindles are an

electrophysiological hallmark of non-rapid eye movement sleep and believed to be involved in sleep protection and sleep-dependent memory consolidation. Here, we sought to examine whether sleep spindles are altered in PTSD and whether the findings are reproducible across nights and subsamples of the study population.

Methods: We obtained 64-channel electroencephalogram (EEG) recordings from 78 combat-exposed Veteran men with (n = 31) and without (n = 47) PTSD during two consecutive nights of sleep. We identified slow (10-13 Hz) and fast (13-16 Hz) sleep spindles during N2 and N3 sleep using an automatic algorithm and performed topographical analyses of spindle parameters (amplitude, duration, oscillatory frequency, and density) on both nights. To assess reproducibility, we used the first 47 consecutive participants (18 with PTSD) for initial discovery and the remaining 31 participants (13 with PTSD) for replication assessment.

Results: In the discovery analysis, compared to participants without PTSD, those with PTSD exhibited 1) increased oscillatory frequency of slow spindles over the antero-frontal regions on both nights (Night 1: p = .020, Cohen's d = 0.92; Night 2: p = .014, Cohen's d = 1.07) and 2) increased oscillatory frequency of fast spindles over the centro-parietal regions on the second night (p = .018, Cohen's d = 0.76). Notably, these trends were preserved in the replication analysis. In contrast, we found no significant group differences in the amplitude, duration, or density of slow or fast spindles.

Conclusion: The elevated sleep-spindle frequency in PTSD may reflect reduced thalamocortical inhibition and, hence, deficient sleep protection. Our findings provide the basis for an initial understanding of sleep-spindle abnormalities in PTSD. The findings, if independently validated, may assist in the development of sleep-focused diagnostics and interventions for PTSD.

Support: This work was sponsored by U.S. Defense Health Program (grant No. W81XWH-14-2-0145) and managed by the U.S. Army Military Operational Medicine Program Area Directorate, Ft. Detrick, MD. The study was also supported by the Clinical and Translational Science Institute at the University of Pittsburgh (UL1 TR001857).

1073

SLEEP DISORDERS CONTRIBUTE TO ANGER IN SERVICE MEMBERS WITH POSTTRAUMATIC STRESS DISORDER

*Miles, S. R.*¹ *Pruiksma, K.*² *Slavish, D.*³ *Nakase-Richardson, R.*¹ *Nicholson, K.*⁴ *Wardle, S.*⁵ *Young-McCaughan, S.*² *Resick, P.*⁶ *Williamson, D.*⁶ *Dondanville, K.*² *Litz, B.*⁷ *Mintz, J.*² *Keane, T.*⁷ *Peterson, A.*² *Taylor, D.*⁵

¹James A. Haley Veterans' Hospital, Tampa, FL, ²University of Texas Health Science Center at San Antonio, San Antonio, TX, ³University of North Texas, 76203, TX, ⁴Carl D. Darnall Army Medical Center, Fort Hood, TX, ⁵The University of Arizona, Tucson, AZ, ⁶Duke University, Durham, NC, ⁷VA Boston Healthcare System, Boston, MA.

Introduction: The emotion of anger and behavioral acts of aggression can lead to severe negative consequences, including family violence, legal charges, and death. Anger can be a symptom of posttraumatic stress disorder (PTSD), particularly in service members. Service members report difficulties managing their anger and trouble with the subsequent results. Factors that differentiate service members with PTSD who have anger related problems from those who do not are still unknown. Impaired sleep is associated with negative mood states in the general population and may be

a risk factor for anger in those with PTSD. This project examines how sleep disorders commonly diagnosed in service members (i.e., obstructive sleep apnea and insomnia) relate to PTSD and anger.

Methods: Ninety-three service members with comorbid PTSD, insomnia, and nightmares (mean age = 35.86 years, SD = 8.38, 27%female, 45% white) completed polysomnography and other measures as part of a clinical trial. A multiple regression model examined how total Apnea Hypopnea Index (AHI), AHI during REM sleep, insomnia (Insomnia Severity Index), age, and race related to PTSD symptoms (Clinical Administered PTSD Scale-5: CAPS-5). A second multiple regression model examined the same variables' associations with anger (Dimensions of Anger Reactions-5; DAR-5).

Results: More than a third of the sample (37%) met criteria for OSA (AHI scores \geq 5) and 99% met criteria for insomnia (ISI \geq 10). Total AHI and REM AHI were not associated with CAPS-5 scores or ISI. Across OSA and PSG indices, only greater REM AHI (b=.07, p<.05) and Insomnia (b=.43, p<.05) were positively associated with DAR-5 anger scores. Total AHI was unrelated to anger. **Conclusion:** Elevated REM AHI and insomnia were associated with greater anger in service members with PTSD. Current treatments for anger are only moderately effective. Assessing and treating comorbid sleep disorders may reduce anger and enhance successful PTSD treatment.

Support: Consortium to Alleviate PTSD (W81XWH-13-2-0065), DVA (I01CX001136-01), GDHS (W91YTZ-13-C-0015) for DVBIC.

1074

FEAR REACTIVITY AND SLEEP IN TRAUMA-EXPOSED MALES AND FEMALES WITH AND WITHOUT PTSD

Dukes, C.¹ Inslicht, S.² Hubachek, S. Q.³ Straus, L.² Ruoff, L.¹ Huie, R.² Felmingham, K.⁴ Woodward, S.⁵ Neylan, T. C.² Richards, A.²

¹San Francisco VA Medical Center, San Francisco, CA, ²UCSF Department of Psychiatry, San Francisco, CA, ³University of Maryland, College Park, MD, ⁴University of Melbourne, Melbourne, AUSTRALIA, ⁵National Center for PTSD, Menlo Park, CA.

Introduction: Sleep disturbance is considered central to mechanisms of PTSD development and maintenance, and fear learning protocols have been used as laboratory models to understand PTSD disease mechanisms. Some research indicates that fear learning may influence subsequent sleep, especially REM sleep, and that sleep may influence subsequent extinction. In this study, we examined the relationship of startle reactivity during conditioning and later extinction with objectively-measured sleep in PTSD-positive and negative subjects.

Methods: These analyses were performed as part of a larger study of PTSD and sleep. Thirty-four (34) trauma-exposed male and female participants with and without PTSD completed a fearpotentiated startle conditioning procedure at 9:45am, followed by a PSG-monitored nap (13:30-15:30), followed by an extinction protocol at 4:30pm. All visits were preceded by an adaptation nap visit at least 7 days prior. Eye-blink EMG was used to measure startle reactivity. Mixed-model analyses were performed in SPSS.

Results: PTSD-positive subjects had higher REM sleep duration (p<.05) and a trend towards shorter REM sleep latency (p=.06). There were no other group or sex effects on standard PSG sleep parameters. During conditioning, PTSD-positive status was associated with higher startle reactivity across stimulus types (p<.01),

driven by increased reactivity in PTSD-positive vs. PTSD-negative females. A PTSD x sex interaction effect on startle reactivity showed the opposite effect in males (p<.01). Higher startle reactivity during conditioning predicted longer sleep latency during the subsequent nap (p<.05), but reactivity during conditioning and extinction did not otherwise show a relationship to standard PSG sleep measures.

Conclusion: These findings are consistent with previous research indicating REM sleep abnormalities as well as heightened fear responses in PTSD. While the observed relationship between higher startle and longer sleep latency is consistent with studies indicating that stress affects subsequent sleep, further research in larger samples is needed to understand causal mechanisms and to advance our understanding of sleep-PTSD mechanisms.

Support: VA Career Development Award-5IK2CX000871-05

1075

SLEEP-STAGE INDEPENDENT ELECTROENCEPHALOGRAPHY FEATURES FOR CLASSIFICATION OF VETERANS WITH POST-TRAUMATIC STRESS DISORDER

Laxminarayan, S.^{1,2} Wang, C.^{1,2} Oyama, T.^{1,2} Cashmere, D.³ Germain, A.³ Reifman, J.¹

¹DoD Biotechnology High Performance Computing Software Applications Institute, Fort Detrick, MD, ²Henry M. Jackson Foundation for the Advancement of Military Medicine, Inc., Bethesda, MD, ³Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, PA.

Introduction: Prior sleep studies have suggested that electroencephalography (EEG) spectral power and synchrony features in certain sleep stages differ significantly at the group-average level between subjects with and without post-traumatic stress disorder (PTSD). Here, we investigated whether a multivariate combination of sleep-stage independent EEG features could objectively identify individual subjects with PTSD.

Methods: We analyzed EEG data recorded from 78 combatexposed veteran men with (n = 31) and without (n = 47) PTSD during two consecutive nights of sleep. For each subject we computed 780 features from 10 EEG channels covering the whole brain, by averaging the values over the entire night regardless of sleep stage. Using a training set consisting of the first 47 consecutive subjects (18 with PTSD) of the study, we performed univariate feature selection and backward feature elimination using a logistic regression model. We then evaluated the model on the test set, which consisted of the remaining 31 subjects (13 with PTSD). We assessed model performance by computing the area under the receiver operating characteristic curve (AUC).

Results: Feature elimination using the logistic regression model yielded three uncorrelated features that were consistently discriminative of PTSD across the two consecutive nights. When we trained the logistic model consisting of these three features using data from both nights of the training set, the model yielded test-set AUCs of 0.84 and 0.80 for Night 1 and Night 2, respectively. These values were considerably larger than the test-set AUCs of the three individual features, which ranged from 0.55 to 0.74 across both nights.

Conclusion: We identified robust, stage-independent, whole-night features and combined them in a logistic regression model to discriminate subjects with and without PTSD. The model yielded AUCs above 0.80 on the test data, showing promise as an objective approach to diagnose PTSD at the individual level.

Support: This work was sponsored by U.S. Defense Health Program (grant No. W81XWH-14-2-0145) and managed by the U.S. Army Military Operational Medicine Program Area Directorate, Ft. Detrick, MD. The study was also supported by the Clinical and Translational Science Institute at the University of Pittsburgh (UL1 TR001857).

1076

SELF-REFERENTIAL LANGUAGE IN TRAUMA NARRATIVES PREDICTS SHORTER SLEEP DURATION IN WOMEN WITH PTSD

Bullock, A. Burns, A. Taylor, E. Grandner, M. Miller, M. Alkozei, A. Killgore, W. University of Arizona, Tucson, AZ.

Introduction: The use of self-referential language, defined as firstperson singular pronouns (e.g. I, me, my), in trauma narratives has been found to predict post-traumatic stress disorder (PTSD) severity. Additionally, taking a self-immersed perspective correlates with higher blood pressure reactivity than a self-distanced perspective. Given this relationship between self-immersed perspectives and physiological processes, we investigated the relationship between self-referential language and sleep in people with PTSD, as dysfunctional sleep is a major treatment target in this disorder. Methods: Seventy-five participants (49 females; $M_{are}=31.8$, SD_==8.8) meeting DSM-5 criteria for PTSD were administered the PTSD Checklist for the DSM-5 (PCL-5) and the Pittsburg Sleep Quality Index (PSQI). Sleep duration was assessed with the PSOI. Participants provided typed descriptions of their traumatic event, which were then analyzed using the Linguistic Inquiry and Word Count 2015 software to count instances of first-person singular pronouns ("I" words). Linear regression, with PCL-5 scores and "I" words entered stepwise, was used to predict scores on the PSQI sleep duration subscale. Use of "I" words between the sexes

Results: For females but not males, PTSD severity significantly predicted sleep duration (R^2 =.207, p=.001). Additionally, the number of "I" words in the trauma narratives predicted an additional 8% of the variance in sleep duration for females (R^2 change=.083,

 β =.288, *p*=.029) but not males. Females used significantly more self-referential language in their narratives (*M*=11.84, *SD*=8.42) compared to males (*M*=5.25, *SD*=6.10, *p*=.001).

Conclusion: After controlling for PTSD severity, self-referential language in trauma narratives significantly predicted shorter sleep duration in females. While speculative, this finding suggests that treatment approaches for PTSD may benefit from a focus on targeting self-referential processes to improve sleep and PTSD in females but not males. As dysfunctional sleep is a hallmark of PTSD, further investigation into this relationship may illuminate a new treatment avenue for this disorder.

Support: W81XWH-14-1-0570

1077

THE ASSOCIATION BETWEEN SLEEP PROBLEMS AND RISK-TAKING BEHAVIOR DIFFERS BETWEEN RACIAL MAJORITY AND MINORITY GROUPS

Burns, A. I. Bullock, A. Taylor, E. Grandner, M. A. Alkozei, A. Killgore, W. D. University of Arizona, Tucson, AZ.

Introduction: Individuals with Post-Traumatic Stress Disorder (PTSD) often experience poor sleep quality and elevated

self-destructive behaviors. Among healthy individuals, poor sleep quality can lead to increased risk-taking behavior through decreased inhibition and/or increased willingness to take risks. However, it is unclear whether racial/ethnic background may influence this relationship, in particular among individuals with PTSD. We examined whether the relationship between sleep quality and risk propensity would differ between majority and minority racial groups in individuals with PTSD.

Methods: Seventy-six individuals (61.8% female; mean age=31.7, SD=8.8) with a clinical diagnosis of PTSD were administered the Functional Outcomes of Sleep Questionnaire (FOSQ) as a measure of sleep-related functional impairment of daily activities, and the Evaluation of Risk (EVAR) Scale as a measure of risk-taking propensity. Forty-seven individuals identified with the majority racial group (Caucasian) and 29 individuals identified themselves within the minority.

Results: There were no significant group differences for FOSQ and total EVAR risk-taking scores. However, the strength of association between measures differed significantly between groups (Z=1.95, p=.051). For the racial/ethnic majority, functional impairments due to lack of sleep were positively associated with risk-taking propensity (r=.460, p=.001); this relationship was not present for the minority group (r=.016, p=.936).

Conclusion: Self-reported functional impairments due to sleep loss significantly correlated with risk-taking propensity for those who identified themselves as part of the majority racial group but not for individuals who identified as part of a racial minority. Findings suggest that broad conclusions regarding the association between sleep disruption and risk-taking may not apply equally across racial/ethnic groups and such factors should be considered when evaluating studies of sleep and risk behaviors. Whether these differing effects are due to cultural factors or stable differences in biology is not known and will require additional research. **Support:** W81XWH-14-1-0570

1078

SLEEP PROBLEMS IN POSTTRAUMATIC STRESS DISORDER (PTSD) IN A NATIONALLY REPRESENTATIVE US SAMPLE BEFORE AND AFTER CONTROLLING FOR COMORBID DEPRESSION: RESULTS FROM A NATIONALLY REPRESENTATIVE US SAMPLE

Gupta, M. A. Vujcic, B.

Department of Psychiatry, Schulich School of Medicine and Dentistry, University of Western Ontario, London, ON, CANADA.

Introduction: The impact of psychiatric comorbidities on sleep disturbances in PTSD have been studied in the National Comorbidity Survey (NCS)(Leskin GA, 2002) and the NCS-replication (Lauterbach D, 2011) studies. We examined sleep problems in PTSD before and after controlling for comorbid depressive disease, in the National Ambulatory Medical Care Survey(NAMCS) and National Hospital Ambulatory Medical Care Survey (NHAMCS) which use a multi-stage probability design to collect nationally representative data on health care visits.

Methods: We examined patient visits(1995-2015) from NAMCS/ NHAMCS with a PTSD diagnosis (ICD-9-CM 309.81). Both NAMCS/NHAMCS allow \geq 3 reasons for visit(RFV) and \geq 3 physician-assigned diagnoses (using ICD9-CM codes). The following variables were created: 'Insomnia': ICD9-CM codes 307.41,307.42,780.51,RFV 11351; 'Sleep Disturbance'(SD): ICD-9CM codes 780.5, 780.50, 780.59; 'Nightmares': ICD9-CM code 307.4, RFV 11353; Obstructive sleep apnea (OSA): ICD-CM codes 327.23,780.57, RFV 11355, checklist; and 'Depression': ICD9-CM codes 296.2, 296.3, 296.82, 311, 296.20-296.36, 300.4.

Results: There were an estimated 37,262,245±3,203,047 (unweighted count or UWC=3,995; 66.4%±1.8% female;.mean±age: 40.39 ± 0.56 years; 'Depression' was comorbid with $37.7\% \pm 1.5\%$ cases) PTSD patients visits. All sleep variables accounted for 11.2%±1.2% (UWC=303) of PTSD visits with their individual frequencies as follows: 'Insomnia'6.5%±1.1%(UWC=153); 'Nightmares':1.9%±0.4%(UWC=74);'SD':2.0%±0.3%(UWC=67); OSA: 1.3%±0.3%(UWC=27). Logistic regression analysis using PTSD versus all other patient visits as dependent variable revealed the following sleep predictors of PTSD after controlling for age and sex and: (i) before controlling for 'Depression': 'Insomnia': OR=7.16, (95%CI 4.78-10.73); 'SD': OR=4.43(95%CI 'Nightmares':OR=104.29 2.55-7.71): (95%CI56.65-192.02); 'OSA': OR=1.71(95%CI 0.94-3.12); and (ii) after controlling for 'Depression': 'Insomnia': OR=2.88 (95%CI 1.87-4.42); 'SD': OR=2.84 (95%CI 1.73-4.67); and 'Nightmares': OR=58.33(95%CI 26.39-128.94); and 'OSA': OR=2.02(95%CI 1.14-3.57).

Conclusion: In a nationally representative sample, the association of PTSD with insomnia, sleep disturbance and nightmares remained significant, albeit decreased, after controlling for the confounding effect of comorbid depression; however the association of PTSD with OSA emerged only after the effect of depression was controlled for.

Support: None

1079

IS PTSD IN YOUNG WOMEN ASSOCIATED WITH REM SLEEP ABNORMALITIES?

Martinez, D.¹ Yeh, M.² Oliveira, L.¹ Coimbra, B.² Mello, A. F.² Poyares, D.¹ Tufik, S.¹ Mello, M. F.²

¹Psychobiology Department, Universidade Federal de Sao Paulo, Sao Paulo, BRAZIL, ²Psychiatry Department, Universidade Federal de Sao Paulo, Sao Paulo, BRAZIL.

Introduction: The increase in violence against young women has a high impact on the prevalence of Posttraumatic stress disorder (PTSD). The lifetime prevalence of PTSD is twice as high in women. However, most studies assessing sleep disturbances in PTSD were conducted predominantly in male samples and combat veterans. Objective: To analyze the sleep of young women with and without PTSD. Hypothesis: Women with PTSD have worse sleep quality, higher arousability, and higher muscle activity during REM sleep.

Methods: Case-controlled study with young women. Seventy-four women who suffered sexual assault and developed PTSD (DSM-5); and 64 women from the community without PTSD. Women were recruited from the PTSD outpatient clinic (Universidade Federal de São Paulo, Brazil).Clinician-Administered Posttraumatic Stress Scale (CAPS 5), Beck Depression and Anxiety Inventories (BDI) (BAI), full in-lab Polysomnography (PSG), Epworth Sleepiness Scale (ESS), Pittsburgh Sleep Quality Índex (PSQI), Fatigue Impact Scale (FIS), and Insomnia Severity Índex (ISI) were applied to all participants. Analysis of variance, regression models, and general linear modeling were used.

Results: Patients mean age was 28 vs 24 for the control group (p=0.004). CAPS mean score in PTSD-group was 42.5±9.1. BDI, BAI, FIS, PSQI, ISI scores were worse in PTSD-group (p<0.05, all). Pittsburgh Sleep Quality Index (PSQI) score was significantly associated with CAPS 5 independently of depression, fatigue, and

sleep fragmentation. The PTSD women had lower total sleep time (p=0.01) and lower REM sleep percentage (p=0.04). However, the control group had higher arousal index (p=0.01) and had higher muscle activity during REM sleep (p=0.03) than PTSD.

Conclusion: Women with PTSD had significantly worse score in PSQI, FIS, and ISI. PSQI score was associated with PTSD severity. However, when PSG results are concerned, we found higher sleep fragmentation in the control group. We speculate that women with PTSD may have felt safer and taken care of in the lab, which might explain the difference between objective and subjective measures of sleep quality in PTSD.

Support: Acknowledgments: FAPESP: Fundação de Apoio à pesquisa de São Paulo, AFIP: Associação Incentivo a Pesquisa

1080

SELF-REPORTED HYPERAROUSAL PREDICTS LOWER PARASYMPATHETIC ACTIVITY DURING SLOW-WAVE SLEEP IN TRAUMA-EXPOSED INDIVIDUALS

Ragas, T. L.¹ Oliver, K. I.¹ Daffre, C.¹ Seo, J.¹ Gannon, K.² Pace-Schott, E. F.¹

¹Massachusetts General Hospital, Charlestown, MA, ²Massachusetts General Hospital, Boston, MA.

Introduction: Hyperarousal and abnormal autonomic functioning are among the core manifestations of posttraumatic stress disorder (PTSD). In this study, we examined the association of parasympathetic activity during slow wave sleep (SWS) with self-reported hyperarousal measures in recently traumatized individuals.

Methods: Individuals exposed to a PTSD Criterion-A trauma within the past 2 years (N=76) aged 18-40 (mean 24.06, SD 4.76), of whom 43% met DSM-5 criteria for PTSD, underwent a night of ambulatory polysomnography (PSG) following an acclimation night. ECG recordings during SWS-sleep periods of at least 5 min were analyzed for 2 parasympathetic indices: Root Mean Square of the Successive Differences (RMSSD) and High Frequency (0.14-0.4Hz) power (HF power) using Kubios software. Hyperarousal indices included the hyperarousal items from the PTSD Checklist for DSM-5 (PCL-5) excluding the sleep item #20 (PCLhyp), those from the Clinician-Administered PTSD Scale (CAPS-5) including sleep items (CAPShyp), as well as a published Hyperarousal Scale (HAS) and Hypervigilance Questionnaire (HVQ). In addition, a Composite Hyperarousal Index (CHI) was computed from combined hyperarousal items on the PCL-5 and the CAPS-5 as well as the HAS total score.

Results: SWS RMSSD was negatively associated with PCLhyp (R = -.244, p = 0.035), CAPShyp (R = -.250, p = 0.03), CHI (R = -.280, p = 0.014), and HAS (R = -.229, p = 0.049). SWS HF power was negatively associated with CHI (R = -.227, p = 0.049). **Conclusion:** The hyperarousal (Criterion E) symptoms of PTSD are associated with lowered parasympathetic tone during SWS across the spectrum of posttraumatic severity from resilient individuals to those diagnosed with PTSD. **Support:** R01MH109638

1081

ASSOCIATIONS AMONG REM DENSITY AND PARASYMPATHETIC ACTIVITY, NIGHTMARES, AND HYPERAROUSAL IN TRAUMA-EXPOSED INDIVIDUALS

Oliver, K. I.¹ Hinton, J. A.¹ Daffre, C.¹ Dominguez, J.¹ Seo, J.¹ Gannon, K.² Lasko, N. B.¹ Pace-Schott, E. F.¹ ¹Massachusetts General Hospital, Charlestown, MA, ²Massachusetts General Hospital, Boston, MA.

Introduction: Individuals with posttraumatic stress disorder (PTSD) exhibit autonomic hyperarousal and nightmares. We hypothesized that REM density (REMD) and REM heart rate variability would predict self-reported hyperarousal, nightmares, and PTSD diagnosis in trauma-exposed individuals.

Methods: Ninety-nine individuals (aged 18-40, 68 females) exposed to a DSM-5 PTSD criterion-A trauma within the past two years (48 meeting PTSD criteria) completed a night of ambulatory polysomnography (PSG) preceded by an acclimation night. REMD in scored sleep recordings were computed using the Matlab program written by Benjamin Yetton. Indices of parasympathetic tone during REM were computed using Kubios software and included Average Root Mean Square of the Successive Differences (RMSSD) and High Frequency power (HFpower). Participants completed two weeks of sleep diaries with nightmare questionnaire and completed the Clinician-Administered PTSD Scale (CAPS-5) and the PTSD Checklist for DSM-5 (PCL-5). Hyperarousal-item scores were computed from the PCL-5 without the sleep item (PCLhyp) and from the CAPS-5 (CAPShyp), and these scores (with their sleep items) were combined into a Composite Hyperarousal Index (CHI). Nightmare rate was the proportion of sleep diaries reporting a nightmare. Simple regressions measured associations among REMD, REM parasympathetic indices, hyperarousal measures, and nightmare rate.

Results: REMD did not significantly predict PTSD diagnosis or hyperarousal scores but did predict decreased parasympathetic activity for both RMSSD (p=0.002, R=-0.316) and HFpower (p=0.016 R=-0.250). REMD predicted increased nightmare rate (p=0.011 R=0.262). Parasympathetic tone was negatively correlated with CAPShyp, PCLhyp, and CHI for both RMSSD (p=0.04, 0.011, <0.000, respectively) and HFpower (p=0.051, 0.021, 0.010, respectively). Lower parasympathetic tone also predicted PTSD diagnosis with both RMSSD (p=0.012, t=2.559) and HFpower (p=0.010, t=2.627), but did not predict nightmare rate.

Conclusion: REMD predicted decreased parasympathetic tone and higher nightmare rate. Parasympathetic tone, but not REMD, predicted hyperarousal and PTSD diagnosis.

Support: R01MH109638

1082

OFFSPRINGS AUTISTIC BEHAVIORS MODIFY THE RELATIONSHIPS BETWEEN MATERNAL PERITRAUMATIC DISTRESS AND SLEEP DISTURBANCE FOLLOWING TRAUMA EXPOSURE

Chanko, N.¹ Williams, N.¹ Jean-Louis, G.¹ Casimir, G.² Blanc, J.¹ ¹NYU Grossman School of Medicine, New York, NY, ²SUNY Downstate Medical Center, Brooklyn, NY.

Introduction: Neurodevelopmental impairments may alter parents' sleep and add tremendous stress to their families' routine. This study examined the relationship between peritraumatic distress, and sleep disturbances among mothers who were exposed to the 2010 Haitian earthquake during pregnancy and whether this relationship is moderated by offspring's autistic behaviors.

Methods: Sample includes 361 mother-offspring dyads [mean (SD) age= 27.31 (5.93); 3.1/2(3.88)] who survived the 2010 earthquake in Haiti. Maternal data were collected 3 years following the event via the Earthquake Experience Questionnaire (EEQ), the Peritraumatic Distress Inventory (PDI), and the sleep items of the PTSD Checklist (PCL-S) (such as trouble falling or staying asleep, and repeated disturbed dreams in response to the disaster exposure). Child-related data were obtained from maternal completion of the Echelle d'*Evaluation des Comportements Autistiques Revisee* (ECAR) (Autistic Behaviors Scale Revised). Pearson Correlations, multilinear regression and interaction effect analyses were conducted to explore the association between peritraumatic distress, offspring's autistic behaviors and sleep disturbance among the participants.

Results: 10.7% of mothers were caught under rubble or were seriously injured during the event. Three years later, 56.83% and 52.9% of them had consequent disturbed sleep and nightmares, respectively. Maternal sleep disturbance correlated positively with peritraumatic distress (r=.38, p=.01) and offspring autistic behaviors (r=.13, p=.05). As hypothesized, adjusting for covariates, peritramatic distress was the strongest predictor of maternal sleep disturbance (B=.310, p<.001). The relationship between maternal peritraumatic distress and sleep disturbance was modified by off-spring autistic behaviors (B=.138, p=.015).

Conclusion: This is the first study to document the prolonged effect of peritraumatic distress during the 2010 Haitian earthquake on mother's sleep disturbance and whether this relationship is moderated by offspring autistic behaviors. The findings support the importance of a sleep component in maternal and child health in disaster preparedness program.

Support: This study was supported by funding from the NIH: T32HL129953

1083

NIGHTMARES ARE NEGATIVELY ASSOCIATED WITH IMMEDIATE MEMORY AND VISUOSPATIAL PERFORMANCE IN INDIVIDUALS WITH PTSD

Bullock, A. Burns, A. Alkozei, A. Taylor, E. Grandner, M. Killgore, W.

University of Arizona, Tucson, AZ.

Introduction: Disturbing dreams and nightmares are common in individuals with post-traumatic stress disorder (PTSD). At present, little research has investigated the associations between nightmares and cognition in these individuals. However, a robust body of research has shown memory and attention impairments among those with PTSD. The present study sought to investigate the potential relationships between cognitive performance and nightmares in this population.

Methods: Seventy-five individuals (49 female; M_{age} =31.8, SD_{age} =8.8) were administered the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), the PTSD Checklist for the DSM-5 (PCL-5), the Insomnia Severity Index (ISI), the Functional Outcomes of Sleep Questionnaire (FOSQ), the Clinician-Administered PTSD Scale (CAPS), and the Disturbing Dreams and Nightmares Index (DDNSI). Five linear regressions were conducted with index scores on the RBANS subscales (immediate memory, visuospatial/constructional, language, attention, and delayed memory) as the dependent variables and PCL-5, ISI, FOSQ, CAPS symptom class subscales (intrusion, avoidance, cognition, and arousal), and DDNSI scores entered stepwise.

Results: A linear regression revealed that nightmares predicted 15% of the variance in RBANS immediate memory scores (R^2 change=.152, β =-.390, p=.003). A second linear regression revealed that nightmares predicted 9.6% of the variance in RBANS visual memory scores (R^2 change=.096, β =-.310, p=.019). No other independent variables added to either model. None of the independent variables predicted any variance in language, attention, or delayed memory scores.

Conclusion: Our analysis revealed a unique contribution of nightmares to immediate memory and visuospatial performance in individuals with PTSD. This finding was not better explained by variation in PTSD severity or sleep. Because sleep and dreams are implicated in memory consolidation, one explanation for our finding is that highly distressing trauma-related dreams (i.e. nightmares) may lack the same memory-improving qualities as ordinary dreams. Additionally, given that immediate memory and visuospatial functioning utilize working memory, perhaps nightmares and deficits in working memory share similar mechanisms.

Support: W81XWH-14-1-0570

1084

DEPRESSION AND STRESS GENERATION: CAN SLEEP QUALITY BRIDGE THE GAP?

Summers, C. Ciesla, J. Bean, C. Kent State University, Kent, OH.

Introduction: The stress generation literature has established a bidirectional relationship between depression and stress. Not only do stressful life events predict depressive episodes, but a depressive history is also linked to increased, future stressors. One relevant mechanism that has received little attention to account for this relationship is sleep. Sleep difficulties are intertwined with depression, both as a predictive and maintenance factor. Beyond depression, sleep disruption is also a factor in a plethora of stressful events, from an increased risk of automotive accidents to higher reports of interpersonal conflict. The present study explored the role of sleep quality to account for depression's association with stressors.

Methods: Ninety-six college students (Age: M = 19.56, SE = .20) reported on their depressive symptoms before undergoing a twoweek, online diary, where they reported on sleep quality and the number of stressors experienced. A generalized structural equation model (GSEM) was used to test the relevance of sleep quality to account for baseline depressive symptoms predicting average differences in stressors over the diary. Within the GSEM, a multilevel model was used to explore the daily, within-person association between sleep quality and the number of stressors reported.

Results: Baseline depression was predictive of poorer sleep quality (b = .01, p < .001) and more stressors across the diary (b = .02, p = .017). Sleep quality mediated the effect of depression on stress generation (b = .002, p = .036), accounting for 13% of the variance. On a daily level, poorer sleep quality the night before predicted more stressors the next day (b = .16, p = .027).

Conclusion: The results suggest that sleep quality is a relevant mechanism in the prediction of future stressors from depression. Sleep difficulties may represent a pivotal area of future research and intervention target in breaking the cycle between depression and stress generation.

Support: Hammen, C. (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology*, *100*, 555-561.

Tsuno, N., Besset, A., & Ritchie, K. (2005). Sleep and depression. *The Journal of Clinical Psychiatry*, *66*, 1254-1269.

1086

NON-LINEAR ASSOCIATIONS BETWEEN DEPRESSION AND SLEEP DURATION IN AN INTERNATIONAL SAMPLE OF 16,997 RESPONDENTS

Bender, A. M.^{1,2} Babins-Wagner, R.^{1,3} Laughton, A.¹ ¹Calgary Counselling Centre, Calgary, AB, CANADA, ²University of Calgary, Faculty of Kinesiology, Calgary, AB, CANADA, ³University of Calgary, Faculty of Social Work, Calgary, AB, CANADA.

Introduction: Abnormal sleep duration is common in people with depression and can be both a risk-factor and a symptom of depression. Here we determine the prevalence of depression likelihood and assess associations between long and short sleep duration in an international convenience sample.

Methods: N=16,997 respondents (age range: 8-98y, mean age 39.7y \pm 13.1 SD; 43% female) completed the 10-item Harvard Department of Psychiatry National Depression Screening Day Scale (HANDS) online from October 7 to October 13, 2019. Higher total scores on the HANDS indicate higher likelihood of major depressive episode with scores >8 indicate the presence of a major depressive disorder is likely. Additional questions were added to the survey including the question "During the past two weeks, how many hours of actual sleep did you average at night?" Answer choices ranged from "less than 5 hours" to "more than 10h" in half-hour increments.

Results: Respondents came from 115 different countries with the majority of respondents from Canada (48%) and the United States (38%). Sixty-four percent of the sample were recommended for further evaluation for depression. Of those recommended, 66% reported <7h of sleep per night and 3% reported >9h. Those who reported 7-9h of sleep per night had the lowest depression scores (9.2 points) compared to those who reported <7h (11.8 points) and >9h (13.7 points), F(2,15366)=434.81, p<0.001. The amount of sleep associated with the lowest depression scores (15.5 points).

Conclusion: We found a high prevalence of depression likelihood in 64% of an international convenience sample, with 69% of those not meeting the recommended 7-9h of sleep per night. Both short and long sleep were associated with higher levels of depression with 7.5h of sleep associated with the lowest depression scores. Future research on depression should focus on sleep interventions aimed at improving both short and long sleep duration. **Support:** N/A.

1087

EARLY SESSION EFFECTS OF CBT-I ON INSOMNIA AND DEPRESSION

Bishop, T. M.¹ Crean, H. F.¹ Funderburk, J. S.³ Speed, K. J.¹ Pigeon, W. R.¹

¹VA Center of Excellence for Suicide Prevention, Canandaigua, NY, ²VA Center of Excellence for Suicide Prevention,

Canandaigua, NY, ³VA Center for Integrated Healthcare, Syracuse, NY, NY.

Introduction: Cognitive behavioral therapy for insomnia (CBT-I) has been shown to reduce depressive symptomatology among patients with co-occurring insomnia and depression. Brief forms of CBT-I have been tested in various settings including primary care. As delivery formats of CBT-I broaden, it is important to enhance our understanding of what doses and what components of CBT-I, provide the optimal balance of treatment efficacy and brevity. In

the present study, we examine session-by-session effects of CBT-I on insomnia and depression.

Methods: Fifty-four Veterans with insomnia and co-occurring depression or posttraumatic stress disorder were randomized to either four sessions of CBT-I or treatment as usual in a published parent study. We report here on the effects among those who received CBT-I (n = 22). At each session participants provided a completed sleep diary and completed the Insomnia Severity Index (ISI) and Patient Health Questionnaire-9 for depression (PHQ-9).

Results: At baseline, participants endorsed a moderate level of both insomnia (ISI score = 18.5 [SD=4.2]) and depression (PHQ-9 score = 15.6 [SD=5.2]). A mean decrease of 4.0 points in ISI total score was observed between sessions 1 and 2 [t(21)=-3.88, p<.001] and a 3.3 points between sessions 2 and 3 [t(19)=-2.63, p<.05]. Mean PHQ-9 scores decreased by 2.9 points between sessions 1 and 2 [t(21)=-2.84, p<.01] and a 2.8 points between sessions 2 and 3 [t(19)=-2.77, p<.05]. In contrast, changes in ISI and PHQ-9 scores between baseline and session 1, and sessions 3 and 4 did not reach significance.

Conclusion: The majority of improvements in both insomnia and depression were observed following sessions 1 and 2 of CBT-I. Findings suggest that even a limited exposure to CBT-I may have a clinically significant impact on functioning across multiple domains. Whether such early improvements represent an optimal balance compared with the more modest additional improvements achieved by adding more sessions is discussed.

Support: This work was supported by the VISN 2 Center of Excellence for Suicide Prevention at the Canandaigua VAMC.

1088

HEART RATE VARIABILITY DURING SLEEP IN ADULTS WITH AND WITHOUT A HISTORY OF DEPRESSION AND THE INFLUENCE OF ANTIDEPRESSANT USE

Egeler, M. E.¹ Bowman, M.¹ Thayer, J. F.² Brindle, R. C.³ Hall, M.¹ Kline, C. E.¹

¹University of Pittsburgh, Pittsburgh, PA, ²University of California Irvine, Irvine, CA, ³Washington and Lee University, Lexington, VA.

Introduction: Individuals with depression have an increased risk for cardiovascular disease. While lower daytime HRV may be one mechanism of this association, it is less clear whether HRV during sleep (sHRV) differs between adults with and without depression. Examining sHRV is an important measure to test given evidence that nocturnal differences in physiology are strong predictors of cardiovascular disease.

Methods: 118 adults who completed psychiatric interviews between the ages of 21-60 y (T1) returned 18.7 ± 4.5 years later for a re-evaluation of their mental health and assessment of sleep (T2). 71 participants were diagnosed with Major Depressive Disorder (MDD) at either T1 or T2, while 47 participants were free from depression at both time points. At T2, participants underwent an overnight polysomnogram with concurrent assessment of electrocardiography. The primary measures of sHRV were root mean square of successive differences (RMSSD) values during rapid eye movement (REM) sleep and non-REM (NREM) sleep stages N2 and N3. Lower values of RMSSD suggest lower parasympathetic activity. RMSSD values were natural log-transformed prior to analysis. Differences in RMSSD between depressive groups were examined using analysis of covariance (ANCOVA), adjusting for age, body mass index, gender, race, and antihypertensive medication use. Additional ANCOVA models adjusted for antidepressant use.

Results: Participants with current or a history of MDD had lower RMSSD values during REM sleep (P=.01) and a trend toward lower values during NREM (P=.06) compared to those without MDD. Antidepressant use was significantly associated with lower RMSSD during both REM (P=.002) and NREM sleep (P<.001). Depression history was no longer associated with RMSSD during NREM or REM sleep following adjustment for antidepressant use (P>.40).

Conclusion: These data indicate that adults with MDD exhibit lower parasympathetic activity during sleep compared to those without MDD. These associations were modified by antidepressant use, suggesting that antidepressants may partially explain the association between depression and sHRV. Future studies investigating the influence of specific antidepressants for modifying nocturnal physiology may help to better understand the link between depression and cardiovascular disease risk.

Support: This study was funded by National Institutes of Health (NIH) grants R01 HL104607 and K23 HL118318.

1089

WATER INTAKE MODERATES THE RELATIONSHIP BETWEEN SLEEP QUALITY AND DEPRESSIVE SYMPTOMS: THE LATINO HEALTH AND WELL-BEING STUDY

Blanc, J.¹ Williams, N.¹ Jean-Louis, G.^{1,2} Lemon, S.² Rosal, M.² ¹New York University Langone Health, NEW YORK, NY, ²University of Massachusetts Medical School, Worcester, MA, ³University of Massachusetts Medical School, Worcester, MA.

Introduction: This study examined the relationships between sleep quality and depressive symptoms, and whether this relationship is moderated by frequency of water intake in a sample of Latino adults.

Methods: Participants in this community-based study were 574 Latino adults from Lawrence, Massachusetts. Assessments included surveys and anthropological measures. Variables in this study included sleep quality (Pittsburgh Sleep Quality Index- PSQI), depressive symptoms (Center for Epidemiologic Studies Depression Scale -CES-D) and frequency of water intake in the previous three months (investigator-developed question). Covariates included demographics, stress (Perceived Stress Scale-PSS), and body mass index (BMI). Multiple linear regression analyses were conducted to explore associations between sleep and depressive symptoms. Potential moderating effect of frequency of water intake was assessed using hierarchical, moderated, multiple regression analysis. Results: The sample was 51.2% female, with a mean age of 46.6 years (SD=15.4) and mean BMI of 29.6 (SD=5.9); 31% of the sample had CES-D scores > 22 (cut off for elevated depressive symptoms), the mean PSQI score was 13.11(SD=3.4) and 92% reported water intake two or more times daily. Sleep quality correlated positively with depression (r=.558; p=.000). After adjusting for covariates, sleep quality was strongly associated with depression (B = .417; SE=13; p = .000). The relationship between sleep quality and depressive symptoms was moderated by frequency of water intake (B= -.186, SE =1.107; *p*= 0.11).

Conclusion: This study is among the first to examine the association between sleep quality and depressive symptoms among Latino adults, and to show that frequency of water intake may moderate this association in this population.

Support: This study was supported by funding from the NIH: R01 MH085653; 1U48DP006381; and T32HL129953.

1090

SELF-REPORTED SLEEP QUALITY IS A ROBUST PREDICTOR OF DEPRESSION IN WOMEN WITH IRREGULAR MENSTRUAL CYCLES

*Meers, J. M.*¹ *Bower, J. L.*² *Alfano, C. A.*¹ ¹University of Houston, Houston, TX, ²De Montfort University, Leicester, UNITED KINGDOM, ³University of Houston, Houston, TX.

Introduction: Menstrual cycle regularity is an important marker of women's health. Abnormalities are associated with serious health complaints, e.g., infertility, cardiovascular, and metabolic disorders. Cycle irregularity is also linked to depression, anxiety and poor quality sleep. In fact, poor sleep and circadian misalignment may precede menstrual irregularity for some. This study describes sleep and affective characteristics of women with menstrual cycle irregularity compared to regularly cycling women and examined the individual contributions of sleep and menstrual regularity to affective symptoms.

Methods: N=314 (*Mage*=20.95, *SD*=2.35) women provided reports of menstrual health characteristics (frequency, duration, related symptoms), sleep over the past month (Pittsburgh Sleep Quality Index; PSQI) and on the previous night, as well as mood (Center for Epidemiologic Studies Depression Scale) and anxiety (State Trait Anxiety Inventory) symptoms.

Results: Among the 20.4% (n=64) of women who endorsed "always irregular" periods, mean cycle length (m=31.33 days, sd=8.5) was significantly greater than among women more regular periods (m= 27.93, sd=3.83, t(55.37)=-2.78, p=.007). This subgroup also reported higher PSQI scores (t(181)=-2.56, p=.011), longer SOL (t(282)=-3.00, p=.003), poorer sleep quality overall (t(299)=-2.35, p=.02), and poorer sleep quality on the previous night (t(300)=2.70, p=.007). Irregular cyclers reported significantly more depressive (t(284)=-2.18, p=.03) but not anxiety symptoms. When sleep and menstrual irregularity were entered into a hierarchical linear regression to examine their relative influence on depressive symptoms (F[2,174]=13.15, p<.001, R^2 =.13), sleep remained the only significant predictor (b= 1.13, p<.001) and menstrual cycle effects were no longer significant.

Conclusion: In line with previous studies, cycle irregularity was associated with poorer sleep quality and depressive symptoms. Sleep quality was found to be a more robust predictor of depression than menstrual cycle irregularity, suggesting that sleep may underlie the affective disturbances in women with more irregular menstrual cycles.

Support: n/a

1091

THE EFFECTS OF SLEEP DURATION, TIMING, AND DEPRESSED MOOD ON DAILY EATING PATTERNS

Wescott, D. L.¹ Dickman, K. D.¹ Franzen, P. L.² Hasler, B. P.² Roecklein, K. A.¹

¹University of Pittsburgh, Pittsburgh, PA, ²University of Pittsburgh Medical Center, Pittsburgh, PA.

Introduction: Insufficient sleep, circadian misalignment, and altered eating patterns are linked to depression. Despite the temporal association between the sleep/wake and feed/fast cycles, it is unclear how depression severity influences this relationship.

Methods: Sixty-eight participants ages 18-65 years across the depression continuum wore an Actiwatch for 5-17 nights and reported daily meal times (730 nights total). Multilevel models

tested previous night's sleep timing and duration as predictors of the length of the next day's eating window. Within-person sleep duration and timing were entered as Level 1 predictors to account for nightly variation in sleep. Between-person sleep duration and timing were entered as Level 2 predictors. A three-way interaction between depression severity and Level 2 sleep duration/timing was entered. Covariates included age, gender, and day (weeknight/ weekend).

Results: Across participants, average later sleep timing predicted a longer eating window (Β= -.222; p =.005). Earlier sleep timing (Β= -.186; p < .001) and shorter sleep duration(Β= -.103; p < .001) relative to a person's average each predicted a longer next-day eating window. A three-way interaction was found between sleep duration, timing, and depression (Β= -.159; p = .002). At shorter sleep durations, individuals with higher depression severity had a positive relationship between sleep timing and eating window length, and individuals with low depression had a negative relationship between sleep timing and eating window length.

Conclusion: At shorter sleep durations (< 6.5 hr), individuals with high depression and later sleep timing or low depression and early sleep timing had the longest eating windows, which have been linked to adverse metabolic health. Future experiments should test short sleep duration, sleep timing, and depression as potential causes of lengthened eating windows to determine if targeting sleep duration and timing could improve adverse metabolic markers in depression.

Support: NIMH K.A.R. MH103303

1092

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER SYMPTOMS AND SLEEP CHARACTERISTICS WITHIN A SEASONAL AFFECTIVE DISORDER SPECTRUM

Kim, K. N.¹ Wescott, D. L.² Franzen, P. L.³ Hasler, B. P.³ Roecklein, K. A.²

¹Carnegie Mellon University Department of Psychology, Pittsburgh, PA, ²University of Pittsburgh Department of Psychology, Pittsburgh, PA, ³University of Pittsburgh Department of Psychiatry, Pittsburgh, PA.

Introduction: Seasonal affective disorder (SAD) increases risk for attention-deficit/hyperactivity disorder (ADHD), although the mechanism linking SAD and ADHD is unknown. Prior research has identified insomnia and delayed sleep phase in both ADHD and SAD. We hypothesized that sleep duration and timing in SAD would be associated with the severity of ADHD symptoms.

Methods: Adults with SAD (n = 45) and subsyndromal SAD (S-SAD; n = 18) aged 19-66 years from Pittsburgh, PA., were assessed for ADHD symptoms, self-report sleep quality, depression severity, and daytime sleepiness in the Winter. Participants wore an Actiwatch for 4-14 days, from which we calculated sleep-onset latency, total sleep time, sleep midpoint, and sleep efficiency. We conducted a hierarchical multivariate linear regression to determine if sleep characteristics predict ADHD symptoms. Age and gender were added in Step 1, seasonal depression severity in Step 2, actigraphy-based total sleep time, sleep onset latency, midpoint, and efficiency in Step 3, and self-reported sleep quality and daytime sleepiness in Step 4.

Results: Participants mostly scored in the "likely" or "highly likely" ADHD range (87.30%, n=55), higher than the national prevalence rate (4.4%). When controlling for age, gender, and

depression severity, only shorter actigraphy-based total sleep time was associated with higher ADHD symptom severity (β =-0.30, p<0.05). However, when self-reported sleep quality and daytime sleepiness were added as predictors, total sleep time was no longer a statistically-significant predictor of ADHD symptom severity and only daytime sleepiness predicted ADHD symptom severity (β =0.31, p<0.05).

Conclusion: Our results suggest that individuals with SAD who experience daytime sleepiness and/or possibly shorter actigraphybased sleep duration experience higher ADHD symptom severity. Treatments like Trans-C or CBT-I to improve daytime sleepiness and sleep duration may be indicated for SAD patients who present with comorbid ADHD symptoms.

Support: NIMH K.A.R. MH103303

1093

POPULATION-LEVEL SUICIDE IDEATION: IMPACT OF COMBINED ROLES OF SLEEP DURATION, SLEEP DISTURBANCE, AND DAYTIME SLEEPINESS

Jajoo, A.¹ Tubbs, A.¹ Perlis, M. L.² Chakravorty, S.² Seixas, A.³ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹

¹University of Arizona, Tucson, AZ, ²University of Pennsylvania, Philadelphia, PA, ³New York University, New York, NY.

Introduction: Poor sleep has been shown to be related to suicide ideation and depressed mood, but population-level studies have not been done to explore the specific issues within sleep that effect mood, specifically leading to suicide ideation.

Methods: Data from adults 18 and older in the 2015-2016 National Health and Nutrition Examination Survey (NHANES) who provided complete data were used (N=5,123). Suicide ideation was recorded as the presence of thinking that "you would be better off dead" in the past 2 weeks. Sleep duration was recorded in half-hour increments and transformed to represent absolute distance from 7 hours (to model u-shaped association). Sleep disturbance was recorded as presence of "difficulty falling asleep, staying asleep, or sleeping too much" non, several days, or more than half the days of the past 2 weeks. Sleepiness was frequency feeling "overly sleepy during the day" in the past 12 months. Covariates included age, sex, race/ethnicity, and presence of depressed mood in the past 2 weeks. Additional impact of difficulty thinking/concentrating in the past 2 weeks was explored. NHANES sample weights were used in analyses.

Results: In adjusted analyses, increase likelihood of suicide ideation was associated with distance from 7hrs (OR=1.24/hr, p=0.008), sleep difficulties most of the time (OR=2.46, p=0.001), but not sleepiness. When both sleep variables were adjusted for each other, results remained significant for U-shaped sleep duration (OR=1.21/hr, p=0.02) and sleep disturbance (OR=2.31, p=0.003). These were attenuated but remained significant when difficulty thinking/concentrating was introduced; a significant sobel test (p<0.0001) suggested partial mediation, with this variable accounting for approximately 13% of the variance of the relationship to sleep.

Conclusion: In the population, improper and poor sleep was associated with a greater risk of suicide ideation.

Support: Dr. Grandner is supported by R01MD011600

1094

DEPRESSION IS NOT ASSOCIATED WITH THE PRESENCE OF OR THE SEVERITY OF OBSTRUCTIVE SLEEP APNEA: RETROSPECTIVE STUDY OF 841 POLYSOMNOGRAPHY SUBJECTS

Im, K.¹ Kim, L.² Immen, R.³

¹UC Irvine School of Medicine, Irvine, CA, ²UC Irvine Medical Center, Orange, CA, ³Family Sleep and Wellness Clinic, Towanda, IL.

Introduction: Both depression and obstructive sleep apnea (OSA) are very common medical conditions. Studies showed a co-occurrence of depression and OSA with a higher prevalence of one if the other is present. However, there is relative paucity of studies assessing the rate of depression based on the OSA severity. **Methods:** Retrospective analysis of data collected from patients undergoing polysomnography (PSG) at an academic sleep disorders center was performed. A total of 841 subjects were included and stratified into four groups using AHI. A Chi-square analysis was applied to assess the association of varying levels of AHI and the presence of depression.

Results: Although a significant proportion of patients with AHI greater than 5 endorsed depression (60/165 in group with AHI 15 or greater and 115/278 in group with AHI between 5 and 15), this finding was also replicated in patients with AHI less than 5 (86/202 in AHI between 1 and 5 and 88/196 in those with AHI less than 1). As there was significant difference in rate of depression among women (54.1%) and men (26.1%) (p <0.0001), Chi-square analysis was performed for the rate of depression based on the level of AHI, adjusted for gender. In women the rate of depression from the most severe AHI to less severe AHI group were 0.48, 0.53, 0.60, and 0.53 respectively and in men it was 0.30, 0.27, 0.20, and 0.27 respectively, with no statistical difference between any groups.

Conclusion: Among patients who seek PSG assessment, depression appears to be more prevalent than the general public. Rate of depression is much higher among women than men in this group. However, the presence of OSA or severity of OSA does not have any correlation with the rate of depression in both women and men. These findings might be suggestive of the complexity of the association between depression and OSA. One limitation of this study is the dichotomous nature of depression (presence or absence of). The finding from this study warrants a future study utilizing a numerical rating scale of depression for severity measure to correlate it with the severity of OSA. **Support:** NA.

Support. 1

1095

USE OF MOBILE DEVICES AT NIGHT ASSOCIATED WITH MENTAL HEALTH IN YOUNG ADULTS

Mason, B.¹ Tubbs, A.¹ Hale, L.² Branas, C.³ Barrett, M.⁴ Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹ ¹University of Arizona, Tucson, AZ, ²Stony Brook University, Stony Brook, NY, ³Columbia University, New York, NY, ⁴University of Pennsylvania, Philadelphia, PA.

Introduction: Mobile technology use in bed is becoming commonplace and associated with habitual short sleep duration. The present study examined whether device use at night was related to mental health.

Methods: Data from the Sleep and Healthy Activity, Diet, Environment, and Socialization (SHADES) study comes from a community-based sample, which was restricted to N=473 between the ages of 22-29. Device use was assessed as presence in the room at night, any use at night, texting, emailing, browsing the internet, making or receiving calls, and using social media. Participants were also asked how often they are woken by a call/alert from their phone (unplanned), how often they are woken by their phone alarm (planned), and how often they check their phone at night.

These were recorded as never, rarely, some nights, almost every night, and every night, and were assessed as an ordinal outcome. Predictors included score on the Patient Health Questionnaire depression scale (PHQ9), GAD7 anxiety scale, Perceived Stress Scale (PSS), and Multidimensional Scale of Perceived Social Support (MSPSS). Ordinal logistic regression analyses were adjusted for age, sex, race/ethnicity, education, and income.

Results: Depression was associated with texting (oOR=1.03, p=0.025), email (oOR=1.03, p=0.022), internet (oOR=1.05, p=0.003), unplanned awakenings (oOR=1.05, p=0.001), and checking the phone (oOR=1.09, p<0.0005). Anxiety was associated with texting (oOR=1.05, p=0.001), email (oOR=1.05, p=0.001), internet (oOR=1.05, p=0.002), social media (oOR=1.04, p=0.009), unplanned awakenings (oOR=1.06, p<0.0005), planned awakenings (oOR=1.10, p<0.0005). Perceived stress was associated with internet (oOR=1.02, p=0.034), unplanned awakenings (oOR=1.02, p=0.045), and checking (oOR=1.04, p<0.0005). Social support was associated with decreased checking (oOR=0.98, p=0.018).

Conclusion: Mobile device use at night itself is not associated with mental health, but specific activities may be. Also, those who report more disruptions from the device and more checking of the device also report worse mental health. Relationships might be bidirectional.

Support: Dr. Grandner is supported by R01MD011600 The SHADES study was funded by R21ES022931

1096

MORNING WAKEFULNESS IS ASSOCIATED WITH REDUCED SUICIDAL IDEATION IN A NATIONALLY-REPRESENTATIVE US SAMPLE

Tubbs, A.¹ Khader, W. S.¹ Fernandez, F.¹ Perlis, M. L.² Chakravorty, S.² Grandner, M. A.¹

¹University of Arizona, Tucson, AZ, ²University of Pennsylvania, Philadelphia, PA.

Introduction: Nocturnal wakefulness is a unique risk factor for suicidal ideation in clinical as well as community samples. Preliminary data suggest that morning wakefulness may also be a protective factor against such thinking. However, these associations have not been explored in a nationally-representative dataset.

Methods: Data were collected from the 2015-2016 wave of the National Health and Nutrition Examination Survey. Participants reported typical bedtimes and waketimes. From these values, wakefulness during the night (00:00 to 05:59), morning (06:00 to 11:59), afternoon (12:00 to 17:59), and evening (18:00 to 23:59) was determined. Suicidal ideation was assessed by a question about "thoughts that you would be better off dead, or thoughts of hurting yourself in some way." Ordinal logistic regression estimated the association between the number of hours awake at particular times of day and the frequency of suicidal ideation. Additional analyses adjusted for demographic factors and depressed mood.

Results: Out of 5133 respondents with available data, 125 reported suicidal ideation several days a week, 36 reported suicidal ideation more than half the days, and 29 reported suicidal ideation nearly every day. When controlling for demographics, morning wakefulness was associated with reduced frequency of suicidal ideation (OR: 0.69, 95% CI: [0.59,0.8]). Controlling for depressed mood attenuated, but did not eliminate, this association. Nocturnal wakefulness was not associated with suicidal ideation in this sample.

Conclusion: Using data from a nationally representative sample, morning wakefulness was associated with less frequent suicidal

ideation. However, previous findings regarding nocturnal wakefulness were not replicated. The limited number of individuals in the sample endorsing both suicidal ideation and nighttime wakefulness may have insufficient power to detect an association. **Support:** Dr. Grandner is supported by R01MD011600.

1097

SLEEP DISTURBANCES, SLEEP BURDEN, AND DEPRESSIVE SYMPTOMS IN US HISPANICS/LATINOS: RESULTS FROM THE HCHS/SOL SUEÑO STUDY

Alcantara, C.¹ Wallace, M.² Sotres-Alvarez, D.³ Vetter, C.⁴ Phillips, A. J.⁵ Shafazand, S.⁶ Johnson, D. A.⁷ Wallace, D.⁶ Gallo, L. C.⁸ Ramos, A. R.⁶ Penedo, F.⁶ Wohlgemuth, W. K.⁹ Zee, P. C.¹⁰ Redline, S.¹¹ Patel, S. R.²

¹Columbia University, New York, NY, ²University of Pittsburgh School of Medicine, Pittsburgh, PA, ³University of North Carolina--Chapel Hill, Chapel Hill, NC, ⁴University of Colorado--Boulder, Boulder, CO, ⁵Monash University, Clayton, AUSTRALIA, ⁶University of Miami, Miami, FL, ⁷Emory University, Atlanta, GA, ⁸San Diego State University, San Diego, CA, ⁹US Department of Veterans Affairs, Miami, FL, ¹⁰Feinberg School of Medicine, Northwestern University, Chicago, IL, ¹¹Harvard Medical School, Boston, MA.

Introduction: While sleep disturbances and depression often co-occur, these associations are understudied among Hispanics/ Latinos. We examined the associations of sleep disturbances and sleep burden with depressive symptoms among Hispanic/Latino adults in the United States.

Methods: We used cross-sectional data from the Hispanic Community Health Study/Study of Latinos Sueño Ancillary study (2010-2013). The study enrolled 2072 adults (ages 18-64; 51.5% females) who completed one-week wrist-actigraphy and sleep questionnaires. Sleep burden was operationalized as the total count of sleep disturbances across six domains (duration, efficiency, midpoint, variability, insomnia, sleepiness). Depressive symptoms were assessed using the Center for Epidemiological Studies Depression scale (CESD-10). We used weighted survey linear regressions to evaluate the association of sleep disturbances and sleep burden with elevated depressive symptoms (CESD≥10) in individual models adjusted for age, gender, site, heritage, nativity, education, income, and employment. Sensitivity analyses further adjusted for behavioral health risk factors and apnea-hypopnea index.

Results: An estimated 28.3% had elevated depressive symptoms, 8.0% had short sleep duration (<6 hours of sleep), 10.9% had long sleep duration (>9 hours), 45.2% exhibited a later sleep midpoint (≥4:00AM), 38.4% had high sleep timing variability (upper third tertile for between day sleep midpoint), 15.3% had insomnia (ISI≥10), 17.3% had excessive daytime sleepiness (ESS ≥10), 21.5% had poor sleep efficiency (<85%), and 77.4% had a total sleep burden count of ≥0. Insomnia (B=0.49,95%CI:.43,.56), later sleep timing (B=0.10,95%CI:.04,.16), excessive daytime sleepiness (B=0.19,95%CI:.11,.27), poor sleep efficiency (B=0.09,95%CI:.02,.17), high variability (B=0.07, 95%CI:.01,.12), and sleep burden (B=0.11,95%CI:.09,.13), were each positively associated with elevated depressive symptoms in individual adjusted models and sensitivity analyses. Extreme sleep durations were not associated with elevated depressive symptoms.

Conclusion: Multiple inter-related sleep disturbances, particularly those pertaining to sleep quality and timing, are associated with depression and may be targets for future interventions aimed at improving mood among Hispanics/Latinos.

Support: HL127307, HL098927, HL125748

1098

ALTERED ACTIGRAPHIC BEHAVIORAL ACTIVITY RHYTHM IN DEPRESSION

Chen, I. Y.¹ Neikrug, A. B.¹ Adams, J.² McMillan, L.² Yassa, M. A.² Benca, R. M.¹

¹Department of Psychiatry and Human Behavior, University of California, Irvine, Irvine, CA, ²Department of Neurobiology and Behavior, Center for the Neurobiology of Learning and Memory, University of California, Irvine, Irvine, CA.

Introduction: Disturbances in sleep and behavioral activity rhythms (BAR) are frequently observed in individuals with depression. However, it remains unclear how activity variability across the 24-hour period is specifically associated with this disorder. The present study aimed to examine actigraphy-measured sleep and BAR in depression.

Methods: As part of a larger study, fourteen patients with DSM-5 major depressive episode $(27.8\pm7.7 \text{ years}, 69.2\% \text{ female})$ and 13 healthy controls $(21.8\pm1.2 \text{ years}, 76.5\% \text{ female})$ were evaluated with 7-14 days of wrist-actigraphy. Actigraphy-derived sleep parameters included total sleep time (TST), sleep onset latency (SOL), wake after sleep onset (WASO), and sleep efficiency (SE). Minute-by-minute activity counts were aggregated into hour-by-hour bins; hourly mean activity levels were then generated to depict 24-hour activity patterns (i.e., BAR). Factorial (GroupxTime) mixed models were conducted to examine whether BAR differed between patients with MDD and controls. Generalized Additive Models (GAM), by fitting smoothed nonlinear curves to log-transformed aggregated activity, were performed as exploratory analyses to characterize onset (UP slope) and offset (DOWN slope) of BAR.

Results: Compared to healthy controls, patients with MDD exhibited greater actigraphic TST (p=.026); no other between-group differences were detected for the remaining sleep parameters. Significant between-group differences were observed for mean activity during wakefulness (p<.001). Mixed models assessing hour-by-hour daily activity revealed a significant GroupxTime interaction (p=.001) with significant main effects of group (p=.017) and time (p<.001); patients with MDD had lower activity from 6 to 9 pm (ps<.005). Exploratory GAMs results showed an attenuated DOWN slope in patients with MDD (p=.014), indicating a slower decrease in activity during the evening.

Conclusion: Altered BAR, characterized by an overall dampened activity pattern that was most prominent during the evening, was associated with depression. Furthermore, patients with MDD took longer to wind down in the evening. Future studies are needed to explore the potential benefits of adjunctive interventions addressing both BAR along with sleep in mitigating symptoms of depression.

Support: Research supported by National Institutes of Health R01 MH102392.

1099

SLEEP RESTRICTION LEADS TO GREATER NEXT-DAY ANXIETY: THE MODERATING ROLE OF ANXIETY AND DEPRESSION

Bean, C. A. Ciesla, J. A. Kent State University, Kent, OH.

Introduction: Evidence from both experimental and daily-diary studies suggests that a single night of sleep restriction leads to higher levels of anxiety the following day. Depressive symptoms also increase the next day in healthy populations, although sleep

restriction has demonstrated short-term antidepressant properties in depressed populations. Relatively little research has examined symptoms of anxiety and depression separately on days following naturally-occurring sleep restriction and whether any change from the previous day might be moderated by baseline levels of anxiety or depression.

Methods: Undergraduates completed electronic daily diaries twice per day for 14 days (N = 96). Upon waking, participants answered questions assessing their sleep, and every evening, before going to bed, completed the Mood and Anxiety Symptom Questionnaire - Short Form to assess daily levels of general distress, anxious arousal, and anhedonic depression. The State-Trait Anxiety Inventory - State and the Center for Epidemiologic Studies Depression Scale provided baseline measures of anxiety and depression, respectively. A night of sleep restriction was operationally defined as one for which participants reported obtaining 4 hours of sleep or less.

Results: Multilevel modeling revealed that anxious arousal was higher following sleep restriction ($\beta =.92$, z=2.40, p=.017). This was moderated by baseline anxiety ($\beta =.09$, z=2.89, p=.004) and baseline depression ($\beta =.09$, z=3.39, p=.001) so that anxious arousal was even higher the next day for individuals reporting a high number of anxious and depressive symptoms. These effects remained significant after controlling for lagged anxious arousal, gender, baseline anxiety and depression, substance use, and napping. Conversely, no significant results were found when next-day general distress or anhedonic depression were examined.

Conclusion: Our results suggest that naturally-occurring sleep restriction is accompanied by increases in anxiety, but not depressive symptoms, the following day. This effect is heightened in individuals with higher baseline levels of anxiety and depression.

Support: N/A

1100

SELF-MONITORING OF PVT PERFORMANCE IN HEALTHY ADULTS AND INDIVIDUALS WITH MDD

Galli, O.¹ Goel, N.² Basner, M.¹ Detre, J.¹ Thase, M.¹ Sheline, Y.¹ Rao, H.¹ Dinges, D.¹ Gehrman, P.¹

¹University of Pennsylvania, Philadelphia, PA, ²Rush University Medical Center, Chicago, IL.

Introduction: Negativity bias in depression has been repeatedly demonstrated in the judgment and decision-making literature. Research investigating the impact of sleep deprivation on self-evaluation of performance in healthy or depressed populations is limited. We examined 1) whether individuals with Major Depressive Disorder (MDD) exhibit a negativity bias in subjective ratings of performance on the Psychomotor Vigilance Task (PVT) as compared with healthy adults, and 2) the impact of total sleep deprivation (TSD) on these ratings.

Methods: N=33 individuals with MDD and n=9 healthy adults completed a 5-day study protocol including two baseline nights (B1-B2, 9h TIB), 36 hours of TSD, and one night of recovery sleep opportunity (Rec). The PVT was administered every 2-4 hours. A brief questionnaire was administered immediately prior to (PRE) and following (POST) the PVT, asking participants to estimate their average reaction time (RT) using a 9-point Likert-type scale. Mixed-effects models examined the impact of group (MDD, Control), protocol day (B1, B2, SD, Rec), and their interaction on objective PVT performance (mean RT) and subjective performance estimates (PRE and POST ratings).

Results: Mean RT was significantly slower during TSD (p<0.001) for all participants. Individuals with MDD and healthy adults did not differ in objective PVT performance (p=0.25) across days. There was no significant interaction between group and protocol day (p=0.96). Both groups predicted slower RTs during TSD as compared with baseline or recovery days (PRE-PVT, p=0.006). Individuals with MDD anticipated slower RTs as compared with healthy adults (p=0.001). On POST-PVT estimates, all participants reported subjective poorer performance during TSD (p<0.008). Individuals with MDD reported slower RTs as compared with healthy adults (p=0.002). Interaction effects between group and protocol day on PRE- and POST- performance ratings were not significant.

Conclusion: This project is the first to investigate subjective estimates of PVT performance in healthy and depressed individuals. Individuals with MDD subjectively reported slower response times as compared with control participants, despite similar objective performance. Depressive symptoms may be a potential confounder of subjective, but not objective, PVT performance. **Support:** 5R01MH107571

1101

DAYTIME INTERACTIONS OF ANXIOUSNESS, STRESSFULNESS, SLEEPINESS, AND FATIGUE IN COLLEGE STUDENTS: A LONGITUDINAL CLUSTER ANALYSIS

LaJambe, C. M. Shaffer, V. N. Brown, F. M. The Pennsylvania State University, University Park, PA.

Introduction: Reports of anxiety and stress are increasing among college-age students. Previous findings show bi-directional relationships with nighttime sleep loss and elevated anxiety and stress. Less is known about changes across the day (profiles) in anxiousness and stress, and their relationship to daytime sleepiness and fatigue profiles. The primary objective of this study was to examine these potential interactions.

Methods: Using an ecological momentary assessment design, university undergraduates (N=102, female= 77.5%) made smart phone ratings (0-9 Scale) of anxiousness, stressfulness, fatigue, sleepiness from Monday-Friday, six times per day. Participant were either Low-Anxious (0-1 score: n=34) or High-Anxious (>7 score; n=68) on the GAD-Q-IV Scale, had > 7h sleep per weeknight, and bed- and wake times between 06:30-09:30 and 22:30-02:00, respectively. Mean daytime profile interactions were examined using joint-trajectory longitudinal clustering.

Results: Clustering identified two subtypes, Lo (n=46) and Hi (n=56), which differed primarily in their quadratic curve levels, and in times of highest and lowest ratings. Subtypes differed (p<.001) in overall mean ratings of anxiousness (Lo=1.6 \pm 0.9, Hi=3.6 \pm 1.1), stressfulness (Lo=2.0 \pm 0.9, Hi=4.3 \pm 1.1), fatigue (Lo=2.4 \pm 1.0, Hi=4.4 \pm 1.0), and sleepiness (Lo=2.9 \pm 1.0, Hi=4.3 \pm 1.1). Subtypes were similar in age (Lo=18.6 \pm 0.9, Hi=18.9 \pm 1.3), morningness-eveningness BALM (Lo=33.2 \pm 4.1, Hi=32.1 \pm 5.1) and MEQ (Lo=47.5 \pm 6.2, Hi=47.7 \pm 6.8) scores, and hours sleep (Lo=8.5 \pm 0.8, Hi=8.4 \pm 0.9). Notably, more females were Hi subtype (83.9%) than Lo subtype (69.6%). Also, GAD-Q-IV scores differed within subtypes: Hi (80.4% High-Anxiety, 19.6% Low-Anxiety) versus Lo (50.0% for both High- and Low-Anxiety).

Conclusion: Despite apparent adequate nightly sleep, elevated anxiousness and stressfulness may be accompanied by increased daytime fatigue and sleepiness in college students. Furthermore, standardized clinical tests designed to identify levels of anxiety and stress may not align with daily experiences of those states. Better

understanding of concomitant daytime profiles of anxiousness, stress, sleepiness, and fatigue could facilitate treatment development and reduce the burden on student counseling centers. **Support:** None

1102

RELATIONSHIP BETWEEN EMOTIONAL DISTRESS AND SLEEP DURATION AMONG HISPANICS USING THE 2018 NATIONAL HEALTH INTERVIEW SURVEY DATASET

Garcia, J.¹ Moore, J.¹ Payano, L.¹ Rogers, A.² Poke, P.¹ Casimir, G.³ Jean-Louis, G.¹ Seixas, A.¹

¹NYU Grossman School of Medicine, New York, NY, ²St. John's University, Queens, NY, ³SUNY Downstate Medical Center, Brooklyn, NY.

Introduction: Although Hispanics experience a high level of shorter sleep duration (< 7 hrs./24 period), a clear mechanism or cause is lacking. Previous research indicate that emotional distress may explain the burden of shorter sleep among blacks. Applying these findings to Hispanics, we investigated whether emotional distress explains the burden of short sleep duration (< 7 hrs.) among Hispanics and if this relationship varies by sex

Methods: We used data from the 2018 National Health Interview Survey (NHIS) dataset, a nationally representative sample, in which only Hispanic ethnicity participants (N=3,091) were analyzed. Average sleep duration was self-reported and measured in hours. Emotional distress was measured using Kessler 6, which measures how an individual felt over the past 30 days: nervous, hopeless, restless/fidgety, depressed, effortful and worthless. To assess the association between short sleep duration and emotional distress, we performed Pearson correlation, hierarchical regression analyses, and stratified this relationship by sex to determine if this relationship differed between males and females, adjusting for covariates.

Results: Of the total sample of 3,091 Hispanics, 1,762 were female, and 1,329 were male. Sleep duration and emotional distress were negatively correlated among females (r = -.27, p <.001) and males (r=-.18, p <.001). Among Hispanic females, sleep duration significantly predicted emotional distress, $\beta = -.27$, t = -11.60, p <.001, and explained a significant portion of variance in emotional distress, $R^2 = .07$, F = 134.63, p <.001. While, among Hispanic males, sleep significantly predicted emotional distress ($\beta = -.18$, t = -6.5, p <.001) and explained a significant portion of the variance in emotional distress ($R^2 = .03$, F = 42.37, p <.001).

Conclusion: Our findings indicate that a negative sleep-ED relationship, suggesting that shorter sleep was predictive of higher levels of emotional distress among Hispanics and that this relationship is greater among Hispanic females, compared males. **Support:** K01HL135452, R01MD007716, R01HL142066, and K07AG052685

1103

UNDERSTANDING THE ROLE OF SLEEP AS A RISK FACTOR FOR SUICIDAL IDEATION IN ACTIVE DUTY SERVICE MEMBERS

Paxton Willing, M. M.¹ Pickett, T. C.² Tate, L. L.¹ Rhodes, C.² DeGraba, T.²

¹Uniformed Services University of the Health Sciences, Bethesda, MD, ²National Intrepid Center of Excellence, Walter Reed National Military Medical Center, Bethesda, MD, ³Uniformed Services University of the Health Sciences, Bethesda, MD.

Introduction: Suicide is an important public health concern with many factors contributing to increased risk. Sleep is one such factor that may elevate risk, yet this association is not well understood. By identifying the strongest sleep-related predictors of suicidal ideation (SI), providers may be able to better intervene and reduce risk of suicide.

Methods: Data were obtained from the clinical database at the National Intrepid Center of Excellence (NICoE). Patients were active duty service members, predominantly male, and with a mean age of 38. As part of standard care, patients receive a polysomnography sleep study and complete a battery of intake measures offering a comprehensive view of sleep. Individual symptoms were analyzed in an effort to understand the role of each sleep symptom within the context of the many other factors that may contribute to SI in service members.

Results: Of the many data points collected during polysomnography, only rapid eye movement (REM) sleep latency and minimum sleeping heart rate were related to SI. REM latency was associated with increased odds of SI, while minimum sleeping heart rate was related to decreased odds. Subjective reports of bad dreams, trauma-specific bad dreams, sleepiness, and sleep quality were related to increased odds of SI. Notably, subjective reports of sleep were associated with greater odds than objective measures. Traumatic nightmares had the greatest odds, with these patients being much more likely to have SI.

Conclusion: These results support the importance of considering sleep factors when evaluating SI in service members. Subjective sleep reports, specifically, appear to be particularly important, as they were associated with increased odds of SI. These findings focus on the role of individual sleep factors in increasing the odds of SI and suggest it is important to evaluate sleep in combination with comorbid conditions when conducting risk assessments. **Support:** N/A

1104

INCREASING ACCESSIBILITY OF NIGHTMARE TREATMENT VIA MOBILE HEALTH

Speed, K. J.¹ Nadorff, M.² Bishop, T.¹ Stearns, M.² Pigeon, W.¹ ¹Center of Excellence for Suicide Prevention, Canandaigua, NY, ²Mississippi State University, Starkville, MS.

Introduction: Nightmares have been tied to a myriad of adverse mental health outcomes and are known to persist after treatment of other concerns such as posttraumatic stress, depression, and anxiety. When reaching clinical levels, nightmare disorder is known to effect 2-6% of the general population, Although many treatments exist for nightmare disorder and posttraumatic nightmares, Imagery Rehearsal Therapy has consistently been cited as the first line treatment. Mobile health (mHealth) technology has emerged as a viable platform from which to deliver sleep medicine interventions.

Methods: We assessed the efficacy of an Imagery Rehearsal Therapy-based mobile application (Dream EZ) developed by the National Center for Telehealth and Technology. College students (n = 99) were recruited in a two-part online study and randomized to the treatment condition or waitlist control. Repeated measures analysis of variance were used to assess the efficacy of smartphone-based mHealth application treatment (Dream EZ) in reduction of psychological symptoms (nightmare distress, PTSD symptoms, and suicide risk) as compared to waitlist control.

Results: Findings support the use of Dream EZ for nightmares distress reduction (main effect: p = .004, d = .57; interaction: p = .049,

d = .41). Results regarding effectiveness of Dream EZ in relation to reduction of PTSD symptoms (main effect: p = .415, d = .17; interaction: p = .262, d = .23) showed no significant interactions between PTSD symptoms and treatment group assignment. In relation to changes in suicidality (main effect: p = .007, d = .57; interaction: p = .758, d = .07), findings were nonsignificant.

Conclusion: Use of nightmare-focused treatment through a mHealth smartphone application may be a viable avenue for promoting management of nightmare distress in college students. These findings present an opportunity to explore further options for increasing accessibility of sleep-focused treatment options in a challenging and fast-paced population. **Support:** No support to disclose.

1105

A RANDOMIZED CONTROLLED TRIAL OF A PSYCHOLOGICAL INTERVENTION FOR DECREASING BEDTIME PROCRASTINATION: THE BED-PRO STUDY

Suh†, S. KIM, G. Jeoung, S. An, H. SUNGSHIN WOMEN'S UNIVERSITY, SEOUL, KOREA, REPUBLIC OF

Introduction: Bedtime Procrastination (BP) is defined as the behavior of going to bed later than intended, without having external reasons for doing so. Previous studies have shown that BP has a negative effect on sleep and health, and there is a need to develop interventions to decrease BP. This study (BED-PRO) is an ongoing study evaluating a behavioral intervention to reduce BP.

Methods: Fifteen participants who scored higher than 33 on the Bedtime Procrastination Scale were randomized to either the treatment (TRT, n=6) or control group (CTRL, n=9). Treatment consisted of four face-to-face individual sessions. All participants completed self-report questionnaires on Bedtime Procrastination Scale (BPS), Epworth Sleepiness Scale (ESS), Positive Affect and Negative Affect Schedule (K-PANAS-R) and completed the 7-day sleep diary. Data was analyzed using two-way mixed Measures Analysis of Variance (ANOVA).

Results: Mean age of the participants was 21.78 (\pm 1.8) years and 80% (n=12) were females. Group by time interactions from repeated measures analyses revealed significant post intervention improvements in the TRT group compared to the CTRL group on all bedtime procrastination duration and scores, sleep efficiency, refreshment after waking, daytime sleepiness and negative affect of K-PANAS-R. Specifically, bedtime procrastination duration in the TRT group measured by sleep diaries decreased significantly from 75.30 (\pm 58.57) min to 14.83 (\pm 7.83) min, while the CTRL group did not change from 57.60 (\pm 32.01) to 54.36 (\pm 40.82) min (p=0.019). In addition, the TRT group reported significant improvements in bedtime procrastination scores from 36.00 (\pm 4.05) to 22.50 (\pm 6.72).

Conclusion: Based on results, the behavioral intervention used in this study looks promising in improving bedtime procrastination and sleep.

Support: This work was supported by the Ministry of Education of the Republic of Korea and the National Research Foundation of Korea(NRF-2018S1A5A8026807)

1106

ASSOCIATIONS BETWEEN SEVERE MENTAL ILLNESS AND POSITIVE AIRWAY ADHERENCE IN A VETERAN COHORT

May, A. M.^{1,2,3} Gandotra, K.^{1,2,3} Jaskiw, G. E.^{1,3}

¹VA Northeast Ohio Healthcare System, Cleveland, OH,

²University Hospitals Cleveland Medical Center, Cleveland, OH, ³Case Western Reserve University School of Medicine, Cleveland, OH.

Introduction: Serious mental illness (SMI) is associated with excess morbidity and mortality irrespective of healthcare access. Adherence differences may contribute to this health disparity. In those with sleep disorders, adherence to positive airway pressure (PAP) can improve health outcomes. We hypothesized that SMI is associated with lower PAP adherence.

Methods: In this retrospective cohort study, 5047 veterans receiving a PAP machine from the VANEOHS (1/1/2010 - 6/31/2015) were evaluated for 90-day PAP adherence (% days used ≥4 hours). A composite variable of any billing diagnosis of psychotic spectrum disorder, bipolar disorder, post-traumatic stress disorder (PTSD), or major depression was examined via linear regression. Analyses were adjusted for demographics, comorbidities, and medications. We conducted sensitivity analyses of 30-day adherence as well as subset analyses of associations between each disorder and adherence.

Results: The group was 38.8 ± 11.9 years old, 3.9% female with 52.6% with major depression, 25.0% with PTSD. 58.4% of the cohort had at least one psychiatric disorder. PTSD with depression was the most common comorbidity in those with two or more psychiatric diagnoses. Unadjusted analyses showed worse adherence in those with any SMI ($\beta = -7.5\%$, 95% CI: -9.8% -5.3%), which was mitigated in adjusted analyses ($\beta = -1.6\%$, 95% CI: -5.1%, 1.9%). All individual SMIs were negatively associated with adherence, but only PTSD was associated with less adherence in adjusted analyses ($\beta = -6.4\%$, 95%CI: -11.2%, -1.6%). Sensitivity analyses of 30-day adherence were similar to primary analyses.

Conclusion: In this large cohort of veterans, broadly defined SMI was associated with lower 30- and 90-day adherence in unadjusted but not adjusted analyses. Replication and refinement of the link between SMI, particularly PTSD, and adherence may provide opportunities for targeted interventions and improve health disparities.

Support: This work was supported in part by Career Development Award IK2CX001882 from the United States (U.S.) Department of Veterans Affairs Clinical Sciences Research and Development Service.

The contents of this work do not represent the views of the Department of Veterans Affairs or the United States government.

1107

AROUSAL AND SLEEPINESS IN OPIOID USE DISORDER COMPARED TO INSOMNIA DISORDER WITH AND WITHOUT COMORBID PSYCHIATRIC CONDITIONS

Krishnamurthy, V. B.¹ Hussain, N.² Puzino, K.¹

Yadav, S.¹ Del Tredici, S.³ Vgontzas, A. N.¹ Bixler, E. O.¹ Fernandez-Mendoza, J.¹

¹Penn State College of Medicine, Hershey, PA, ²Yale University, New Haven, CT, ³WellSpan Health, York, PA.

Introduction: Insomnia is frequent in opioid use disorder patients on buprenorphine (OUDB) and increases risk of relapse. There is lack of data evaluating specific differences in hyperarousal and daytime sequelae between OUDBs as compared to individuals with insomnia disorder without (ID) or with comorbid psychiatric conditions (CID).

Methods: We studied 112 patients with ID (47.8 \pm 16.3y, 55% female, 13% minority) and 148 with CID (44.7 \pm 15.6y, 69% female,

16% minority) evaluated at the Behavioral Sleep Medicine program of Penn State Hershey Sleep Research & Treatment Center and 71 OUDB (37.8±11.2y, 51% female, 16% minority) evaluated at the Recovery, Advocacy, Empowerment and Service program and WellSpan Internal Medicine clinics (York, PA). Subjects completed the Insomnia Severity Index (ISI), Ford Insomnia Response to Stress (FIRST), Arousal Predisposition Scale (APS), Presleep Arousal cognitive (PSAS-C) and somatic (PSAS-S) Scale, Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS) and Epworth Sleepiness Scale (ESS). Excessive daytime sleepiness (EDS) was defined as an ESS score \geq 10. MANCOVA included age, sex, race/ethnicity and depression as covariates, while logistic regression further included ISI, APS and PSAS-S.

Results: No differences across groups were observed in PSAS-C or DBAS scores. Subjects with CID and OUDB had significantly higher PSAS-S (15.7 ± 0.5 and 16.4 ± 0.7 , respectively) and APS (35.6 ± 0.6 and 36 ± 1 , respectively) scores as compared to the ID group (14.2 ± 0.6 and 33.2 ± 0.7 , respectively). Subjects with OUDB had significantly higher ESS score (9.8 ± 0.6) as compared to the ID or CID groups (6.2 ± 0.5 and 6.4 ± 0.4 , respectively). The odds of EDS were 2.7 times (95%CI=1.2-6.1) higher in the OUDB group as compared to the ID group.

Conclusion: OUDB may present with similar phenotypic insomnia symptoms as patients with ID or CID but report more sleepdisturbing somatic symptoms and EDS. These data have important implications for tailoring behavioral and pharmacological treatments of insomnia to this specific patient population.

Support: Junior Faculty Development Program, Penn State College of Medicine

1108

ASSOCIATIONS BETWEEN INSOMNIA AND ANXIETY SYMPTOMS: WHICH ELEMENTS OF INSOMNIA ARE ASSOCIATED WITH WHICH ELEMENTS OF ANXIETY?

Kapoor, A.¹ Perlis, M. L.² Bastien, C.³ Williams, N.⁴ Hale, L.⁵ Branas, C.⁶ Barrett, M.² Killgore, W. D.¹ Wills, C. C.¹ Grandner, M. A.¹

¹University of Arizona, Tucson, AZ, ²University of Pennsylvania, Philadelphia, PA, ³Laval University, Quebec, QC, CANADA, ⁴New York University, New York, NY, ⁵Stony Brook University, Stony Brook, NY, ⁶Columbia University, New York, NY.

Introduction: It is still not clear which aspects of insomnia are associated with various aspects of anxiety problems. Knowing this could better guide treatment of insomnia comorbid with anxiety. Methods: Data from the Sleep and Healthy Activity, Diet, Environment, and Socialization (SHADES) study were used, including N=1003 adults age 22-60. All participants completed the Insomnia Severity Index (ISI) and the GAD7 anxiety questionnaire. The ISI was divided into 3 sections, based on prior work: SLEEP symptoms (difficulty sleeping), DAYTIME symptoms (difficulty functioning), and PERCEPTION symptoms (dissatisfaction). GAD7 items included anxiety level, loss of control, worry about many things, difficulty relaxing, restlessness, irritability, and fear. Logistic regression analyses examined each symptom, with each component of the ISI as predictor, as well as age, sex, race/ ethnicity and education as covariates.

Results: SLEEP symptoms were independently associated with control (OR=1.09, p=0.03), many worries (OR=1.1, p=0.017), restlessness (OR=1.1, p=0.009), and irritability (OR=1.1, p=0.04). DAYTIME symptoms were independently associated with anxiety level (OR=1.3, p<0.0005), control (OR=1.2, p<0.0005),

many worries (OR=1.3, p<0.0005), difficulty relaxing (OR=1.2, p=0.004), restlessness (OR=1.3, p=0.001), and irritability (OR=1.2, p<0.0005). PERCEPTION symptoms were uniquely, independently associated with anxiety level (OR=1.1, p=0.03), control (OR=1.2, p=0.001), many worries (OR=1.2, p=0.001), difficulty relaxing (OR=1.4, p<0.0005), irritability (OR=1.2, p=0.018), and feelings of fear (OR=1.2, p=0.002).

Conclusion: The DAYTIME and PERCEPTION symptoms of insomnia were strongly related to anxiety symptoms. Current treatments for insomnia focus mainly on improving sleep. Future research should test the hypothesis that treating daytime symptoms of insomnia may aid patients with comorbid anxiety.

Support: The SHADES study was funded by R21ES022931. Dr. Grandner is supported by R01MD011600.

1109

BROADLY ASSESSING SLEEP COMPLAINTS IN A SAMPLE OF PATIENTS WITH ADHD

Seewald, M.¹ Alio, C.² Rosenfield, B.² DiTomasso, R.² Muench, A. L.¹ Rostain, A. L.³ Ramsay, J.³ Klingman, K.⁴ Perlis, M. L.¹

¹Behavioral Sleep Medicine Program, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA, ²Philadelphia College of Osteopathic Medicine, Philadelphia, PA, ³Adult ADHD Treatment & Research Program, University of Pennsylvania, Philadelphia, PA, ⁴SUNY Upstate Medical University College of Nursing, Syracuse, NY, ⁵Behavioral Sleep Medicine Program, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA.

Introduction: It is commonly observed in clinical settings that patients with ADHD regularly present with comorbid "sleep disturbances". In the absence of broad based sleep disorders assessments, it is thought that this represents Delayed Sleep Phase Disorder (DSPD). Recently, a surveillance study was undertaken in a university-based, outpatient specialty clinic for adults with ADHD, by adding a comprehensive sleep disorders screener (SDS-CL-25) to the clinical intake procedures. These data were used to ascertain which sleep disorders symptoms are common in this clinical cohort.

Methods: SDS-CL-25 data were collected in 150 subjects (93/57 male/female, mean age 32.8, age range 18-79). The SDS-CL-25 is a 25 item instrument developed to screen for 13 sleep disorders at one time (Sleep Dx symptoms are endorsed on Likert-scales; 0 [never] 4 [>5x/ week]). For the purposes of this study, the percentage of subjects endorsing frequent symptomatology (sum of the percent of endorsements for columns 3 & 4)was calculated per symptom. Sums of \geq 20% were considered, a priori, to be of clinical significance.

Results: Patients endorsed: increased fatigue (59%); SL or WASO or EMA's \geq 30 minutes (40%; 26%; 21%, respectively); late preferred time to bed (31%); work & school limits sleep opportunity (30%); variable time to and out of bed (27%); and snoring (21%). The average percent endorsement was 15% (range 0-59%).

Conclusion: These results suggest that, consistent with clinical observations, adult patients diagnosed with ADHD frequently endorse late preferred time to bed, variable sleep wake schedules, work/school limitations on sleep opportunity, and sleep onset problems that are accompanied by daytime fatigue. This constellation of symptoms is consistent with the notion that patients with ADHD tend to have comorbid DSPD. The high prevalence of middle and late insomnia was unexpected and suggests that Insomnia Disorder (proper) may also be a feature of ADHD.

Support: No support was provided for this abstract.

1110

CHARACTERISTICS OF UNTREATED SLEEP DISTURBANCE DURING INTENSIVE OUTPATIENT TREATMENT FOR SUBSTANCE USE DISORDERS: PRELIMINARY RESULTS FROM A LONGITUDINAL STUDY

Wilkerson, A. K.¹ Taylor, D. J.² Sahlem, G. L.¹ Simmons, R. O.¹ Russell, A.³ Book, S. W.¹ Smith, J. P.¹ Uhde, T. W.¹ McRae-Clark, A. L.¹

¹Medical University of South Carolina, Charleston, SC, ²University of Arizona, Tucson, AZ, ³College of Charleston, Charleston, SC.

Introduction: Previous studies have shown that sleep problems are commonly reported during treatment for substance use disorders (SUDs) and sleep complaints have been linked to subsequent relapse. However, most of these findings were in well-controlled clinical trials and may not generalize to the public. Little is known about the natural progression of sleep complaints during treatment in community clinics, the most common treatment approach for SUDs. The aim of this study is to longitudinally assess prevalence of clinically significant sleep disturbance at baseline and post-treatment in a community-based intensive outpatient (IOP) SUD treatment program using a multi-method approach with well-validated measures of sleep.

Methods: Adults beginning IOP SUD treatment completed the Pittsburgh Sleep Quality Index (PSQI), Insomnia Severity Index (ISI), Nightmare Disorder index (NDI), and one week of actigraphy and sleep diaries. Measures were repeated following treatment (approximately 5 weeks later).

Results: Preliminary analyses on 21 adults who have been enrolled thus far revealed 85.6% of participants experienced sleep disturbance (PSQI > 5) at baseline. 28.5% of participants reached cutoff for moderate-to-severe insomnia symptoms (ISI > 15) and 42.9% reported nightmares more than once per week. Sleep parameters taken from actigraphy and sleep diaries revealed mean sleep efficiency was 77.5% (TST M = 6.2 hours; TIB M = 7.9 hours). These variables did not improve from baseline to post-treatment. Further, most measures indicated a worsening of sleep, though this did not reach significance (all ps > .05).

Conclusion: This preliminary data show a high prevalence of selfreported sleep complaints and objectively measured poor sleep efficiency that do not improve over the course of treatment. Data collection is ongoing and expected to at least double. More robust analyses, including differences between SUD type (e.g., cannabis vs. opioid) and relationship to relapse at post-treatment, will then be completed. **Support:** K12DA031794

1111

DO SLEEP DISORDER SYMPTOM ENDORSEMENTS DIFFER BETWEEN ADHD SUBTYPES?

Seewald, M.¹ Muench, A.¹ Alio, C.² Rosenfield, B.² DiTomasso, R.² Rostain, A.³ Ramsay, J.³ Klingman, K.⁴ Perlis, M. L.¹ ¹Behavioral Sleep Medicine Program, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA, ²Philadelphia College of Osteopathic Medicine, Philadelphia, PA, ³Adult ADHD Treatment & Research Program, University of Pennsylvania, Philadelphia, PA, ⁴SUNY Upstate Medical University College of Nursing, Syracuse, NY, ⁵Behavioral Sleep Medicine Program, Department of Psychiatry, University of Pennsylvania, Philadelphia, PA. **Introduction:** To date, research on differences in sleep complaints between patients with different subtypes of ADHD has been mixed. On balance, the evidence tends towards ADHD-Combined Presentation (ADHD-C) being associated with more severe sleep and sleep-related daytime complaints than ADHD-Primarily Inattentive (ADHD-I). In order to further assess this issue a surveillance study was undertaken in an active ADHD clinic by adding a comprehensive sleep disorders screener (SDS-CL-25) to the clinical intake procedures. These data were used to ascertain whether the two subtypes differ for any of 13 sleep disorders symptoms.

Methods: Subjects (n = 132; 83 male, 49 female, mean age 32.8, age range 18-79), presenting to the clinic for evaluation for ADHD were given the SDS-CL-25. The SDS-CL-25 is a 25-item instrument developed to screen for multiple sleep disorders at one time (problems are endorsed on a Likert-scale; 0 = never and 4 = more than 5x/week). Endorsements greater than 3x/week were counted as positive for the symptom and less than three days per week was considered negative. Percent per group was compared using Chi Square Analyses. Cumulative morbidity means were also analyzed using t-tests. The subtype, ADHD-I (n=71) and ADHD-C (N=61), was established using EMR records.

Results: No significant differences between patients with ADHD-I and ADHD-C were detected.

Conclusion: The lack of finding in the present analysis may reflect a lack of difference or a failure to detect differences based on the small sample sizes or lack of statistical control for likely confounders (age, sex, illness severity or chronicity, SES status, etc.). Analyses are ongoing.

Support: No support was provided for this abstract.

1112

MENTAL HEALTH AND SLEEP DISORDERS ARE ASSOCIATED WITH ELEVATED C-REACTIVE PROTEIN IN RETURNING VETERANS

Straus, L. D.¹ Colvonen, P. J.² Bertenthal, D.¹ Neylan, T. C.¹ O'Donovan, A.¹

¹San Francisco VA Medical Center, San Francisco, CA, ²San Diego VA Healthcare System, San Diego, CA.

Introduction: Mental health disorders and sleep disorders are associated with systemic inflammation, which may be a key element linking these highly co-occurring conditions to negative health outcomes. This study used national VA medical records to examine C-reactive protein (CRP) levels in Iraq/Afghanistan veterans based on presence of mental health and/or sleep disorder diagnoses.

Methods: We examined medical records for 16,576 Iraq/ Afghanistan veterans under age 55 who had high-sensitivity CRP results reported. ICD diagnostic codes were used to compare CRP values for: a) veterans without sleep disorders or mental health diagnoses, b) veterans with mental health disorders only, c) veterans with sleep disorders only, and d) veterans with both conditions. In generalized linear models controlling for demographics, we examined the impact of diagnostic category on continuous CRP value as well as the risk of elevated CRP (>3mg/L).

Results: Veterans with mental health disorders (coeff=.14, p<.001) and comorbid sleep and mental health disorders (coeff=.21, p<.001) had higher continuous CRP values compared to veterans without either condition. Veterans with comorbid sleep and mental health disorders had higher continuous CRP values than veterans with sleep disorders alone (coeff=.22, p<.041); however, there were few patients in the current sample who were diagnosed with sleep disorders alone (n=401, 2.4%). Additionally, veterans with

mental health disorders (ARR=1.12, p=.004) and comorbid sleep and mental health disorders (ARR=1.15, p=.001) were more likely to have CRP values >3mg/L compared to veterans without either condition.

Conclusion: Sleep disorders were highly likely to co-occur with mental health disorders in this sample of Iraq/Afghanistan veterans. Mental health disorders and comorbid mental health/ sleep disorders were associated with elevated C-reactive protein, indicating these patients are at highest risk for negative health outcomes. Future studies should investigate directionality of relationships among sleep disruption, mental health symptoms, and inflammation.

Support: VA Advanced Fellowship Program in Mental Illness Research and Treatment

1113

PHYSICAL ACTIVITY MODERATES THE SLEEP-EMOTIONAL DISTRESS RELATIONSHIP, BUT LESS SO AMONG BLACKS VS. WHITES

Moore, J. Williams, N. Chung, D. Parra, Y. Jean-Louis, G. Seixas, A.

NYU Grossman School of Medicine, New York, NY.

Introduction: Emotional distress (ED) is associated with poor sleep. Research shows that minority populations experience greater vulnerabilities to both ED and poor sleep. Interventions such as relaxation training and behavioral therapy address this relationship but are not always successful. Research shows that physical activity (PA) is negatively associated with ED and positively associated with sleep duration. However, it is unclear whether PA attenuates the relationship between ED and sleep, and if this relationship differs by race/ethnicity.

Methods: We analyzed data from the 2005-2015 National Health Interview Survey (NHIS), a nationally representative dataset of 416,152 participants. ED, hours of PA per day, and average sleep duration were collected. Regression models with covariates (age, sex, employment status, BMI) were used to analyze the moderation effect of PA within sleep and ED. Regression models were stratified by race/ethnicity.

Results: 261,686 participants (45,926 blacks, 17.55%, and 215,760 whites, 82.45%) responded with the required variables for analysis. 63% of participants reported at least some physical activity. The results of the regression showed that a significant amount of variance in ED stemmed from sleep duration; *F* (7, 121088) = 1,619.72, *p* < 0.001. PA was found to have a significant main effect, t(121,088) = 9.01, p = <0.001. There was a significant moderation effect of PA, t(121088) = 7.26, *p* < 0.001. Stratification showed that the moderation effect of PA was not significant among blacks t(121,088) = -1.45, p=0.149 and significant among whites b = -.08, t(101,754) = -7.82, *p* < 0.001.

Conclusion: The present study found support for moderation of PA in the sleep-ED relationship. However, it found that blacks do not experience the same benefits of PA in this relationship as whites. Further research should be performed to understand the connection of PA to sleep duration and ED.

Support: This study was supported by funding from the NIH: R01MD007716, R01HL142066, R01AG056531, K01HL135452, and K07AG052685

1114

URBANICITY AND THE SLEEP-MENTAL HEALTH RELATIONSHIP

Moore, J.¹ Seixas, A.¹ Casimir, G.² Nunes, J.³ Matadiaby, F.⁴ Khosrof, A.⁵ Jean-Louis, G.¹

¹NYU Grossman School of Medicine, New York, NY, ²SUNY DownState Medical Center, Brooklyn, NY, ³City College / CUNY, New York, NY, ⁴Brooklyn College, New York, NY, ⁵Fordham University, New York, NY.

Introduction: Inadequate sleep has been found to be associated with poor mental health. This is especially true in low-income and minority populations, who are concentrated in cities. It is not understood to what degree living in a city vs. a rural environment affects sleep and resulting mental health outcomes. This study seeks to understand how living in an urban environment affects the relationship between inadequate sleep and mental health.

Methods: The study used data from the 2018 US Behavioral Risk Factor Surveillance System (BRFSS,) a nationwide health dataset collected by telephone. Respondents were classified as living in either an urban or rural environment based on their zip code. Respondents reported hours of sleep per night and mental health status. This study classified mental health status based on whether the respondent reported one or more incidences of poor mental health in the previous 30 days.

Results: After filtration, 348,540 respondents were split into urban and rural groups. Binary logistic regression was run in each group to compare how much living in an urban environment contributed to the relationship between sleep duration and mental health. Sleep in the analysis was found to significantly contribute to both models; urban $X^2(15, N=295,796) = 11,485.70, p < 0.001$ rural $X^2(15, N=52,744) = 2,465.64, p < 0.001$. The estimated odds ratio resulted in a decrease of 13.9% [Exp(B) = 0.861] in reported poor mental health for every unit increase of sleep in the urban population, and decrease of 14.9% [Exp(B) = 0.851] in the rural population.

Conclusion: In urban and rural dwellers, sleep duration predicted poor mental health. Contrary to expectations, sleep was more strongly tied to mental health in rural than urban populations. This was true even after controlling for sex, income, and education level. Further research should seek to understand how environment affects sleep and mental health.

Support: This study was supported by funding from the NIH: R01MD007716, R01HL142066, R01AG056531, K01HL135452, and K07AG052685.

1115

SLEEP MISPERCEPTION, ANXIETY, AND SLEEP QUALITY

Weathers, J. Moran, E. Stiver, J. Zimmerman, M. Fordham University, Bronx, NY.

Introduction: Sleep misperception is a phenomenon often identified in insomnia literature, in which individuals subjective reporting does not match objective measurements of their own sleep. Research indicates that anxiety symptoms may play a role in sleep misperception. This study assessed the relationship between sleep misperception, sleep quality, and anxiety in a young adult population with sub-clinical insomnia and anxiety symptoms. Linear regression models examined the relationships between sleep quality, anxiety symptoms, and sleep misperception.

Methods: This sample consisted of 130 young adults recruited from a University in the Bronx, NY. Anxiety was assessed using the Beck Anxiety Inventory (BAI), and sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). Objective measures of sleep were collected via wrist-based actigraphy over a period of 7-14 days. Subjective sleep measures were collected via an online sleep diary. A misperception index was calculated to determine the discrepancy between subjective and objective sleep measures based on a formula established in previous research.

Results: Higher anxiety symptoms were associated with greater sleep disturbance. Higher sleep misperception was not associated with greater sleep disturbance. There was a significant, positive relationship between sleep misperception and anxiety symptoms (r=0.18, p=0.000). Gender emerged as an important covariate, with males exhibiting significantly higher sleep misperception and underestimating TST (M=-0.31, SD=0.22) compared to females (M=-0.18, SD=0.12).

Conclusion: Sleep misperception was not related to sleep quality, but was strongly related to anxiety symptoms in this population. In a sub-clinical young adult sample, sleep misperception is associated with anxiety but not sleep quality, and has significant gender differences. These findings contribute to sleep misperception literature with potential applications in diagnosis and treatment of insomnia and anxiety.

Support: n/a

1116

INTERACTIONS AMONG INTEROCEPTIVE SENSITIVITY AND INSOMNIA SYMPTOMS IN INDIVIDUALS WITH GENERALIZED ANXIETY DISORDER

Daffre, C.¹ Oliver, K. I.¹ Valli, B.¹ Kleckner, I.² Pace-Schott, E. F.¹ ¹Massachusetts General Hospital, Charlestown, MA, ²University of Rochester Medical Center, University of Rochester Medical Center, NY, ³Massachusetts General Hospital, Charlestown, MA.

Introduction: Abnormal interoception is believed to contribute to anxiety disorders as well as possibly to Insomnia Disorder. We therefore hypothesized that interoceptive sensitivity in persons with Generalized Anxiety Disorder (GAD) would vary with sleep quality and would differ between GAD patients with and without insomnia.

Methods: 29 subjects (86% female) who reported GAD-7 scores \geq 10 underwent psychiatric and sleep-disorders interviews and met DSM-5 criteria for GAD. Participants were assigned into an insomnia group if the Insomnia Severity Index (ISI) score exceeded 12 (N=18) or a non-insomnia group if ISI score < 11 (N=11). Participants completed approximately 2 weeks of actigraphy and sleep diaries (mean=14.09 days; SD=4.85) as well as online surveys of sleep quality including the Pittsburg Sleep Quality Index (PSQI) and interoceptive sensitivity including the Multidimensional Assessment of Interoceptive Awareness, the somatic anxiety scale of the State-Trait Inventory for Cognitive and Somatic Anxiety-Trait (STICSA-T) and the Anxiety Sensitivity Index. In addition, 28 participants underwent the Schandry heartbeat-counting task, an objective measure of interoceptive sensitivity.

Results: GAD-7 scores did not differ between insomnia and noninsomnia groups (t(18.36)=0.074, p = 0.941). However, insomniacs reported significantly higher scores on the somatic anxiety scale of the STICSA than non-insomniacs (t(17.71)=2.094, p=0.051). STICSA-T somatic anxiety scores positively correlated with greater PSQI scores (R=0.588; p=0.002) and trended negatively with actigraphy sleep-efficiency (R = -0.384; p=0.085). Non-insomniacs outperformed insomniacs in the Schandry task (t(25.9)=2.21, p=0.036). Higher Schandry scores were positively correlated with greater total sleep time (R=0.515; p=0.014) and lower ISI scores (R=-0.387; p=0.042).

Conclusion: Results suggest that GAD patients with insomnia have lesser objective interoceptive sensitivity and more subjective

somatic anxiety than those without insomnia. Greater somatic anxiety predicted poorer subjective and objective sleep quality. Hence interoceptive sensitivity may vary with sleep disturbance in GAD. **Support:** R21MH115279

1117

ASSOCIATIONS BETWEEN HEALTH BEHAVIORS AND SLEEP DURATION IN WOMEN WITH DEPRESSION

Peprah, R. Jenkins, D. Donley, T. Sexias, A. Jean-Louis, G. NYU Grossman School of Medicine, New York, NY.

Introduction: Sleep duration can have important effects on health. Long and short sleep has been associated with negative health outcomes in women. Depression may aggravate an already impaired sleep quality. This study explored associations between sleep duration and depression in pregnant women.

Methods: We analyzed data for adult women (n=9,372) from the 2017 and 2018 National Health Interview Survey (NHIS), which is a nationally representative study of the US civilian noninstitutionalized population. Sleep was categorized by short (≤ 6 hrs), normal/healthy (7-8 hrs), and long (≥ 9 hrs) sleep. Using STATA 15.0 for Windows, we report weighted frequencies and Chi-and square tests. Alpha of 0.05 was used for all significance levels.

Results: Of the sample, 81.7% of the women were White, 10.6% were Black and 7.7% were other minorities. The mean age was 51.4 ± 18.3 . We found that the proportion of women who reported short sleep increased with age (p<0.000). Current drinkers (37%) had higher numbers of normal sleep than those who were former drinkers or abstainers (p<0.000). With respect to BMI, more obese women were short and sleepers (17% and 4% respectively), but women with normal BMI (19%) were normal sleepers (p<0.000). In short sleepers, more women had trouble falling asleep (13%) and staying asleep (17%), but reported not using medication and never feeling rested. Similar results were found for long sleepers. Higher proportions of normal sleepers reported not have trouble falling asleep (27%), staying asleep (26%), or using medication for sleep (40%) (p<0.000). However, of those who reported normal sleep, greater frequencies of working up feeling rested occurred only 3-6 times in the past week (p < 0.000).

Conclusion: In this study, women with depression self-reported more normal sleep duration. This finding is inconsistent with previous research. Whether this association is causal and what pathways explain this association is unknown.

Support: This study was supported by funding from the NIH: R01MD007716, R01HL142066, K01HL135452, and K07AG052685.

1118

EXAMINING THE ROLE OF SERUM AND EXOSOMAL **BIOMARKERS IN SYMPTOMS OF FATIGUE AND** DAYTIME SLEEPINESS FOLLOWING TRAUMATIC BRAIN **INJURY**

Leete, J. J.¹ Pattinson, C. L.¹ Guedes, V. A.¹ Lai, C.¹ Devoto, C.¹ van der Merwe, A.^{2,3,4} Lippa, S.^{5,6} Shahim, P.^{2,4} Moore, B. E.^{2,3,7,4} Chan, L.^{2,4} Gill, J.^{1,4}

¹National Institute of Nursing Research, National Institutes of Health, Bethesda, MD, ²Department of Rehabilitation Medicine, National Institutes of Health, Bethesda, MD, ³Henry M. Jackson Foundation, Bethesda, MD, ⁴Center for Neuroscience and Regenerative Medicine, Bethesda, MD, ⁵Defense and Veterans Brain Injury Center, Walter Reed National Military Medical Center, Bethesda, MD, ⁶National Intrepid Center of Excellence, Walter Reed National Military Medical Center, Bethesda, MD, ⁷Uniformed Services University for the Health Sciences, Bethesda, MD.

Introduction: Fatigue and daytime sleepiness are two of the most common chronic symptoms reported after traumatic brain injury (TBI). However, there is limited understanding of the pathophysiological mechanisms following TBI that result in these symptoms. Previous research has observed elevations in peripheral blood levels of proteins in TBI patients versus controls, including neurofilament light chain (NFL)-predominantly expressed in long myelinated subcortical axons-and glial fibrillary acidic protein (GFAP)predominantly expressed in reactive astrocytes responding to central nervous system injuries. This study examines the relationship between serum and exosomal NFL and GFAP, and symptoms of fatigue and daytime sleepiness in TBI patients 1-year after injury. Methods: Sixty-seven patients with TBIs ranging from mild to severe were included in this study. Blood samples were collected from all participants 1-year post TBI, with concentrations of GFAP and NFL measured in serum and exosomes using Single Molecule Array technology (Simoa), an ultrasensitive assay. Participants reported fatigue using the Fatigue Severity Scale (FSS), and daytime sleepiness using the Epworth Sleepiness Scale (ESS).

Results: A linear regression model of fatigue symptoms and exosomal NFL controlling for age revealed that fatigue was negatively associated with exosomal NFL concentrations $(\beta = -.317, p = .041, \eta p 2 = -.343)$ and accounted for 20.2% of the change in NFL. Serum NFL concentrations were not associated with fatigue, nor were GFAP serum or exosomes. No significant associations were found between NFL, GFAP, and daytime sleepiness.

Conclusion: Our findings suggest that exosomal NFL may be related to mechanisms underlying TBI-related fatigue and the potential of NFL as a biomarker of fatigue. To our knowledge, this study is the first to examine the relationship between post-TBI NFL levels and fatigue symptoms. Further investigation into serum and exosome biomarkers of TBI-related fatigue and daytime sleepiness is warranted.

Support: National Institutes of Health and Center for Neuroscience and Regenerative Medicine

1119

SUPERIOR TEST-RETEST RELIABILITY OF A COMPUTERIZED COGNITIVE ASSESSMENT VS SLUMS **DURING AN 18-MONTH LONGITUDINAL STUDY**

Andrefsky, J. C.¹ Cahn-Hidalgo, D.² Benabou, R.³

¹University Hospitals, Parma Medical Center, Parma, OH, ²Private Practice, Rochester, NY, ³Cognivue, Inc., Victor, NY,

Introduction: Chronic sleep deprivation has been associated with cognitive impairment (CI) in multiple studies. Cognivue is an FDA-cleared computerized testing tool that provides adaptive psychophysic evaluation of cognitive functions (CF). A reliable objective method to evaluate CF in patients with sleep disorders could be helpful to optimize long-term treatment. In an FDA clearance study, Cognivue demonstrated good agreement with St. Louis University Mental Status (SLUMS) and other neuropsychological tests, and superior test re-test reliability compared to SLUMS across 2 sessions, 1 to 2 weeks apart (Cognivue regression fit: $R^2 = 0.81$, r = 0.90); SLUMS regression fit: $R^2 = 0.67$, r = 0.82). Methods: 238 subjects from the FDA clearance study enrolled in the longitudinal study. They underwent Cognivue test and SLUMS at 3 sessions over 18 months (6, 12, 18 months post-FDA study). An analysis of rank linear regression test-retest reliability was performed for both tests.

Results: Among these 238 patients, Cognivue demonstrated similar linear regression scores across comparisons (test session 1&2: regression fit: $R^2 = 0.76$; r = 0.87; test session 1&3: regression fit: $R^2 = 0.72$; r = 0.85; test session 1&4: regression fit: $R^2 = 0.73$; r = 0.86). The SLUMS test demonstrated greater variability in regression scores across test sessions (test session 1&2: regression fit: $R^2 = 0.63$; r = 0.79; test session 1&3: regression fit: $R^2 = 0.43 r = 0.65$; test session 1&4: regression fit: $R^2 =$ 0.64; r = 0.80).

Conclusion: Cognivue demonstrated maintained superior test re-test reliability compared to SLUMS over a period of 18 months after the FDA clearance study. With that, Cognivue could be beneficial in detecting early stages of multi-domain CI in patients with sleep disorders, providing an opportunity for early intervention strategies and follow-up over time to improve patient outcomes. Support: Cognivue, Inc.

1120

INVERSE U-CURVE ASSOCIATION BETWEEN SLEEP DURATION AND COGNITIVE PERFORMANCE AMONG PATIENTS WITH DEMENTIA: FINDINGS FROM THE **CRETAN AGING COHORT**

Basta, M.^{1,2} SIMOS, P.¹ KOUTENTAKI, E.¹ ZAGANAS, I.³ TZIRAKI, S.¹ BELOGIANNI, C.¹ PANAGIOTAKIS, S.⁴ VGONTZAS, A.^{1,2}

¹Department of Psychiatry, University Hospital of Heraklion, Crete, Voutes - Heraklion, GREECE, ²Sleep Research and Treatment Center, Department of Psychiatry, Penn State College of Medicine, Hershey, PA, ³Department of Neurology, University Hospital of Heraklion, Crete, Voutes - Heraklion, GREECE, ⁴Department of Medicine, University Hospital of Heraklion, Crete, Voutes - Heraklion, GREECE.

Introduction: Previous research reports an inverse U-curve association between subjective sleep duration and cognition in elderly, while findings on objective sleep duration are inconsistent. Only one study found weak association between objective short sleep duration and cognition, mainly driven by demented elders. Our aim was to examine the non-linear associations between objective sleep duration and cognitive performance among community-dwelling patients with dementia.

Methods: A sub-sample of 46 patients with mild-to-moderate dementia(AD) [mean age: 80.3 (SD=5.6) years, 40% males] and 85 cognitively intact controls(NI) [mean age: 73.0 (SD=7.4) years, 37% males], were recruited from a large, population-based cohort [Cretan Aging Cohort] in the island of Crete, Greece of 3,140 older adults (\geq 60yrs). All participants underwent medical history/physical examination, extensive neuropsychiatric and neuropsychological evaluation, and 3-day 24-h actigraphy. Comparisons between AD and NI participants on sleep parameters and neuropsychological performance were made using ANOVA controlling for demographics. Associations between 24-TST, and age- and education-adjusted cognitive scores and Independent Activity of Daily Living Scale (IADL) scores were assessed using hierarchical, non-linear, regression models, controlling for confounders.

Results: Dementia patients had significantly longer 24-h total sleep time (24h-TST) (491.2 \pm 107.1 min vs. 444.6 \pm 88.5 min, respectively, p=0.027), as well as lower cognitive/IADL sores as compared to the NI group. Significant associations between objective sleep and various cognitive /IADL scores were found only among patients with dementia. Specifically, we found a negative curvilinear association between 24-h TST and IADL, episodic memory indices (AVLT Retention, autobiographic memory) and visuomotor coordination speed (Trail Making Test, Part A).

Conclusion: Our study showed an inverse U-curve association between objective sleep duration and daily function, memory, and executive function in patients with dementia. Possibly, sleep loss may lead to cognitive impairment, whereas, prolonged sleep may be an indicator of worse cognitive performance among patients with dementia.

Support: National Strategic Reference Framework (ESPA) 2007-2013, Program: THALES, University of Crete, title: "A multidisciplinary network for the study of Alzheimer's Disease" (Grant: MIS 377299).

1121

NONMOTOR SYMPTOMS AFFECT SLEEP QUALITY IN EARLY-STAGE PARKINSON'S DISEASE PATIENTS WITH OR WITHOUT COGNITIVE DYSFUNCTION

Zhang, L. Zhu, J.

Nanjing Brain Hospital Affiliated to Nanjing Medical University, Nanjing, CHINA.

Introduction: Parkinson's disease (PD) patients frequently present with sleep disorders. This study was designed to assess the impact of nonmotor symptoms (NMSs) on subjective sleep quality in early-stage PD patients with and without cognitive dysfunction.

Methods: A sample of 389 early-stage PD patients (Hoehn and Yahr score ≤ 2.5 , duration ≤ 5 years) was recruited for the present study. The Non-Motor Symptoms Questionnaire (NMS-Quest) was used to screen for global NMSs. Depressive symptoms were assessed using the Hamilton Rating Scale for Depression (HAMD). PD motor symptoms were measured with the Unified PD Rating Scale (UPDRS), part III. The Montreal Cognitive Assessment (MoCA) was used to evaluate global cognitive status, and the PD Sleep Scale (PDSS) was used to quantify sleep quality. Logistic regression models were built to identify factors associated with sleep disturbances.

Results: In our sample, approximately one-quarter of the PD patients suffered from sleep disturbances (23.7%). Our results also confirmed the high prevalence of cognitive dysfunction in

patients with PD (39.8%). In total, the patients who suffered from NMSs, such as depressive symptoms, anxiety symptoms, urinary tract symptoms and hallucinations/delusions, had poorer sleep quality. Better cognition may protect against sleep disorders. In patients with cognitive dysfunction, the NMS-Hallucinations/ delusions score was the most important risk factor for sleep disorders. In patients without cognitive dysfunction, NMSs such as anxiety and cognition and medication were related to sleep disorder.

Conclusion: NMSs in early-stage PD are highly associated with and are determinants of subjective sleep quality. Future studies should focus on elucidating the pathophysiology of these symptoms. **Support:** Special Funds of the Jiangsu Provincial Key Research and Development Projects (grant No. BE2018610)

1122

SLEEP ARCHITECTURE AND LEG MOVEMENT ACTIVITY DURING SLEEP IN PATIENTS WITH MULTIPLE SCLEROSIS

Castelnovo, A.^{1,2} Ferri, R.³ Tanioka, K.⁴ Tachibana, N.⁵ Carelli, C.¹ Riccitelli, G.^{6,7} Zecca, C.^{7,2} Gobbi, C.^{7,2} Manconi, M.^{1,2} ¹Sleep Center, Neurocenter of the Southern Switzerland, Regional Hospital (EOC) of Lugano, Lugano, SWITZERLAND, ²Faculty of Biomedical Sciences, Università della Svizzera Italiana, Lugano, SWITZERLAND, ³Oasi Research Institute-IRCCS, Troina, ITALY, ⁴Department of Neurology, Osaka City General Hospital, Osaka, JAPAN, ⁵Division of Sleep Medicine, Kansai Electric Power Medical Research Institute, Osaka, JAPAN, ⁶Neuroimaging Research Unit, Institute of Experimental Neurology, Division of Neuroscience, Istituto di Ricovero e Cura a Carattere Scientifico San Raffaele Scientific Institute, Milan, ITALY, ⁷Multiple Sclerosis Center, Neurocenter of the Southern Switzerland, Regional Hospital (EOC) of Lugano, Lugano, SWITZERLAND.

Introduction: Although sleep in patients with Multiple Sclerosis (MS) has been investigated in several studies using subjective measures, objective sleep data collected using polysomnography (PSG) are still scanty and often divergent. We herein present the largest study to date evaluating sleep architecture and total leg movement activity during sleep (LMS) in patients with MS.

Methods: We collected PSG recordings from 80 patients affected by multiple sclerosis (MS, 48.1±10.61yo 67.5% females), and 60 age and gender matched healthy control subjects (HC, 48.5±17.20 yo, 56% females). Group differences were computed using non-parametric statistics for all traditional sleep architecture parameters, LMS, short-interval (SILMS), periodic (PLMS), isolated LMS (ISOLMS) indices and duration, inter-movement interval (IMI) graphs and time-of-night distribution of LMS.

Results: Patients with MS showed a significantly decreased total sleep period, an increased number of awakenings and stages shifts per hour of sleep, and an increased representation of stage 1 (min and %) compared to the HC group; 26 (32.5%) MS patients had PLMS \geq 15/hour versus 8 (13.3%) HC subjects. On average, the comparison between MS and HC groups yielded significant results in terms of an increase in LMS, PLMS, SILMS and ISOLMS indices but not durations. Moreover, MS patients displayed a higher periodicity index, an increased PLMS activity at all inter-movement intervals considered and their PLMS time-of-night distribution revealed that the PLMS increase was stable over the course of the night.

Conclusion: Sleep continuity is significantly impaired in patients with MS. Moreover, MS patients also an increased total LMS activity, including PLMS, which may contribute to disrupt sleep continuity. A disinhibition of lower spinal network due to cervical or supraspinal MS lesions might be implicated in the mechanisms underlying this latter finding.

Support: The Employer Department of Neurology, Regional Hospital Lugano (EOC), Lugano, Switzerland receives financial support from Teva, Merck Serono, Biogen Idec, Bayer Schering, Genzyme, Roche and Novartis. The submitted work is not related to these agreements.

1123

SLEEP, CAREGIVER BURDEN, AND LIFE SATISFACTION IN PARKINSON'S DISEASE CAREGIVERS FROM MEXICO AND THE US

Perez, E.¹ Perrin, P. B.¹ Lageman, S. K.¹ Villaseñor, T.² Dzierzewski, J. M.¹

¹Virginia Commonwealth University, Richmond, VA, ²University of Guadalajara, Guadalajara, Jalisco, MEXICO.

Introduction: Sleep problems are frequently reported by caregivers of individuals with Parkinson's disease (PD) and are associated with greater caregiver burden and poorer caregiver life satisfaction. The primary aim of this study was to examine the pattern of associations among PD patient and caregiver sleep problems, caregiver burden, and life satisfaction in PD caregivers. A secondary aim was to assess whether the pattern of associations differed between Mexican and US caregivers.

Methods: Secondary analyses were performed on data obtained from 253 caregivers (age M=59.92, SD=14.66). A composite score was produced for caregiver and patient sleep problems. The Zarit Burden Interview (ZBI) and Satisfaction with Life Scale (SWLS) measured caregiver burden and life satisfaction, respectively. A structural equation model (SEM) was developed to examine the pattern of associations among sleep problems, caregiver burden, and life satisfaction. An invariance design was employed to assess differences in the associations between Mexican and US caregivers.

Results: Fit indices suggested the SEM fit the data adequately. Path coefficients among all variables were significant ($p \le .005$), except between patient sleep problems and life satisfaction. Three significant indirect effects were found: caregiver sleep problems were negatively associated with life satisfaction via caregiver burden (p=.003); patient sleep problems were positively related to caregiver burden via caregiver sleep problems (p=.005); and life satisfaction via caregiver burden and caregiver sleep problems (p=.002). Despite noninvariance of error terms for the ZBI (z=2.92) and SWLS (z=3.37) between Mexican and US caregivers, the model was generally invariant across countries.

Conclusion: Patient sleep problems were associated with caregiver sleep problems, leading to increased burden in caregivers and poorer life satisfaction. The findings highlight a potential opportunity for empirically-supported sleep interventions in both individuals with PD and their caregivers which may initiate a cascade of salubrious effects on caregiver burden and overall life satisfaction.

Support: Dr. Dzierzewski's effort on this project was supported in part by the National Institute on Aging (K23AG049955 to J.M.D).

1124

CENTRAL SLEEP APNEA AND TRAUMATIC BRAIN INJURY: A NIDILRR AND VA TBI MODEL SYSTEM STUDY

Gulati, G.¹ Schwartz, D. J.^{1,2} Nallu, S.¹ Bell, K.³ Wittine, L.¹ Fann, J. R.⁴ Nakase-Richardson, R.^{1,5,6}

¹University of South Florida College of Medicine, Tampa, FL, ²Medicine, Tampa, FL, ³PMRS, University of Texas Southwestern, Dallas, TX, ⁴Psychiatry, University of Washington, Seattle, WA, ⁵MHBS, Tampa, FL, ⁶4DVBIC, James A. Haley Veterans Affairs Medical Center, Tampa, FL.

Introduction: Sleep-related breathing disorders are common after TBI. To date, two single site studies have reported divergent findings in post-TBI patients with one reporting predominantly obstructive sleep apnea (OSA, Holcomb et al., 2016) and the other central sleep apnea (CSA, Webster and Bell, 1998). The purpose of this analysis is to explore prevalence, demographics, and injury characteristics of patients with a clinical diagnosis of CSA in a recently-completed multicenter comparative-effectiveness trial during inpatient rehabilitation following moderate to severe TBI.

Methods: Participants in a six-center diagnostic comparative effectiveness trial underwent Level-1 polysomnography (PSG) during inpatient rehabilitation for TBI. Studies were scored at a centralized scoring center by one of two certified PSG technicians with final interpretation by a board-certified sleep medicine physician.

Results: 21 of 248 (8.5%) participants evidenced elevated CSA indices >5. Predominant CSA was rare (n=3 [1.2%], age range: 36-59; 100% male; 33-52 days post-TBI). One participant was on opioid, anti-depressant and antiepileptic drugs, one was on an antiepileptic, and another was on an opioid. PAP therapy was not initiated during PSG thus there was no treatment-emergent CSA. All had a central apnea-hypopnea index (AHI) in the moderate to severe range (29-49). Two out of the three had a GCS <8 and one participant had a GCS of 14.

Conclusion: In this multi-center clinical trial, predominant CSA was rare. The common practice of reducing polypharmacy in order to minimize sedation and optimize mental status in specialized inpatient brain-injury rehabilitation programs may contribute to the low CSA incidence in this cohort. Attention to medication side-effects and their influence on sleep-related breathing should be routinely considered.

Support: PCORI (CER-1511-33005), GDHS (W91YTZ-13-C-0015) for DVBIC, NIDILRR (90DPTB0008-03-00; 90DPTB0013-01-00).

1125

WITHDRAWN

1126

RELATIONSHIP BETWEEN HEADACHE AND SLEEP: A LONGITUDINAL STUDY FROM THE POPULATION OF SAO PAULO CITY (BRAZIL)

Lucchesi, L. M. Tempaku, P. F. Smith, A. A. Togeiro, S. Hachul, H. Andersen, M. Tufik, S. Poyares, D. Universidade Federal de Sao Paulo, Sao Paulo, BRAZIL.

Introduction: The complaint of nocturnal awakening with headache (NAH), was prevalent (8.4%) in the São Paulo population and was associated with sleep disturbances, as demonstrated in a study conducted in 2007 (EPISONO). Indeed, this relationship between sleep and headache is well documented in the literature. Objective: To assess the incidence and evolution of NAH and to associate sleep-related variables in an eight-year prospective study. **Methods:** From 1042 volunteers enrolled in the baseline, 712 agreed to participate in the follow-up. Questionnaires and scales were applied and polysomnography and actigraphy performed. The complaint of NAH was analyzed according to a frequency questionnaire and separated into frequent or occasional.

Results: At follow-up, 110 volunteers reported NAH, of which 82 were the same as those from the baseline, but only 38 had frequent complaints. Comparing with volunteers whose headache has become occasional, we have as a difference the insomnia severity index which is significantly higher in the group with frequent NAH (8.40 ± 5.10 vs 11.20 ± 6.40 p:0.03) and worse sleep quality as measured by the Pittsburgh questionnaire (7.25 ± 3.60 vs 10.25 ± 4.60 p:0.002). In addition, these volunteers had higher anxiety (10.40 ± 9.30 vs $12.00\pm10:00$ p: 0.008) and depression (10.60 ± 9.90 vs $12:00\pm9.90$ p:0.005) from Beck's questionnaires and greater fatigue (4.85 ± 3.10 vs 9.75 ± 5.55 p:0.001). The associations of NAH with insomnia, nightmares, and bruxism observed in the baseline continued, but no difference was observed between those who had frequent or occasional complaints at follow-up.

Conclusion: Our study showed that NAH was highly prevalent in the Sao Paulo population in both the baseline and follow-up studies, but this frequency had a reduction in follow-up. Volunteers who persisted with the frequent complaint showed greater severity of insomnia, higher anxiety and depression and greater fatigue.

Support: Associação Fundo de Incentivo à Pesquisa (AFIP) and São Paulo Research Foundation (FAPESP)

1127

UTILITY OF QUANTITATIVE EEG DURING SLEEP AS A POTENTIAL BIOMARKER OF LEWY BODY DISEASE PROGRESSION

Matar, E.¹ Ehgoetz Martens, K. A.² Grunstein, R. R.¹ D'Rozario, A.¹ Lewis, S. J.¹

¹Faculty of Medicine and Health, University of Sydney, Sydney, AUSTRALIA, ²University of Sydney, Sydney, AUSTRALIA.

Introduction: Sleep disturbances are common among patients with Lewy body disorders. Idiopathic REM sleep behavior disorder (iRBD) has been identified as a prodromal Lewy body condition with a significantly increased risk of conversion to either Parkinson's disease (PD) or Dementia with Lewy Bodies (DLB). Pathological involvement of thalamic and brainstem structures involved in sleep regulation has been reported in these disorders, especially in later stages. We hypothesized that progression along the Lewy body disease spectrum may be associated with unique changes in spindle density and EEG power spectra during sleep reflecting involvement of these deep brain structures.

Methods: A cross-sectional design was used. 9 polysomnography confirmed iRBD, 18 early PD, 23 DLB and 13 controls underwent overnight polysomnography, neurological and neuropsychological assessment. Power spectrum analysis during NREM and REM sleep was undertaken using a previously validated quantitative EEG algorithm and compared between groups. Following artefact and outlier removal, results were analysed using the Cz derivation. Groups were statistically compared with a non-parametric Jonckheree-Terpstra test for ordered alternatives, controlling for age and sex.

Results: We found a significant and ordered reduction in power in the spindle frequency band (12-15 Hz) in NREM sleep across the Lewy body disease spectrum compared to controls (Controls > iRBD > early PD > DLB; $T_{JT} = 521.00$, z = -2.902. p<0.001). In REM sleep we found a shift in power to slower frequencies with increased power in the theta (4.5-8 Hz) band in order of disease severity (DLB > early PD > iRBD > Controls; $T_{JT} = 950.00$, z = 2.253. p=0.024). No differences were found across the other frequency bands in NREM or REM sleep.

Conclusion: There is a significant and progressive reduction in spindle density and corresponding slowing in REM sleep frequencies during sleep with clinical Lewy body staging. Thus, such measures have the potential to be useful biomarkers of progression towards Lewy body dementia from prodromal stages.

Support: This work was supported by a NHMRC Dementia Team Grant (#1095127), the NHMRC Postgraduate Scholarship and the University of Sydney Research Excellence Initiative 2020 grant.

1128

SLEEP APNEA AND PERIODIC LIMB MOVEMENTS ARE HIGHLY PREVALENT IN PATIENTS WITH MULTIPLE SCLEROSIS

Palotai, M. Weiner, H. L. Chitnis, T. Duffy, J. F. Guttmann, C. R. Brigham & Women's Hospital/Harvard Medical School, Boston, MA.

Introduction: The pathogenesis of multiple sclerosis (MS)related fatigue is multi-factorial, including neurogenic, inflammatory, endocrine, metabolic, mood, as well as sleep disorder-related mechanisms. The confounding effect of sleep disorders on the association between fatigue and neurodegenerative changes in the brain has not been investigated. Our objectives were to assess the prevalence of sleep apnea and periodic limb movements in the framework of a prospective study which investigates the neurogenic causation of treatment-resistant fatigue in MS.

Methods: MS patients enrolled in a National MS Society-funded prospective study (grant identifier RG-1501-03141) underwent a onenight at-home sleep test (HST) using a NOX T3 portable monitor. HST recordings were scored by a registered polysomnographic technologist. Respiratory Event (REI) and Periodic Limb Movement (PLMI) Indices were calculated for each patient.

Results: Out of 36 patients, 7 (20%) had mild (REI=5-14), 1 (3%) had moderate (REI=15-29), and 1 (3%) had severe sleepdisordered breathing (REI \ge 30). Fourteen (42%) of the patients had mild (PLMI=5-24), 4 (11%) had moderate (PLMI=25-49), and 7 (19%) had severe periodic limb movements (PLMI \ge 50). Overall, 81% of the patients had at least mild sleep-disordered breathing and/or periodic limb movements.

Conclusion: Sleep abnormalities (i.e., sleep apnea and periodic limb movements) are highly prevalent in patients with MS. We plan to compare the MRI exams of subgroups of MS patients with fatigue, to test the hypothesis that fronto-striatal circuitry is more affected by lesions in patients without sleep apnea compared to those with sleep apnea.

Support: This investigation was supported by a grant from the National Multiple Sclerosis Society (grant identifier RG-1501-03141). The home sleep test equipment was provided by a DURIP grant from the Office of Naval Research (grant N00014-15-1-2917).

1129

SEX DEPENDENT EFFECTS ON SLEEP ARCHITECTURE AFTER TRAUMATIC BRAIN INJURY: IMPACT ON OUTCOME

Griesbach, G. S.¹ Robinson, S. E.² Howell, S.² ¹Centre for Neuro Skills, Encino, CA, ²Centre for Neuro Skills, Irving, TX.

Introduction: Traumatic Brain Injury (TBI) is frequently associated with problems with sleep and diurnal somnolence. After determining if subjective somnolence was associated with sleep disturbances, we investigated if alterations in sleep architecture were associated with cognitive, social and emotional health in a sex dependent manner. For patients receiving positive airway pressure (PAP) treatment, we determined if lack of compliance contributed to cognitive and quality of life issues.

Methods: Adult TBI subjects (n=57) were assessed via overnight polysomnography. Mean age was 41 years and mean TBI chronicity was 2.5 years. Overall level of disability was determined by the Mayo Portland Inventory II. Sleep measures included slow wave sleep (SWS), REM latency, percent time in all sleep stages, apnea/hypopnea index, wake after sleep onset (WASO), and arousal index. Outcome measures were the California Verbal Learning Test (CVLT), Montreal Cognitive Assessment (MoCA), Trails A and B, Beck Depression Inventory, and Neuro-QoL.

Results: No sex effects for reporting somnolence were found. Besides being associated with increased subjective anxiety and stigma, somnolence was associated with increased arousals, decreases in SWS and higher incidence of REM AHI. WASO and number of arousals had a negative impact on the amount of SWS and sleep efficiency. Men spent significantly more time in REM sleep, which was correlated with higher scores on the MoCA and CVLT. Women showed more disability. Longer latencies to SWS were associated with increased CVLT performance. AHI was associated with increases in emotional/behavioral dyscontrol, fatigue and self-reported sleep disturbance. All effects were statistically significant.

Conclusion: Female TBI patients show significant impairments in REM sleep, which may impact learning and memory. Sleep disturbances were associated with poorer cognitive performance and may ultimately affect outcome, as indicated by lower scores on quality of life measures.

Support: Centre for Neuro Skills

1130

INSOMNIA SHORT SLEEP PHENOTYPE IS ASSOCIATED WITH FRAILTY IN PATIENTS WITH MILD COGNITIVE IMPAIRMENT (MCI)

BASTA, M.¹ VGONTZAS, A.^{1,2} KOUTENTAKI, E.¹ ZAGANAS, I.³ FERNANDEZ-MENDOZA, J.² BELOGIANNI, C.¹ PANAGIOTAKIS, S.⁴ PUZINO, K.² SIMOS, P.¹

¹Department of Psychiatry, University Hospital of Heraklion, Voutes - Heraklion, Crete, GREECE, ²Sleep Research and Treatment Center, Department of Psychiatry, Penn State College of Medicine, Hershey, PA, ³Department of Neurology, University Hospital of Heraklion, Voutes-Heraklion, Crete, GREECE, ⁴Department of Medicine, University Hospital of Heraklion, Voutes-Heraklion, Crete, GREECE. **Introduction:** Insomnia short sleep phenotype is associated with cardiometabolic morbidity and mortality and neuropsychological impairment. In elderly untreated insomnia is associated with worse cognitive performance. The goal of the study was to examine the association between insomnia, objective sleep duration and physical and mental health in elderly patients with Mild Cognitive Impairment (MCI).

Methods: A sub-sample of 105 patients with MCI (mean age: 75.9 years, males 36%) were recruited from a large populationbased cohort (Cretan Aging Cohort) in the island of Crete, Greece of 3,140 elders ($\geq 60yrs$). All participants underwent a complete medical history/ physical examination, extensive neuropsychiatric and neuropsychological evaluation and 3-day 24hr actigraphy. Insomnia was defined based on a question "do you have insomnia for more than a year". Frailty was assessed with the Simple "Frail" Questionnaire Screening Tool. Comparisons between patients with insomnia and without insomnia were made using ANOVA controlling for age, gender and BMI.

Results: MCI patients with insomnia (n=23) compared to those without insomnia (n=82), had significantly shorter objective total sleep time (TST: 377 vs. 410 min, p=0.05) and significantly higher scores on the Geriatric Depression Scale and the Hospital Anxiety Scale (both p <0.001). Furthermore, total frailty score, as well as scores in individual items, were significantly lower in MCI patients with insomnia (p<0.01). This association remained significant after controlling for demographics, depression and anxiety. Finally, there was a statistical trend of association between insomnia and hypertension (p= 0.1).

Conclusion: In MCI patients, insomnia is associated with objective short sleep duration, and frailty. Improving insomnia and lengthening sleep duration may decrease frailty, a major problem associated with morbidity, disability and mortality in elders with cognitive decline.

Support: National Strategic Reference Framework (ESPA) 2007-2013, Program: THALES, University of Crete, title: "A multidisciplinary network for the study of Alzheimer's Disease" (Grant: MIS 377299).

1131

OBSTRUCTIVE SLEEP APNEA TREATMENT AND DEMENTIA RISK IN OLDER ADULTS

Dunietz, G. L.¹ Chervin, R. D.¹ Burke, J. F.¹ Conceicao, A. S.¹ Braley, T. J.¹

¹University of Michigan, Ann Arbor, MI, ²University of Michigan, Ann Arbor, MI.

Introduction: Obstructive Sleep Apnea (OSA) has been linked to dementia and Alzheimer's Disease (AD), as well as pre-dementia. The potential benefits of OSA *treatment* on cognitive performance are inconclusive. Further, the impact of OSA treatment on the development of neurodegenerative disorders has not been sufficiently studied. This study examined associations between Positive Airway Pressure (PAP) therapy and incident diagnosis of pre-dementia (MCI), dementia (DNOS), or AD in a population-level sample of older adults.

Methods: Using a random 5% sample from Medicare claims data (persons age 65 and older), individuals with an ICD-9 diagnosis code for OSA prior to 2011 (n=53,321) were selected for analyses. Over the survey period (2011-2013), we further identified persons newly diagnosed with MCI (n=443), DNOS (n=378), or AD (n=1,057). We also identified individual HCPCS claims for PAP

equipment as evidence of prescription, and repeated HCPCS claims for supplies as evidence of adherence to PAP treatment. Logistic regression models were used to adjust for potential confounders including age, gender, hypertension, and Parkinson's Disease that might increase risk for dementia.

Results: Seventy-eight percent of beneficiaries with OSA were prescribed PAP, and 74% showed evidence of adherent use. After adjustment for potential confounders, prescription of PAP was associated with significantly lower odds of incident AD and DNOS (OR=0.78, 95% CI: 0.69, 0.89; and OR=0.69, 95% CI: 0.55, 0.85). Lower odds of MCI, approaching statistical significance, were also observed among beneficiaries who were prescribed PAP (OR=0.82, (95% CI: 0.66, 1.02). Evidence of adherence to PAP was significantly associated with lower odds of incident AD (OR=0.65, 95% CI: 0.56, 0.76).

Conclusion: Among older individuals with OSA, PAP prescription and adherence are each associated with a significantly lower risk of incident AD or DNOS, though not MCI. Although a prospective cohort design cannot prove causality, results suggest that treatment of OSA could reduce risk of subsequent dementia.

Support: This study was supported by The American Academy of Sleep Medicine Foundation Strategic Research Award 115-SR-15 (PI Braley).

1132

PEDIATRIC SENSORY SAFE SLEEP STUDY: HOW TO REDUCE SENSORY STIMULATION AND CULTIVATE COMPLIANCE AMONG CHILDREN WITH SPECIAL NEEDS. A NEW CONCEPT

Rice, B. Albertario, C. Veler, H. Weill Cornell Medical College, New York, NY.

Introduction: Children with neurodevelopmental disabilities (NDD) frequently suffer from sleep disturbances causing further worsening of behavior, developmental and day-to-day functioning. The important tool used to assess sleep and sleep disorders is polysomnography (PSG). While PSG provides children a less invasive option, children with NDD and/or sensory disorders are often more resistant to this overwhelming and dysregulating procedure. As a result, many children in need of PSG will avoid scheduling a study, or have inconclusive results due to noncompliance. Previous research had shown a program of desensitization improves the number of successfully completed PSGs in NDD children. Our goal is to develop best practice standards to accommodate the individualized need of patients in the sleep lab, in a way that will be available and practical to any center treating NDD children.

Methods: Children with NDD require more individualized care tactic therefore, we developed staff education and a process that would help troubleshoot the possibility of patient non-compliance by reducing triggering sensations and unaccommodating technician behaviors. Our process is made up of several key steps to address the behavioral needs of patients: (1) identify patients at risk upon referral; (2) referral to a child life specialist to identify individualized preparation strategies and complete a brief behavioral questionnaire for documentation; (3) mail a preparation kit to the family with a summary of preparation techniques and procedural steps; and (4) ensure sleep technicians have been provided with thorough information to best accommodate the unique patient needs.

In addition, PSG conducted with a truncated montage to reduce the number of stimulating sensors applied to the patient and the lab is equipped with sensory toys to provide a relaxing environment.

Results: Results will be determined based on comparing the number of successfully completed overnight sleep studies for children with NDD in the years prior to intervention, to the total success rate following the implementation of the intervention process. As a secondary goal, we will gather parent satisfaction scores via morning questionnaires.

Conclusion: Research has proven across many modalities that adequate preparation and behavioral modifications produce successful encounters when working with the pediatric population.

Support: None

1133

THE RECOVERY OF SLEEP OSCILLATIONS IN ACUTE TO CHRONIC TRAUMATIC BRAIN INJURY

Sanchez, E.¹ Duclos, C.² Van Der Maren, S.¹ El-Khatib, H.¹ Arbour, C.¹ Baril, A.³ Blais, H.⁴ Carrier, J.¹ Gosselin, N.¹ ¹Université de Montréal, Montréal, QC, CANADA, ²McGill University, Montreal, QC, CANADA, ³Boston University, Boston, MA, ⁴CIUSSS-NIM, Montréal, QC, CANADA.

Introduction: Slow waves and spindles are essential oscillations occurring during NREM sleep that may be disrupted by moderate to severe traumatic brain injury (TBI). We investigated these oscillations in the acute and chronic trauma stage.

Methods: Four groups were tested with whole-night polysomnography: hospitalized patients with acute TBI (n=10, 29.7 \pm 13.8y) or severe orthopedic injuries (n=15, 39.9 \pm 17.1y), chronic TBI including 9 returning from the acute TBI group (n=43, 31.9 \pm 13.5y), and healthy controls (n=36, 30.5 \pm 12.7y). Characteristics for slow waves (density, amplitude, slope, frequency, duration) and spindles (density, amplitude, frequency, duration) were quantified over N2 and N3 sleep for the first three sleep cycles, and groups were compared using one-way ANOVAs.

Results: One-way ANOVAs showed group effects only for slow wave density (F=4.11 to 6.04, p=0.009 to 0.0008)) and spindle density (F=3.3 to 8.8, p=0.02 to 0.00003). These effects were present for the 2nd and 3rd sleep cycles, but not the 1st. More specifically, slow wave density in acute TBI was higher than in controls, and returned to normal levels in the chronic stage. Conversely, spindle density in acute TBI was lower than in controls and returned to normal levels in the chronic stage. No group difference was observed for the orthopedic group.

Conclusion: Our results suggest that immediately after a severely disruptive event such as a TBI, the brain needs additional deeper sleep to recover, resulting in more slow waves but also in less spindles. These changes are only present in the 2nd and 3rd sleep cycles, reflecting an absence of the expected dissipation of slow waves, which may suggest increased homeostatic sleep pressure due to the brain injury. Limits to interpretation include the hospital environment and medication, but the absence of changes in the orthopedic group under similar conditions emphasizes the effect of the brain injury itself.

Support: Canadian Institutes of Health Research (CIHR) and Fonds de Recherche Québec-Santé (FRQS)

1134

LIGHT EXPOSURE AT DAYTIME ON SLEEP QUALITY IN STROKE PATIENT DURING REHABILITATION

Liao, W.¹ Lin, S.² Meng, N.² Tin, H.³ Tsai, S.³ Huang, Y.³ ¹China Medical University, Taichung, TAIWAN, ²China Medical University Hospital, Taichung, TAIWAN, ³Chun Shan Medical University Hospital, Taichung, TAIWAN.

Introduction: Lights maintain the day and night rhythm to set patients' "wake-up cycle" and to stabilize their physiological functions, which may be expected to improve sleep. This study was aimed to investigate the relations between sleep quality and daytime light exposure in stroke patient during rehabilitation.

Methods: A cross-sectional study design was adopted and 120 stroke patients were recruited from rehabilitation wards of two medical centers and 116 patients completed this study. Research instruments including the Pittsburgh Sleep Quality Index, Epworth Sleepiness Scale, Sleep Log, and Somnowatch (Germany) for actigaphy sleep and light were used to collect data and urinary melatonin concentration were measured.

Results: 47.4% of the patients had poor sleep quality (PSOI>5). 74.1% had actigraphic sleep efficiency less than 85%, and 90.5% waked more than 30 minutes after sleep onset. The average exposure time at lower level light (≤149 lux) were 288.8 minutes, accounting for 48% of the day (8:00-18:00). Compared to lower light exposure group (less than 319.5 min at > 150 lux), those who exposed to higher level light (more than 319.5 min at >150 lux) had increased 52.1 minutes in actigraphic total sleep time (TST, t=-2.134, p=0.035), increased 8% in actigraphic sleep efficiency (SE, t=-2.053, p=0.042), and decreased 41.1 minutes in actigraphic wake-after-sleep-onset (WASO, t=2.209, p=0.029). Urinary melatonin concentration increased 52.7 pg/ml, but not statistically significant (t=-1.277, p=0.205). Result of multiple regression analysis showed that after controlling for age, gender, post-stroke complications, and environmental interference, time of bright light exposure significantly affected subjective sleep satisfaction (p=0.014), TST (p=0.04), SE (p=0.041), and WASO (p=0.026).

Conclusion: Increasing time of bright illumination (\geq 150 lux) during daytime may improve sleep quality. Results of this study provide empirical references for non-drug intervention to improve sleep quality in patients with stroke.

Support: This study was supported by the Ministry of Science and Technology, MOST 105-2628-B-040 -005 -MY2.

1135

LONGER AND MORE FREQUENT NAPS PREDICT INCIDENT ALZHEIMER'S DEMENTIA IN COMMUNITY-BASED OLDER ADULTS

Li, P.^{1,2} Gao, L.^{1,3} Gaba, A.¹ Yu, L.⁴ Buchman, A. S.⁴ Bennett, D. A.⁴ Hu, K.^{1,2} Leng, Y.⁵

¹Brigham and Women's Hospital, Division of Sleep and Circadian Disorders, Boston, MA, ²Harvard Medical School, Division of Sleep Medicine, Boston, MA, ³Massachusetts General Hospital Department of Anesthesia, Critical Care and Pain Medicine, Harvard Medical School, Boston, MA, ⁴Rush University Medical Center, Rush Alzheimer's Disease Center, Chicago, IL, ⁵University of California, Department of Psychiatry, San Francisco, CA.

Introduction: Excessive napping duration has been associated with cognitive decline. The effect of napping frequency is less understood, and little is known about the development of Alzheimer's

dementia associated with napping. We tested whether longer or more frequent naps in the elderly are linked to the development of incident Alzheimer's dementia.

Methods: We studied 1,180 older adults (age: 81.0 ± 7.3 [SD]) in the Rush Memory and Aging Project who have been followed for up to 14 years. Motor activities of up to 10 days were recorded at baseline to assess napping characteristics objectively. We defined daytime napping episodes as motor activity segments between 10AM and 7PM with continuous zero-activity for ≥ 10 min but <1h (to avoid off-wrist periods). Segments that were <5min apart were merged. Alzheimer's dementia diagnosis was determined using the criteria of the National Institute of Neurological and Communicative Disorders and Strone and the Alzheimer's Disease and Related Disorders Association. Cox proportional hazards models were performed to examine the associations of daily napping duration and frequency with incident AD.

Results: Of 1,180 non-demented participants at baseline (including 264 with mild cognitive impairment), 277 developed Alzheimer's dementia within 5.74 ± 3.36 years. On average, participants napped for 38.3 ± 1.0 (SE) min and 1.56 ± 0.04 (SE) times per day at baseline. After adjustment for age, sex, and education, every 30-min increase in daily napping duration was associated with a 20% increase in the risk of incident AD (95% confidence interval [CI]: 9%-31%; p=0.0002). One more nap per day was associated with a 19% increase in the risk of AD (95% CI: 8%-30%; p=0.0003). These associations remained after further adjustment for total sleep time. **Conclusion:** Longer and more frequent daytime naps predict a higher risk of incident Alzheimer's dementia. Future studies are needed to examine specific underlying mechanisms.

Support: This work was supported by NIH grants RF1AG064312, RF1AG059867, R01AG017917, and R01AG56352.

1136

POLYSOMNOGRAPHY IS FEASIBLE DURING INPATIENT TBI REHABILITATION

Drasher-Phillips, L.¹ Schwartz, D.¹ Ketchum, J.² O'Connor, D.¹ Calero, K.^{1,3} Diaz-Sein, C.¹ Wharton, L.¹ Almeida, E.² Dahdah, M.^{4,5} Bell, K.⁶ Nakase-Richardson, R.^{1,3,7} ¹James A. Haley Veterans' Hospital, Tampa, FL, ²Craig Hospital, Englewood, CO, ³Department of Internal Medicine, Morasani College of Medicine, University of South Florida, Tampa, FL, ⁴Baylor Scott & White Medical Center, Plano, TX, ⁵Baylor Scott & White Institute for Rehabilitation, Dallas, TX, ⁶Department of Physical Medicine and Rehabilitation, UT Southwestern Medical Center, Dallas, TX, ⁷Defense and Veterans Brain Injury Center, Tampa, FL.

Introduction: A recent meta-analytic report highlighted that obstructive sleep apnea was 12 times more prevalent in TBI (mixed severity) than in community-based samples. Recent studies highlight prevalent obstructive sleep apnea during acute inpatient rehabilitation which is a time of critical neural repair. Acute sleep disturbances are associated with therapy cooperation due to effects on daytime sleepiness and are associated with key rehabilitation outcomes. Given the high rates of OSA and risk for negative morbidity, this analysis sought to examine the feasibility of administering polysomnography (PSG) with EEG to diagnose sleep apnea during inpatient rehabilitation in persons with moderate to severe TBI.

Methods: This is a secondary analysis from a prospective diagnostic comparative effectiveness clinical trial (NCT03033901) that took place at six NIDILRR and one VA TBI Model System

Centers. Participants were included if they met the TBI Model System case definition and slept at least 2 hours per night prior to PSG. PSG was conducted following AASM procedures in the participant's hospital bed on the inpatient rehabilitation unit. Studies were scored by RPSGT staff and interpreted by a board certified sleep medicine physician at a centralized sleep scoring center in Tampa, FL.

Results: Of 896 potential TBI participants, 449 met initial eligibility and 345 consented for further screening; a final sample of 263 (76%) completed PSG during hospitalization. Primary reasons for not completing PSG included early discharge or medical instability (n=59) and last-minute withdrawal of consent for PSG (n=23). Of the 263 participants who completed PSG, 3 were excluded from analysis due to technical issues and 12 were excluded as the total sleep time (TST) was less than 120 minutes. Of the 248, 85.5% of the PSGs were rated as interpretable/scoreable by RPSGT and sleep physicians.

Conclusion: For a majority of participants, polysomnography is feasible during inpatient rehabilitation. Participants with shorter lengths of stay, medical instability, prolonged agitation may require polysomnography follow-up after discharge.

Support: Supported by PCORI (CER-1511-33005), VA TBIMS, DVBIC with subcontract from GDIT/GDHS (W91YTZ-13-C-0015, HT0014-19-C-0004), and NIDILRR (90DPTB00070, 90DPTB00130100, 90DPTB0008, 90DPT8000402, 90DPTB0001).

1137

SLEEP DURATION, PHYSICAL ACTIVITY AND COGNITIVE DECLINE IN CHINESE OLDER ADULTS: FINDINGS FROM THE CHARLS

Li, J.¹ Alfini, A. J.² Yu, F.³ Schrack, J. A.² Cotter, V.¹ Taylor, J. L.¹ Spira, A. P.²

¹Johns Hopkins University School of Nursing, Baltimore, MD, ²Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, ³University of Minnesota School of Nursing, Minneapolis, MN.

Introduction: Lack of physical activity and disturbed sleep have been linked to older adult's poor cognitive outcomes; however, little is unknown how they interact to affect cognition long-term. The purpose of this study was to examine the association of baseline sleep duration and physical activity (PA) with change in cognition independently and interactively over four years.

Methods: The sample included 1126 community-dwelling older adults aged 60+ (mean age 67.1±5.9 years, 51% female) from the 2011 baseline and 2015 follow-up data of the China Health and Retirement Longitudinal Study (CHARLS). All variables were assessed through interviews. Sleep duration was measured with hours per 30-minute interval and categorized as very-short (<5h), short (5-6.5h), normal (7-8.5h), and long (≥9h). PA was calculated based on PA intensity, duration, and number of days. Cognition was a composite score of mental capacity, episodic memory, and visuospatial abilities. Data were analyzed using multiple regression (primary outcome: change in cognition; main independent variables: baseline sleep, PA, and sleep PA interaction).

Results: At baseline, 19% of participants had very-short sleep duration, 34.4% had short sleep, 39.2% had normal sleep, and 7.2% had long sleep. At follow-up, 57.5% of participants experienced cognitive decline (-3.5 \pm 2.5). After controlling for age, gender, education, region, body mass index, smoking, drinking, number of chronic conditions, pain, depression, and cognition at baseline, compared to participants reporting 7-8.5h sleep, those with \geq 9h sleep had significantly greater decline in cognition [β =-1.4, 95% CI=2.4, -0.4], while those with <5h sleep [β =-0.5, 95% CI=-1.2, 0.2] and 5-6.5h sleep did not [β =-0.1, 95% CI=-0.7, 0.5]. PA was neither associated with cognitive decline, nor moderated the relationship between sleep duration and cognitive decline.

Conclusion: Long sleep might be a marker of cognitive decline in older adults. Prospective analysis, using objectively measured PA and sleep should be conducted to further examine these associations.

Support: National Institute of Nursing Research R00NR016484

1138

OSA IN A PATIENT WITH PFO, IS IT THE "PERFECT STORM" FOR A STROKE? A CASE SERIES OF 3 PATIENTS WITH STROKE AT YOUNG AGE, DUE TO PFO AND COMORBID OSA.

Wong, J. Gupta, D. Nadhim, A.

JFK Medical Center Neuroscience Institute, Edison, NJ.

Introduction: Approximately 25% of all strokes are cryptogenic in nature, and about 50% of all young patients with cryptogenic stroke are found to have patent foramen ovale (PFO), which is known to be the most common cause of right-to-left shunting (RTLS). Studies have shown a 2.2-fold increase in right-to-left shunt in patients with OSA and PFO, compared to patients with PFO alone. Hence, increasing the risk of cardio-embolic stroke in such patients. We present a case series of 3 patients with PFO that had embolic stroke at a young age and were found to have comorbid OSA, the likely exacerbating factor of a RTLS.

Methods: A 38-year old female, a 59-year old male, and a 27-year old female presented with stroke-like symptoms without clear vascular pathology. Upon further work-up, all were found to have PFO with RTLS. Subsequently, these patients were referred for sleep evaluation, and each one was found to severe REM-related OSA with prominent oxygen desaturations. All three patients were started on PAP therapy for control of their OSA. Two out of the 3 patients opted for PFO closure, and the 3rd patient opted for antiplatelet therapy alone. He has had no recurrence of TIA/stroke after 12 years so far.

Results: These cases illustrate a correlation between right-to-left shunting and severe REM-related OSA, through possible elevation of right-sided pressure due to nocturnal desaturations/hypoxemia. Hence, it is worth consideration that the increased right-sided pressure induced by apneic events in sleep may be a potential exacerbating factor in producing stroke-like symptoms sooner in patients with PFO than in patients with PFO who are without OSA.

Conclusion: It may be beneficial to assess young patients with stroke due to PFO, for comorbid OSA as a cause of the RTLS. This would help to prevent recurrent stroke in such patients and improve quality of life.

Support: No financial support.

1139

SLEEP DISTURBANCES IN CHRONIC STROKE PATIENTS: EMOTIONAL AND COGNITIVE IMPACT

Howell, S. N. Robinson, S. E. Griesbach, G. S. Centre for Neuro Skills, Irving, TX.

Introduction: The objective was to investigate the impact of sleepwake disturbances (SWD) on cognition and quality of life in the post-acute phase of stroke. **Methods:** Adult stroke (n=92) patients were assessed for SWD via overnight polysomnography. The mean age was 52 ± 1 years and mean latency from injury was 117 ± 10 days. Sleep measures included total sleep time (TST), sleep and REM latency, percent time in sleep stages, apnea/hypopnea index (AHI), wake after sleep onset (WASO), and arousal index. The primary cognitive/outcome measures were: Montreal Cognitive Assessment (MoCA), California Verbal Learning Test (CVLT-II), Neuro-QoL and Mayo Portland Adaptability Inventory (MPAI).

Results: Women had lower AHI (F(1.88)=9.360, p<.01), fewer arousals (F(1,90)=4.53, p<.05), and spent significantly more time in SWS (F(1,90)=11.525, p<.001) than men; however, SWS was reduced in both sexes. SWS made up < 3% of TST in 60% of patients and was not correlated with higher AHI. SWDs negatively impacted subjective quality of life (NeuroQOL). Longer latencies to sleep were associated with increased depression (p<.05) and decreased positive affect (p<.01). Increased sleep efficiency led to improved positive affect (p<.05) and decreases in emotional/behavioral dyscontrol (p<.05). Increased time in REM sleep decreased emotional/behavioral dyscontrol (p<.05), while increasing satisfaction with social roles and activities(p<.01). SWDs also negatively impacted cognitive/outcome scores. Increased TST and sleep efficiency led to higher scores on CVLT-II list B and long delay free recall (p<.05), while higher AHI led to poorer performance on long delay and forced choice recognition trials (p<.01). Additionally, non-REM AHI negatively impacted MPAI adjustment scores (F(1,69)=4.036, p<.05).

Conclusion: Male stroke patients displayed significantly more arousals and spent less time in SWS than females. For both sexes, better sleep indicated improved quality of life. Sleep measures were correlated with cognitive/outcome measures. Non-REM AHI significantly predicted outcome at discharge from rehabilitation facility.

Support:

1140

SLEEP DISTURBANCES, LIFESTYLE, AND SELF-MANAGEMENT IN ADULTS WITH SUBARACHNOID HEMORRHAGE

Byun, E. McCurry, S. Kwon, S. Kim, B. Thompson, H. University of Washington, Seattle, WA.

Introduction: Subarachnoid hemorrhage (SAH) survivors often suffer sleep disturbances. Self-management strategies focusing on lifestyle changes and health-promoting behaviors may improve sleep in SAH survivors. Few studies have examined sleep in SAH survivors, and little is known about sleep management practices used to improve their sleep. The purposes of this study were to: 1) describe the prevalence of sleep disturbances using subjective and objective sleep measures, and 2) explore interest in and engagement with self-management practices to promote sleep health in SAH survivors.

Methods: We conducted a cross-sectional study with a convenience sample of 30 SAH survivors recruited from a university hospital. We assessed sleep quality using the Pittsburgh Sleep Quality Index (PSQI), daytime sleepiness using the Epworth Sleepiness Scale (ESS), and objective sleep using wrist actigraphy. We conducted content analysis of semi-structured interviews, with two authors each coding sleep disturbances and self-management practices addressing sleep.

Results: Seventy-three percent of SAH survivors reported poor sleep quality (PSQI > 5) and 27% had daytime sleepiness (ESS > 10). Actigraphy analysis indicated that 41% of SAH survivors slept

less than 7 hours or more than 9 hours. Interview content analyses suggested 3 themes and 15 sub-categories: 1) Sleep disturbances (difficulties in falling asleep, wake after sleep onset, daytime sleepiness, too much or insufficient sleep, and poor sleep quality), 2) Sleep management practices (exercise, regular sleep schedule, relaxation, keeping busy and staying active, changing beverage intake, taking supplements, taking medication, recharging energy, and barriers to sleep management), and 3) Healthcare providers (discussing sleep problems with health care providers).

Conclusion: Sleep disturbances are highly prevalent and an urgent need exists to focus on improving sleep in SAH survivors. Developing tailored interventions that incorporate self-management and lifestyle change would be a critical next step to improve sleep and promote health in this at-risk population.

Support: This research was supported by grants from the National Institutes of Health/National Institute of Nursing Research (K23 NR017404), University of Washington Institute of Translational Health Science Translational Research Scholars Program (UL1 TR000423), and University of Washington School of Nursing Research and Intramural Funding Program.

1141

DAYTIME NAPPING TRAJECTORY OVER TIME AND ITS ASSOCIATION WITH COGNITIVE AGING: A 13-YEAR COMMUNITY-BASED LONGITUDINAL STUDY OF OLDER ADULTS

Li, P.^{1,2} Gao, L.^{1,3} Gaba, A.¹ Buchman, A. S.⁴ Bennett, D. A.⁴ Hu, K.^{1,2} Leng, Y.⁵

¹Brigham and Women's Hospital, Division of Sleep and Circadian Disorders, Boston, MA, ²Harvard Medical School, Division of Sleep Medicine, Boston, MA, ³Massachusetts General Hospital Department of Anesthesia, Critical Care and Pain Medicine, Harvard Medical School, Boston, MA, ⁴Rush University Medical Center, Rush Alzheimer's Disease Center, Chicago, IL, ⁵University of California, Department of Psychiatry, San Francisco, CA.

Introduction: Daytime napping is common in elderly adults and has been associated with cognitive impairment. Prior studies have assessed napping at one time point, making it difficult to examine the longitudinal progression of napping and its association with cognitive aging. We examined objectively measured daytime napping longitudinally across different stages of Alzheimer's disease (AD): from no cognitive impairment (NCI), to mild cognitive impairment (MCI), and to Alzheimer's dementia.

Methods: We studied 1,066 participants (female: 810; age: 81.0±7.3 [SD]) in the Rush Memory and Aging Project who have been followed for up to 13 years. Motor activities of up to 10 days were recorded annually and used to assess napping objectively. We defined daytime napping episodes as segments between 10AM and 7PM with continuous zero-activity for ≥10min but <1h (to avoid off-wrist periods). Segments that were <5min apart were merged. Cognitive and clinical evaluations were administered annually to render a clinical diagnostic classification of NCI, MCI, or Alzheimer's dementia. To examine how napping duration and frequency change with the progression of AD, we performed linear mixed-effects models with 2 change points anchored at the diagnoses of MCI and AD while adjusted for age, sex, and education. Results: At baseline, participants had 1.44±0.04 (mean±standard error) naps with an accumulated duration of 35.0±1.1 min per day. Napping duration increased by 5.2±0.3 min and frequency increased by 0.21±0.01 times every year (both p<0.0001). The rate of increase was more than doubled after MCI diagnosis with an

annual increase of 11.4 ± 0.7 min in duration and 0.40 ± 0.02 times in frequency (both p<0.0001); these were doubled further after AD diagnosis with an annual change of 26.3 ± 3.1 min in duration and 0.84 ± 0.08 times in frequency (both p<0.0001).

Conclusion: Daytime napping duration and frequency increase with aging, and the increase was accelerated with AD progression. **Support:** This work was supported by NIH grants RF1AG064312, RF1AG059867, R01AG017917, and R01AG56352.

1142

COMPREHENSIVE PHENOTYPING OF AMBULATORY SLEEP PATTERNS IN MULTIPLE SCLEROSIS

Braley, T. J.¹ Kratz, A. L.² Whibley, D.² Goldstein, C.³ ¹University of Michigan Department of Neurology, Division of Neuroimmunology, Ann Arbor, MI, ²University of Michigan Department of Physical Medicine and Rehabilitation, Ann Arbor, MI, ³University of Michigan Department of Neurology, Division of Sleep Medicine, Ann Arbor, MI.

Introduction: The majority of sleep research in persons with multiple sclerosis (PwMS) has been siloed, restricted to evaluation of one or a few sleep measures in isolation. To fully characterize the impact of sleep disturbances in MS, multifaceted phenotyping of sleep is required. The objective of this study was to more comprehensively quantify sleep in PwMS, using a recently developed multi-domain framework of duration, continuity, regularity, sleepiness/alertness, and quality.

Methods: Data were derived from a parent study that examined associations between actigraphy and polysomnography-based measures of sleep and cognitive function in MS. Actigraphy was recorded in n=55 PwMS for 7-12 days (Actiwatch2[®], Philips Respironics). Sleep metrics included: duration=mean total sleep time (TST, minutes); continuity=mean wake time after sleep onset (minutes), and regularity=stddev wake-up time (hours). 'Extreme' values for continuity/regularity were defined as the most extreme third of the distributions. 'Extreme' TST values were defined as the lowest or highest sixth of the distributions. Sleepiness (Epworth Sleepiness Scale score) and sleep quality [Pittsburgh Sleep Quality Index (PSQI) sleep quality item] were dichotomized by accepted cutoffs (>10 and >1, respectively). Results: Sleep was recorded for a mean of 8.2 days (stddev=0.95). Median (1st, 3rd quartile) values were as follows: duration 459.79 (430.75, 490.60), continuity 37.00 (23.44, 52.57), regularity 1.02 (0.75, 1.32), sleepiness/alertness 8 (4, 12), and sleep quality 1.00 (1.00, 2.00). Extreme values based on data distributions were: short sleep <=426.25 minutes (18%), long sleep >515.5 minutes (16%), poor sleep continuity \geq 45 minutes (33%), and poor sleep regularity \geq 1.17 hours (33%). Sleepiness and poor sleep quality were present in 36% and 40% respectively. For comparison, in a historical cohort of non-MS patients, the extreme third of sleep regularity was a stddev of 0.75 hours, 13% had ESS of >10, and 16% had poor sleep quality. Conclusion: In this study of ambulatory sleep patterns in PwMS, we found greater irregularity of sleep-wake timing, and higher prevalence of sleepiness and poor sleep quality than published normative data. Efforts should be made to include these measures in the assessment of sleep-related contributions to MS outcomes. Support: The authors received no external support for this work.

1143

WAKE-UP STROKE IN HISPANIC VETERANS: CLINICAL CHARACTERISTICS

Colon-Feliciano, M.¹ Prats, N.² Sierra-Gonzalez, A.² Jovet, G.² Jimenez, L.¹

¹VA Caribbean Healthcare System, San Juan, PR, ²VA Caribbean, San Juan, PR.

Introduction: Wake-up stroke (WUS) is a stroke that occurs during sleep and accounts for 14-29.6% of all acute ischemic stroke (AIS) cases. The use of intravenous alteplase, the therapeutic standard, requires identifying time of stroke onset. Recent studies suggest that obstructive sleep apnea (OSA) is an independent risk factor for WUS. This study aims to describe a population of Puerto Rican Veterans with WUS and to evaluate clinical differences between patients WUS and non-WUS (NWUS).

Methods: The study was a cross-sectional analysis of all patient records with AIS from April 2018 to July 2019. One hundred forty records were reviewed. Patients who woke up with new AIS symptoms were labeled as WUS. Study variables included demographics, comorbidities, medications, sleep disorders, prior stroke, administration of alteplase and the National Institute of Health Stroke Scale (NIHSS).

Results: Among 140 participants predominantly male (98.6%), 27.1% had WUS. The mean age was 75 (range 21-89). NIHSS mean was 5.9. Comorbidities for WUS vs NWUS were as follows: overweight or obese (60% vs 62.6% p=0.667), hypertension (100% vs 95.1% p=0.323), hyperlipidemia (95% vs 93% p=0.999), diabetes mellitus (55.3% vs 67% p=0.212), atrial fibrillation (26.3% vs 24.5% p=0.826), anxiety (36.8% vs 33.3% p=0.697), depression (55.3% vs 51.0% p=0.652), non-smokers (89.% vs 89%), smokers (10.5% vs 11.1%), and prior stroke (34.2% vs 35.3% p=0.905). Insomnia was observed in 42% vs 40% (p= 0.838) and 24% vs 23% (p=0.955) had OSA.

Conclusion: There were no significant differences in clinical characteristics between patients with WUS and NWUS in this sample of Puerto Rican Veterans. OSA was less prevalent than previously reported. Lack of recognition of OSA as a risk factor for stroke and under-diagnosis of OSA might explain the study results. **Support:**

1144

ACTIGRAPHY-DEFINED SLEEP AND NEUROCOGNITIVE DECLINE IN MIDDLE-AGE HISPANIC/LATINO ADULTS

Agudelo, C.¹ Tarraf, W.² Wu, B.³ Wallace, D. M.^{1,1} Patel, S. R.⁴ Redline, S.⁵ Daviglus, M. L.⁶ Zee, P. C.⁷ Simonelli, G.⁸ Levin, B. E.¹ Mossavar-Rahmani, Y.⁹ Sotres-Alvarez, D.¹⁰ Zeng, D.¹⁰ González, H. M.³ Ramos, A. R.¹

¹Department of Neurology, University of Miami Miller School of Medicine, Miami, FL, ²Department of Healthcare Sciences and Institute of Gerontology, Wayne State University, Detroit, MI, ³Department of Neurosciences and Shiley-Marcos Alzheimer's Disease Research Center, University of California San Diego School of Medicine, San Diego, CA, ⁴Department of Medicine and Center for Sleep and Cardiovascular Outcomes Research, University of Pittsburgh School of Medicine, Pittsburgh, PA, ⁵Departments of Medicine and Neurology, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, 6Institute for Minority Health Research, University of Illinois at Chicago College of Medicine, Chicago, IL, ⁷Department of Neurology, Northwestern University Feinberg School of Medicine, Chicago, IL, 8Center For Military Psychiatry and Neuroscience, Walter Reed Army Institute of Research, Silver Springs, MD, 9Department of Epidemiology and Population Health, Albert Einstein College of Medicine, Bronx, NY, ¹⁰Department of Biostatistics, University of North Carolina Gillings School of Global Public Health, Chapel Hill, NC.

Introduction: Few studies have evaluated objective sleep measures and longitudinal neurocognitive decline, particularly in middleage or Hispanic/Latino adults. We evaluated prospective associations between actigraphy-defined sleep and 7-year neurocognitive change among Hispanic/Latino adults. We hypothesized that sleep duration would be associated with neurocognitive decline.

Methods: We analyzed data from 1,036 adults 45-64 years of age from the Hispanic Community Health Study/Study of Latinos (HCHS/SOL), a multi-center prospective cohort study of diverse community-dwelling Hispanic/Latino adults. At Visit 1 (2008-2011), participants underwent neurocognitive assessments, 7-days of actigraphy, home sleep testing, and sleep questionnaires (including the Insomnia Severity Index). Seven years later, participants repeated neurocognitive assessments. The neurocognitive battery included the Six-Item Screener, Brief Spanish-English Verbal Learning Test, phonemic word fluency test, and Digit Symbol Subtest. Survey linear regression was used to evaluate prospective associations between actigraphy-defined or self-reported sleep variables and neurocognitive change. Final models adjusted for objectively-defined variables (age, body-mass index, Field Center, and time between neurocognitive assessments), and selfreported variables (sex, education, Hispanic/Latino background, alcohol consumption, physical activity, heart failure, cerebrovascular events, depression and anxiety symptoms, and antidepressant use).

Results: At Visit 1, the sample was 55% female and mean age was 54.9±2.2 years. The mean sleep duration was 402.6±27.6 minutes, mean sleep-onset latency was 11.3±9.7 minutes, mean number of days with naps of \geq 15 minutes duration was 1.1±0.7, and mean sleep-time per nap was 51±14.1 minutes. Increased sleep-onset latency was associated with 7-year declines in global neurocognitive function (β =-0.0026, p<0.01), verbal learning (β =-0.0028, p<0.001) and verbal memory (β =-0.036, p<0.05). Increased sleep-time per nap predicted better verbal memory (β =0.0038, p<0.05). In contrast, sleep duration, sleep fragmentation, and self-reported sleep measures were not associated with neurocognitive change.

Conclusion: Among middle-age adults, sleep-onset latency and nap duration were associated with neurocognitive change. These findings may serve as targets for intervention of neurocognitive decline.

Support: This work is supported by the National Institute on Aging: R01AG048642, RF1AG054548, R01AG061022, R21AG056952, and R21HL140437 (AR).

1145

LONGITUDINAL ASSOCIATION BETWEEN CIRCADIAN ACTIVITY RHYTHMS AND RISK OF INCIDENT PARKINSON'S DISEASE IN OLDER MEN

Leng, Y.¹ Blackwell, T.² Cawthon, P. M.² Ancoli-Israel, S.³ Stone, K.² Yaffe, K.⁴

¹Department of Psychiatry, University of California, San Francisco, San Francisco, CA, ²Department of Research Institute, California Pacific Medical Center, San Francisco, CA, ³Department of Psychiatry, University of California, San Diego, San Diego, CA, ⁴Departments of Psychiatry, Neurology, and Epidemiology, University of California, San Francisco, San Francisco, CA.

Introduction: Disruption in circadian activity rhythms are very common in older adults, particularly among those with neurodegenerative diseases. However, the longitudinal association between circadian disruption and subsequent risk of developing

neurodegenerative diseases, including Parkinson's disease (PD), is unclear.

Methods: We examined rest-activity rhythms in 2930 communitydwelling older men (mean age 76.3 \pm 5.5 years) without PD and followed them for incident PD over the next 11 years. 24-h rest-activity rhythm parameters (amplitude, mesor, robustness, acrophase) were generated by wrist actigraphy-extended cosinor analysis. Incident PD cases were identified based on physician-diagnosed PD between 2005 and 2016. Logistic regression was used to determine the association between quartiles of rest-activity parameters and risk of incident PD.

Results: 78 (2.7%) men developed PD during 11 years of follow-up. The risk of PD increased with decreasing circadian amplitude (strength of the rhythm), mesor (mean level of activity) or robustness (how closely activity follows a cosine 24h pattern); p for trend across quartiles <0.05. After accounting for demographics, clinic site, education, depressive symptoms, body mass index, physical activity, benzodiazepine use, alcohol, caffeine, smoking, comorbidities and baseline cognition, those in the lowest quartile of amplitude, mesor or robustness had approximately three times the risk of developing PD compared to those in the highest quartile of amplitude [ORs (95% CI)= 3.11 (1.54-6.29)], mesor [3.04 (1.54-6.01)] and robustness [2.65 (1.24-5.66)]. The association remained after further adjustment for nighttime sleep disturbances and sleep duration. These associations were somewhat attenuated, but the pattern remained similar after excluding PD cases developed within 2 years after baseline. Acrophase was not significantly associated with risk of PD.

Conclusion: Older men with reduced circadian rhythmicity had an increased risk of incident PD over 11 years. Circadian disruption in the elderly may represent an important prodrome or risk factor for PD. Randomized trials should evaluate whether strategies to improve circadian function impact risk of PD.

Support: This work was supported by the NIA, NIAMS, NCATS, NIH Roadmap for Medical Research and the NHLBI under the grant numbers: U01AG027810, U01AG042124, U01AG042139, U01AG042140, U01AG042143, U01AG042145, U01AG042168, U01AR066160, UL1TR000128, R01HL071194, R01HL070848, R01HL070847, R01HL070842, R01HL070841, R01HL070837, R01HL070838, and R01HL070839.

1146

INTERACTION OF MILD COGNITIVE IMPAIRMENT AND LATE-LIFE DEPRESSION IN ACTIGRAPHY AND SELF REPORT OF SLEEP PROBLEMS

Aronis, J.¹ Daigle, K.¹ Almaghasilah, A.² Gilbert, C.¹ Fremouw, T.¹ Singer, C.³ Abedi, A.² Hayes, M.¹

¹University of Maine, Psychological Sciences, Orono, ME, ²University of Maine, Electrical & Computer Engineering, Orono, ME, ³Northern Light Acadia Hospital, Geriatric Psychiatry Program, Bangor, ME.

Introduction: Late-life depression has been proposed as a precursor to amnestic Mild Cognitive Impairment (aMCI), the prodrome of Alzheimer's disease. Both conditions are associated with sleep and cognitive problems. We hypothesized that MCI and current depressive symptoms would co-occur more frequently, but express distinct sleep phenotypes.

Methods: Independently living older adults (N=80), age 62-90 (M=71.78, SD=5.98), were recruited from a geriatric psychiatry clinic and the community for a home sleep study. A clinical decision board and neurocognitive battery were used to determine MCI status.

Participants completed the CES-D and depression history interview where endorsement of current depression was considered positive. Sleep was examined with wrist actigraphy for 7 days. Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), Stanford Sleepiness Scale (SSS) provided subjective sleep quality.

Results: Based on these criteria, 41.2% of the sample were determined to be MCI (n=33); the remainder were deemed normative for age (NC; n=47). Chi-square analyses showed a higher frequency of MCI were positive for current depression than expected (14.2%; p=0.017). Repeated-measures MANOVA, using current depression symptoms and MCI as factors, revealed MCI was associated with longer sleep latency (p=0.035) and wake bout time (p=0.039); whereas, current depression was associated with longer sleep latency (p=0.035), and lower sleep efficiency (p's<0.05), self-report of poorer daytime dysfunction (p=0.005), and greater daytime sleepiness (p=0.001). MCI x current depression interactions were found for sleep latency (p=0.029); and PSQI sleep disturbances (p=0.005) and sleep medication (p=0.025).

Conclusion: Despite distinct sleep disordered phenotypes, the interaction of MCI and current depression is associated with delayed sleep onset, use sleep medication and report of sleep disturbances. **Support:** This project was sponsored by: NASA, Maine Space Consortium; AG 056176, AG 053164 Vice President for Research, U. Maine; Maine Technology Institute; DoD Phase I SBIR and R44AG059536-01 SBIR Phase II Award.

1147

SLEEP CORRELATES WITH IMPROVED FUNCTIONAL OUTCOME IN THE INTENSIVE CARE UNIT

Markun, L. C.¹ Sampat, A.¹ Dutta, R.¹ Palchik, G. A.² Chow, M.¹ Park, K. J.¹ Yee, A. H.¹

¹University of California Davis School of Medicine, Sacramento, CA, ²California Pacific Medical Center, San Francisco, CA.

Introduction: Disruption of sleep may have significant implications in acute brain injury, functional recovery, and critical illness. Few data exist characterizing sleep architecture in patients admitted to an intensive care unit (ICU). We aim to describe sleep and clinical characteristics in patients with acute brain injury and critical illness.

Methods: Retrospective analysis was performed in ICU patients who underwent continuous electroencephalographic (EEG) monitoring from 2018-2019. Sleep was scored based on AASM-defined EEG criteria. Clinical variables, EEG characteristics, and modified Ranking Scale (mRS) were collected. Good outcome was defined as mRS<3. Differences were assessed using chi-square analysis and t-test.

Results: 205 patients were reviewed with a mean age of 57 years (range 18-91) and a majority (57%) were male. Patients carried a primary neurologic/neurosurgical (61%) or medical/surgical (39%) diagnosis. Status epilepticus, subdural hemorrhage, traumatic brain injury, encephalopathy and cardiac arrest accounted for the majority of diagnoses encountered. Only 58 patients (28%) achieved N1 sleep; of these 76.4% achieved N2, 2.8% N3, and none achieved REM. Of those achieving any sleep, 43% had good outcomes versus only 23% in those who did not (t=-7.45, p<0.001). Neurological patients were more likely to attain sleep compared to those with other primary diagnoses (χ^2 (1)=7.08, p=0.008). Centrally acting anesthetics did not account for sleep differences between neurologic and non-neurologic patients (χ^2 (1)=2.01,

p=0.16). However, those with primary brain injury reached sleep more often in the absence of anesthetic use (χ^2 (1)=4.82, p=0.03). The overall mortality was 32% in this cohort.

Conclusion: Most critically ill patients do not achieve electrophysiologic sleep. Of those who do, N1/N2 stages are seen most often. Neurological patients were more likely to sleep, and achieving any sleep was associated with improved functional outcome. Further studies are needed to determine whether sleep augmentation in the critically ill impacts functional outcome. **Support:** N/A.

1148

CONCEIVING A CONNECTED, PREVENTATIVE TREATMENT STANCE ACROSS THE BRAIN-GUT-MICROBIOTA AXIS IN PRODROMAL PARKINSON'S DISEASE: THE POWER OF PREVENTATIVE SLEEP HEALTH

Chandler, J. F.¹ Bullock, M. M.¹ Chandler, N. G.² Nelson, S. M.¹ Hoyle, S. P.¹ Guice, J. O.¹

¹Birmingham-Southern College, Birmingham, AL, ²Methodist University, Fayettville, NC.

Introduction: Early non-motor symptoms of Parkinson's Disease (PD) include sleep and digestive disturbances that share a common pathology via alpha-synuclein (AS) deposition in the central nervous system (CNS) and formation in the enteric (ENS). The concept of prodromal PD as expressed by the braingut-microbiota axis continues to gain credibility across multiple literatures; yet, no unified treatment plan has been suggested. Disconnected, symptomatic treatment of prodromal PD may unintentionally hasten its development via compromise of REM sleep quality and reciprocal gut health. A more connected, comprehensive approach is needed. We conducted a systematic literature review to hypothesize next steps in treatment research for prodromal PD.

Methods: A systematic literature review using the Boolean combinations of "ALPHA-SYNUCLEIN" & - "SLEEP", "GUT MICROBIOME", and "EXERCISE" in all fields, restricted to the last 5 years, focusing on specification of prodromal PD, was conducted. Results were combined with an examination of current treatment practices in PD.

Results: 38 articles met inclusion criteria. Results were categorized through a presupposed primacy of sleep.

Conclusion: Due to the emerging nature of the prodromal PD idea, current treatment practice is myopic and may contribute to the progression of PD. Specifically, 1) sedative-hypnotic sleep interventions suppress REM, where clearing of CNS alphasynuclein occurs, and 2) proton-pump inhibitors (PPI) cause significant gut dysbiosis, which may contribute to initial AS misfolding in the gut. Early identification of prodromal PD symptoms via cross-referenced clinical interview may allow for early behavioral interventions that underlie healthy brain-gut-microbiota axis functioning. Results outline specific measures that may slow PD-related synucleinopothies. The highest impact practices in this regard are REM-focused sleep hygiene and cardiovascular conditioning in a reciprocal relationship, highlighting the necessity of an early-intervention, preventative health model for conquering PD.

Support: This work was made possible in part by a donation from Drs. Shane Pitts and Michelle Hilgeman in support of Birmingham-Southern College's Southern Sleep Laboratory.

1149

A PILOT QUALITY IMPROVEMENT (QI) STUDY TO ASSESS WHETHER BILEVEL POSITIVE AIRWAY PRESSURE (BIPAP) SUPPORT IN ACUTE ISCHEMIC STROKE PATIENTS WITH SLEEP DISORDERED BREATHING, CAN IMPROVE NEUROLOGICAL RECOVERY DURING ACUTE STROKE CARE

Nadhim, A. N.¹ wong, J.¹ Gupta, D.¹ Suhan, L.¹ Siegel, M.¹ Bhat, S.¹ strauss, S.¹ Fourcard, F.³ Pandya, V.³

¹JFK Neuroscience institute, Edison, NJ, ²JFK Neuroscience institute, Edison, NJ, ³JFK Neuroscience institute, perth amboy, NJ.

Introduction: Obstructive sleep apnea (OSA) has been associated with adverse outcomes in patients with stroke. While data is limited, it suggests that treatment of OSA may improve neurological recovery. With this quality improvement (QI) project, we aim to develop an interprofessional-team workflow process for screening and correction of OSA in acute ischemic stroke, with the goal to improve outcomes of neurological recovery.

Methods: This is an ongoing study to screen all eligible patients admitted to JFK Medical Center stroke unit, with MRI-proven Supratentorial acute ischemic stroke. The patients are screened using an overnight Pulse Oximetry test. A 3% oxygen desaturation index (ODI) of $\geq 10/hr$ or 4% ODI of $\geq 5/hr$ is considered at high risk for OSA. Such Patients will receive nocturnal Auto-adjusting BIPAP therapy during their acute care stay, for up to 5 days, for at least 4 hours per night. Eligible Patients who refused BiPAP therapy or were non-compliant will be considered as a controls. Baseline NIH stroke scale (NIHSS), and bilateral MCA mean flow velocity (MFV) in the morning, by transcranial doppler (TCD) will be assessed at baseline for cases and controls, and after BiPAP therapy, for the case group. The two groups of patients will also be compared in terms of Modified Rankin Scale at time of discharge and at phone follow-up after 6 weeks.

Results: Between Oct 17^{th} , 2019 to current, 15 patients were admitted to the stroke unit with MRI confirmed stroke. Ages ranged from 34 - 88 years (average age 66.5 years). 8 patients (60%) were female. Of those, 6 patients consented to being screened for OSA. Of these, 1 had 4%ODI >5/hr, and therefore received treatment with BIPAP. However, compliance was < 4 hrs on 2 consecutive nights.

Conclusion: This is ongoing QI project and results will be available after few more months of continued recruitment.

Support: Auto-adjusting BIPAP machines were provided by RESMED.

1150

OBSTRUCTIVE SLEEP APNEA-DEPENDENT RACIAL/ ETHNIC AND SEX-SPECIFIC MECHANISMS UNDERLYING ALZHEIMER'S DISEASE RISK: A RETROSPECTIVE COHORT ANALYSIS OF IN-LAB PSG SLEEP STUDY DATA

Bubu, O. M.¹ Turner, A. D.¹ Parekh, A.² Mullins, A.² Kam, K.² Umasabor-Bubu, O. Q.³ Mbah, A. K.⁴ Williams, N. J.¹ Varga, A. W.² Rapoport, D. M.² Ayappa, I.² Jean-Louis, G.¹ Osorio, R. S.¹

¹NYU School of Medicine, New York, NY, ²Icahn School of Medicine at Mount Sinai, New York, NY, ³SUNY Downstate Medical Center, Brooklyn, NY, ⁴University of South Florida, Tampa, FL.

Introduction: We examined race and sex-specific biologic mechanisms of the relationship between obstructive sleep apnea (OSA) and incident AD.

Methods: Retrospective cohort analysis utilizing in-lab PSG sleep study data conducted among older adults between 2001 and 2005. OSA was defined using AHI4%. Participants had no history of cognitive decline or AD at baseline and included 663 (284 Non-Hispanic White (NHW), 207 Black/African-American (AA) and 172 Hispanic) OSA-patients matched on age, sex, race, BMI, 1:1 ratio to 663 (unexposed cohort I from sleep clinic) and 1:4 ratio to 2652 (unexposed cohort II from non-sleep clinics) non-OSA individuals. Incident AD was assessed annually from 2001-2013 with ICD-9-CM code 331.0. Adjusted cox proportional hazard regression models examined race and sex-specific biologic mechanisms including hypoxia, fragmentation and duration measures of OSA and AD risk.

Results: Of the 3,978 participants, 2,148 (54%) were women. Mean age at baseline was 72.6 (7.3) years. Over a mean follow-up time of 8.6 (1.4) years, 358 (9%) individuals (212 female) developed AD (119 NHW, 134 AAs, and 105 Hispanics). Relative to non-OSA individuals, OSA-patients had a higher risk of incident AD, with AAs and females showing stronger risk estimates (aHR: 2.24, 1.83, and 1.73, P <.001 for all, for AAs, Hispanics and NHW respectively; and aHR: 2.38, and 1.37, P <.001 for all, for female and male respectively). Measures of hypoxia, sleep fragmentation and sleep duration were associated with increase AD risk (P <.01 for all). Relative to NHW, AAs and Hispanics demonstrated up to 20% stronger effects/estimates on hypoxia and sleep duration measures. Relative to males, females demonstrated up to 25% stronger effects/estimates on hypoxia measures (P <.01 for all).

Conclusion: Among OSA-patients, mechanisms related to hypoxia, sleep fragmentation and duration measures increase AD risk and may underlie race/ethnicity and sex disparities in AD.

Support: NIH/NIA/NHLBI (L30-AG064670, CIRAD P30AG059303 Pilot, T32HL129953, R01HL118624, R21AG049348, R21AG055002, R01AG056031, R01AG022374, R21AG059179, R01AG056682, R01AG056531, K07AG05268503, K23HL125939)

1151

IS TIMING OF LIGHT EXPOSURE DIFFERENT IN WOMEN WITH CHRONIC MIGRAINE?

Dawson, S. C.¹ Kim, M.¹ Reid, K.¹ Burgess, H. J.² Wyatt, J. K.³ Hedeker, D.⁴ Park, M.⁵ Rains, J. C.⁶ Espie, C. A.⁷ Taylor, H. L.⁸ Ong, J. C.¹

¹Department of Neurology, Northwestern University, Chicago, IL, ²Department of Psychiatry, University of Michigan, Ann Arbor, MI, ³Rush University Medical Center, Chicago, IL, ⁴University of Chicago, Chicago, IL, ⁵Chicago Sleep Health, Advocate/Illinois Masonic Hospital, Chicago, IL, ⁶Center for Sleep Evaluation, Elliot Hospital, Manchester, NH, ⁷Nuffield Department of Clinical Neurosciences, University of Oxford, Oxford, UNITED KINGDOM, ⁸The Maine Sleep Center at Chest Medicine Associates, South Portland, ME.

Introduction: Light avoidance is a common coping behavior of individuals with migraine headaches. It is not known whether timing of light exposure is different in individuals with chronic migraine (CM) compared to those without migraine and how this may relate to headache frequency and severity. We tested this by examining timing of the brightest and darkest light and headaches in women with chronic migraines and healthy controls.

Methods: Sixteen women with CM (mean age = 33.07) and 18 female healthy controls (HC; mean age = 32.22) completed daily

ratings of headache severity (0-10, severity > 2 classified as headache) concurrent with light exposure measured by wrist actigraphy for approximately one month (M=28.00 days, range=21-36). Start time of each day's 10-hour periods of maximum light (M10) and 5-hour periods of lowest light (L5) were calculated and averaged for each participant. T-tests and Cohen's d effect sizes were used to compare groups. Pearson correlation coefficients were calculated to examine associations between M10/L5 timing and headache frequency and severity.

Results: M10 was earlier in the CM group compared to the HC group (07:42 \pm 00:47 vs. 08:50 \pm 00:58, t(32)=3.69, p=0.0008, d=1.08). The CM group exhibited non-significant trend towards earlier L5 compared to the HC group (12:26 \pm 00:48 vs. 01:07 \pm 01:03, t(32)=1.89, p=0.0723, d=0.62). Among individuals with CM, later M10 timing was associated with more severe average daily head-ache (r=0.60, p=0.0136) and more frequent headaches (r=0.55, p=0.0257). Later L5 timing was significantly associated with more severe average daily headache (r=0.66, p=0.0136) and sociated with more severe average daily headache (r=0.66, p=0.0052) and showed a non-significant trend toward association with more frequent headaches (r=0.47, p=0.0686).

Conclusion: Timing of the greatest light exposure period was earlier in CM compared to HC. Within the CM group, those who had earlier light and dark periods reported lower headache severity and fewer days with headaches. These findings suggest the possibility of a role for the circadian system in chronic migraine.

Support: This study was supported by grant R21NS081088 from the National Institutes of Health.

1152

GENETIC RISK OF ALZHEIMER'S DISEASE IS LINKED TO SHORT SLEEP DURATION

Leng, Y.¹ Yaffe, K.² Ackley, S.³ Glymour, M.³ Brenowitz, W.¹ ¹Department of Psychiatry, University of California, San Francisco, San Francisco, CA, ²Department of Psychiatry, Neurology and Epidemiology& Biostatistics, University of California, San Francisco, San Francisco, CA, ³Department of Epidemiology & Biostatistics, University of California, San Francisco, San Francisco, CA, ⁴Department of Psychiatry, University of California, San Francisco, San Francisco, CA.

Introduction: Sleep disturbances including short sleep duration are common in older adults, especially in those with Alzheimer's disease (AD). However, it is unclear to what extent sleep duration is a manifestation of AD disease process. We examined whether genetic variants related to AD influence sleep duration in middle-aged and older adults and estimated the causal effects of AD on sleep duration using a mendelian randomization (MR) analysis.

Methods: We examined 406,687 UK Biobank participants with Caucasian genetic ancestry who self-reported sleep duration at baseline (2006-2010). Sleep duration was assessed by asking: "About how many hours sleep do you get in every 24 hours? (please include naps)." A genetic risk score for AD (AD-GRS) was calculated as a weighted sum of 23 previously identified AD-related single nucleotide polymorphisms in individuals of European ancestry. We evaluated whether AD-GRS predicted sleep duration using linear regression, adjusting for age, sex and principle components for genetic ancestry. We also stratified the analysis by age at baseline (\leq 55y or >55y) and conducted a MR analysis to estimate the effect of AD (ICD-9/10 codes for AD/dementia diagnosis) on sleep duration.

Results: The participants (aged 56.91±8.00y) had an average sleep duration of 7.2 (Standard deviation [SD]=1.1) hours and AD-GRS

of 0.11 (SD=0.40) (range: -1.15~1.85). Higher AD-GRS score predicted shorter sleep duration (b= -0.013, 95%CI:-0.022,-0.005), mainly among those aged over 55y (b= -0.023, 95%CI:-0.034,-0.012) and not in those 55y or younger (b= 0.006, 95%CI:-0.012,0.013); p for interaction by age=0.02. MR analysis using AD-GRS as an instrumental variable suggested that AD was associated with 1.76 hrs (b=-1.76, -2.62~-0.90) shorter sleep duration in those aged >55y.

Conclusion: Using a novel analytical approach, we found that higher genetic risk for AD predicted shorter sleep duration among older adults. This suggests shared genetic pathways; the biologic processes that lead to AD may also affect sleep duration.

Support: Dr. Leng received support from the National Institute on Aging (NIA) 1K99AG056598, and from GBHI, Alzheimer's Association, and Alzheimer's Society (GBHI ALZ UK-19-591141).

1153

ASSOCIATION OF NOCTURNAL SLEEP DISTURBANCE AND PROSPECTIVE COGNITIVE DECLINE IN COGNITIVE NORMAL ELDERLY: FINDINGS FROM THE NATIONAL ALZHEIMER'S COORDINATING CENTER UNIFORM DATASET

Bubu, O. M.¹ Mbah, A. K.² Williams, N. J.¹ Turner, A. D.¹ Parekh, A.³ Mullins, A. E.³ Kam, K.³ Umasabor-Bubu, O. Q.⁴ Varga, A. W.³ Rapoport, D. M.³ Ayappa, I.³ Jean-Louis, G.¹ Osorio, R. S.¹

¹NYU School of Medicine, New York, NY, ²University of South Florida, Tampa, FL, ³Icahn School of Medicine at Mount Sinai, New York, NY, ⁴SUNY Downstate Medical Center, Brooklyn, NY.

Introduction: We determined whether nocturnal sleep disturbance (NSD) is associated with prospective cognitive decline in clinically normal older adults

Methods: Prospective longitudinal study utilizing data from the National Alzheimer's Coordinating Center (NACC) Uniform Data set (UDS). NSD data, as characterized by the Neuropsychiatric Inventory Questionnaire (NPI-Q), were derived from 10,600 participants at baseline, with at least one UDS follow-up visit, from 32 National Institute of Aging Alzheimer's Disease Research Centers (ADRC). Prospective cognitive decline was characterized as incident mild cognitive impairment (MCI) diagnosis during UDS follow-up. Logistic mixed-effects model with random intercept and slope examined associations between the NSD and longitudinal cognitive decline. All models included age at baseline, sex, years of education, APOE &4 status and their interactions with time. Time was operationalized as years from baseline for each participant.

Results: Of the 10,600 cognitively normal participants at baseline, 1,017 (8.6%) had NSD. The proportion of males versus females with sleep problems was 10.1% vs. 9.3% respectively. For participants with NSD and no NSD, the mean (SD) age was 71 (7.3) and 70 (5.7) years and average follow-up time was 5.2 (2.6) and 4.9 (2.7) years, respectively. Participants with NSD were significantly more likely to develop incident MCI during UDS follow-up (OR: 1.42, p =.003). The interaction of NSD with time was significant (p < .001) suggesting an increase in the likelihood of conversion to MCI increased over time. Furthermore, there were significant differences in mean conversion rates to MCI in the NSD group when the previous time-point was compared to the next (p < 01), with a time dependent dose response in the risk of conversion to MCI observed. Conclusion: In elderly cognitive-normal individuals, nocturnal sleep disturbance is associated with a time-dependent progression risk to MCI. These findings are consistent with the role of disturbed sleep in the development of Alzheimer's Disease.

Support: NIH/NIA/NHLBI (L30-AG064670, CIRAD P30AG059303 Pilot, T32HL129953, R01HL118624, R21AG049348, R21AG055002, R01AG056031, R01AG022374, R21AG059179, R01AG056682, R01AG056531, K07AG05268503, K23HL125939)

1154

SELF-REPORTED SLEEP DURATION AND QUALITY ARE ASSOCIATED WITH POST-TRAUMATIC STRESS DISORDER FOLLOWING STROKE

Romero, E. K. Kronish, I. M. Shechter, A. Columbia University Medical Center, New York, NY.

Introduction: Up to one in eight patients may experience post-traumatic stress disorder (PTSD) within the year following a stroke or transient ischemic attack (TIA). Sleep disturbance is a chief complaint in PTSD and is common following stroke. We therefore examined whether sleep was associated with post-stroke PTSD. Methods: The Reactions to Acute Care and Hospitalization (REACH)-Stroke study is an observational cohort study examining factors related to long-term health outcomes following stroke/TIA. Typical sleep duration (self-report) and quality (1: very good to 4: very bad) over the month following hospital discharge was assessed at 1-month follow-up. At 1 month, patients also completed the PTSD checklist for DSM-5 (PCL-5 cued to the stroke/TIA event). Binary logistic regression was conducted, producing odds ratios (OR) on the association between sleep within the month following discharge and PTSD symptoms at 1 month post-stroke, controlling for age, sex, and race/ethnicity.

Results: Analyses included 459 patients (age: 61.1 ± 15.6 y, 53.2% female). Short sleep (<7 h/night) and poor sleep quality (fairly/very bad) was reported in 49.2% and 25.5% of patients, respectively. Elevated PTSD symptoms (PCL-5 score \geq 30) at 1 month were reported in 10.9% of patients. Sleep was significantly shorter and worse quality in those with PTSD vs. without (p-values<0.001). Short sleep duration vs. not short duration throughout the month following discharge was significantly associated with elevated PTSD symptoms at 1-month (OR=3.34, 95% CI: 1.51-7.38, p=0.003). Poor sleep quality (fairly or very bad rating) vs. good sleep (fairly or very good rating) was also significantly associated with elevated PTSD symptoms at 1-month (OR=2.23, 95% CI: 1.13-4.41, p=0.021).

Conclusion: Patients with short duration and poor quality sleep in the month following stroke are at an increased risk of having elevated PTSD symptoms. Understanding factors related to the development of post-stroke PTSD is important since PTSD in stroke survivors can reduce quality of life, contribute to non-adherence to prescribed medications, and increase risk of recurrent stroke and/or cardiovascular events. Future studies should be conducted to determine whether sleep is a modifiable determinant of PTSD symptoms after stroke. **Support:** R01HL141494, R01HL132347

1155

THE ASSOCIATION BETWEEN STOPBANG RISK AND SLEEP QUALITY IN AN MTBI SAMPLE

*Garcia, A.*¹ *Reljic, T.*² *Kenney, K.*³ *Amma, A.*⁴ *Troyanskaya, M.*⁵ *Elisabeth, W.*⁵ *William, W.*⁴ *Richardson, R.*⁷

¹Defense and Veterans Brain Injury Center, Tampa, FL, ²Morsani College of Medicine, University of South Florida, Tampa, FL, ³National Intrepid Center of Excellence, Walter Reed National Military Medical Center, Bethesda, MD, ⁴Department of Physical Medicine and Rehabilitation, Virginia Commonwealth University, Richmond, VA, ⁵Department of Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston, X. Sleep and Neurologic Disorders

TX, ⁶Department of Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston, TX, ⁷Mental Health and Behavioral Sciences and Defense and Veterans Brain Injury Center, James A. Haley VA Hospital, Tampa, FL.

Introduction: Although Obstructive Sleep Apnea (OSA) is prevalent in the military population, traditional scoring/clinical cutoffs of available screening tools may not be appropriate for this younger, slimmer population. We additionally have limited information regarding utility of OSA screening in those with history of mild traumatic brain injury (mTBI). The current study compared multiple STOPBANG scoring methods to determine how well they were associated with self-reported sleep measures in an mTBI sample.

Methods: Secondary analyses were conducted from a large database of evaluations from a multi-center, longitudinal study of mTBI. Participants were included if they had completed the STOPBANG and additional sleep measures. The subsequent sample (N=486) included participants with history of mTBI (n=408) and controls (n=78). The sample was predominantly male (n=421) with a mean age of 39 (IQR = 31/37/47).

Results: Sleep efficiency was not significantly associated with any STOPBANG scoring, in the total sample or when controlling for mTBI. In the total sample, sleep quality was most strongly associated with traditional STOPBANG scoring (STOPBANG $\geq 3, \beta=0.51$). Sleep duration was most strongly associated with Snoring/Tired/Hypertension (STP = 3, $\beta=0.79$). When controlling for mTBI, duration was most strongly associated with traditional scoring ($\beta=0.48$), while sleep quality was most strongly associated with Snoring/Tired/Hypertension ($\beta=0.78$). Follow-up analyses demonstrate a significant difference in correlation between groups for STP/Sleep Quality, with a stronger correlation for those without mTBI.

Conclusion: The STOPBANG measure was significantly associated with self-reported sleep quality/duration measures, but not sleep efficiency. Although traditional clinical cut-offs for OSA predicted sleep measures in this sample, the relationship between risk scores and outcomes became more nuanced when history of mTBI was included. Future studies are needed to understand the relationship between OSA risk and subsequent diagnosis in the mTBI population.

Support: Defense and Veterans Brain Injury Center (GDHS,W91YTZ-13-C-0015), DOD(W81XWH-12-2-0095), VA(I01 CX001135)

1156

POLYSOMNOGRAPHIC TOTAL SLEEP TIME: A NOVEL BIOMARKER FOR DEMENTIA

Nowakowski, S.^{1,2} Razjouyan, J.^{1,2} Naik, A. D.^{1,2} Agrawal, R.^{1,2} Velamuri, K.^{1,2} Singh, S.^{1,2} Sharafkhaneh, A.^{1,2} Kunik, M. E.^{1,2} ¹Baylor College of Medicine, Houston, TX, ²Michael E DeBakey Veteran Affairs Medical Center, Houston, TX, ³Baylor College of Medicine, Houston, TX.

Introduction: Neuroprotection, early diagnosis, and behavioral intervention are national priorities for dementia research. Sleep duration is emerging as an important potential remediable risk factor. In this study, we examined whether total sleep time (TST) derived from attended overnight polysomnography (PSG) studies is associated with an increased prevalence of dementia diagnosis and determined the optimal cut-point.

SLEEP, Volume 43, Abstract Supplement, 2020

Methods: We identified 69,847 PSG sleep studies using CPT code 95810 from 2000-19 in the US Department of Veteran Affairs (VA) national database of patient care. We used natural language processing to verify PSG reports and extract TST values from the patient free-text notes. We examined a TST of 240-420 minutes in 10-minute increments using a run chart (time series) approach to determine the optimal cut-point for determining greater odds of dementia.

Results: Patients had a mean age of 55.4 ± 13.8 , 91.5% were male, and 64% were Caucasian. PSG studies revealed a mean TST of 310.6 ± 79.5 minutes. The run chart time series analysis revealing < 360 minutes being the optimal cut-point for increased odds of dementia (OR: 1.64, 95% CI: 1.36-1.99, *p*<.05).

Conclusion: Lower TST predicted higher prevalence of dementia diagnosis. TST of 360 minutes may serve as the optimal cut-point to determine greater odds of dementia. This is an important study examining PSG sleep duration and the prevalence of dementia across 19 years in the largest integrated healthcare system in the US. TST may function as a potential biomarker for developing dementia.

Support: This material is based upon work supported in part by the Department of Veteran Affairs, Veterans Health Administration, Office of Research and Development, and the Center for Innovations in Quality, Effectiveness and Safety (CIN 13-413). Dr. Nowakowski is also supported by a National Institutes of Health (NIH) Grant (R01NR018342).

1157

COMPARISON OF POLYSOMNOGRAPHY TOTAL SLEEP TIME IN VETERANS WITH A DEMENTIA DIAGNOSIS, INCIPIENT DEMENTIA, AND NO DEMENTIA

Razjouyan, J.^{1,2} Nowakowski, S.^{1,2} Naik, A. D.^{1,2} Sharafkhaneh, A.^{1,2} Kunik, M. E.^{1,2}

¹Baylor College of Medicine, Houston, TX, ²Michael E DeBakey Veteran Affairs Medical Center, Houston, TX.

Introduction: Neuroprotection, early diagnosis, and behavioral intervention are national priorities for dementia research. Sleep duration is emerging as an important potential remediable risk factor. In this study, we examined the total sleep time derived from overnight polysomnography (PSG) studies in veterans with a current dementia diagnosis at the time of PSG study (dementia), future diagnosis of dementia following the PSG study (incipient dementia), and no diagnosis of dementia at any time point (no dementia) over a 19-year period.

Methods: We identified 69,847 PSG sleep studies using CPT code 95810 and all-cause dementia diagnosis using ICD 9/10 codes (e.g., F03.90) from 2000-19 in the US Department of Veteran Affairs (VA) national database. To be included patients must have \geq 1 VA visits in 12 months leading up to PSG. Dementia diagnosis must be documented on two separate visits between 12 months prior to 6 months following PSG for current dementia group and anytime after the PSG for incipient dementia. We used natural language processing to extract TST values from the patient free-text notes. Analysis of variance was used to compare PSG TST of the three groups.

Results: Patients had a mean age of 55.4 ± 13.8 at the time of PSG study, 91.5% were male, and 64% were Caucasian. TST of dementia patients (N=1,031) was m= $257\pm110m$ (d=0.33, p<.05), incipient dementia (N=1,875) was m= $253\pm116m$ (d=0.35, p<.05) versus no dementia (61,871) m= $292\pm104mins$.

Conclusion: Patients with a diagnosis of dementia at the time of PSG study and patients that went on to receive a diagnosis following their PSG study had a significantly lower total sleep time compared to patients that have never received a dementia diagnosis. This is an important study that compares sleep duration during overnight PSG studies and dementia diagnosis across 19 years in the largest integrated healthcare system in the US.

Support: This material is based upon work supported in part by the Department of Veteran Affairs, Veterans Health Administration, Office of Research and Development, and the Center for Innovations in Quality, Effectiveness and Safety (CIN 13-413). Dr. Nowakowski is also supported by a National Institutes of Health (NIH) Grant (R01NR018342).

1158

DAYTIME SLEEPINESS, DEPRESSION, AND POST-CONCUSSIVE SYMPTOMS IMPROVE FOLLOWING PRESCRIBED MORNING EXPOSURE TO BLUE LIGHT

Raikes, A. C. Dailey, N. S. Alkozei, A. Vanuk, J. R. Grandner, M. A. Killgore, W. D. University of Arizona, Tucson, AZ.

Introduction: Long-term sleep disruption, fatigue, and depression are common after mild traumatic brain injuries (mTBIs). Efficacious treatments for these disturbances in the context of mTBIs are lacking. Morning blue light therapy (BLT) effectively treats sleep disruption and improves mood. This study evaluated the treatment effects of morning BLT on post-mTBI daytime sleepiness, depression, and post-concussion symptoms.

Methods: 62 individuals (Boston: n=31; age: 23.11 ± 7.20 ; 17 females; days post-injury: 236.00 ± 121.40 ; Tucson: n=31; age: 26.35 ± 8.08 y; 20 females; days post-injury: 272.94 ± 167.69) received either BLT (n=30) or placebo amber light therapy (ALT; n=32). All participants completed the Epworth Sleepiness Scale (ESS), Pittsburgh Sleep Quality Index (PSQI), Beck Depression Inventory (BDI), and Rivermead Post-concussion Symptom Questionnaire (RPQ3 and RPQ13 subscales) pre- and post-treatment. Treatment consisted of direct exposure to either blue or amber light (30 minutes each morning), delivered via tabletop light-box, over six weeks. Baseline and post-treatment values were compared to a non-mTBI, non-treated control sample (Tucson: n=32, age: 23.94 ± 5.41 y; 19 females).

Results: Baseline scores were higher in both mTBI light groups (BLT, ALT, respectively) than controls for the ESS (Cohen's d=0.83, 0.83), PSQI (d=1.45, 1.71), BDI (d=1.46, 1.62), RPQ3 (d=1.72, 1.62) and RPQ13 (d=1.86, d=1.76). BLT resulted in lower within-group ESS (d=-0.58), BDI (d=-0.50), PSQI (d=-0.57), and RPQ13 (d=-0.45, p=0.005) scores. No improvements were seen following ALT. Minimal ESS score differences between the BLT and controls were observed after treatment (d=0.25).

Conclusion: Daily morning BLT resulted in moderate improvements in post-mTBI daytime sleepiness, sleep quality, depression, and post-concussion symptoms. These improvements may contribute to enhanced academic and job performance, post-mTBI quality of life, and general recovery. Future work is needed to clarify optimal dosage and precision medicine factors indentifying those most likely to benefit from morning BLT.

Support: This research was supported by multiple grants from the US Army Medical Research and Materiel Command (USAMRMC) to Dr. William D. S. Killgore, including W81XWH-11-1-0056 and W81XWH-14-1-0571.

1159

SLEEP TRAITS AND INCIDENT DELIRIUM DURING A DECADE OF FOLLOW-UP IN 173,000 PARTICIPANTS.

Gao, L.^{1,2} Li, P.^{2,3} Cui, L.² Johnson-Akeju, O.¹ Hu, K.^{2,3} ¹Department of Anesthesia, Critical Care and Pain Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, MA, ²Division of Sleep and Circadian Disorders, Brigham and Women's Hospital, Boston, MA, ³Division of Sleep Medicine, Harvard Medical School, Boston, MA.

Introduction: Delirium is an acute decline in attention and cognition that is with associated long-term cognitive dysfunction in elderly patients. Accumulating evidence points to strong associations between sleep health and disorders of the brain. We tested whether baseline sleep duration, chronotype, daytime dozing, insomnia or sleep apnea predict incident delirium during hospitalization.

Methods: We studied participants from the UK Biobank who have been followed for up to 10 years until 2017. We included 173,221 participants (mean age 60±5; range 50-71 at baseline) who had at least one episode of hospitalization/surgery and were free from prior episodes of delirium. Delirium diagnosis, hospitalization and surgical events were derived using ICD-10 coding. Multivariate logistic regression models were performed to examine the associations of self-reported baseline sleep duration (<6hrs/6-9h/>9h), daytime dozing (often/rarely), insomnia (often/rarely) and presence of sleep apnea (ICD-10 and self-report) with incident delirium during follow-up. Models were adjusted for demographics, education, Townsend deprivation index, and major confounders (number of hospitalizations/surgical procedures, BMI, diabetes, major cardiovascular diseases and risk factors, major neurological diseases, major respiratory diseases, cancer, alcohol, depression/ anxiety, sedatives/sleep aides, antipsychotics, steroids and opioids). Results: In total, 1,023 (5.7 per 1,000 subjects) developed delirium. A prior diagnosis of sleep apnea (n=1,294) saw almost a two-fold increased odds (OR 1.96, 95% CI: 1.30-2.30 p=0.001) while those who often had daytime dozing were also at increased risk (OR 1.35, 95% CI: 1.02-1.80, p=0.025). Both these effects were independent of each other. No independent effects on incident delirium were observed from sleep duration, insomnia, or chronotype.

Conclusion: Certain sleep disturbances, in particular sleep apnea and daytime dozing, are independently associated with an increased risk for developing delirium. Further work is warranted to examine underlying mechanisms and to test whether optimizing sleep health can reduce the risk of developing delirium.

Support: This work was supported by NIH grants T32GM007592, RF1AG064312, and RF1AG059867.

1160

IMPROVED DAYTIME SLEEPINESS FOLLOWING DAILY MORNING BLUE LIGHT THERAPY IS ASSOCIATED WITH ALTERED RESTING-STATE NETWORK CONNECTIVITY

Raikes, A. C. Dailey, N. S. Vanuk, J. R. Alkozei, A. Grandner, M. A. Killgore, W. D. University of Arizona, Tucson, AZ.

Introduction: Light exposure, particularly blue wavelength light, has consistent positive effects on daytime sleepiness following mild traumatic brain injuries (mTBIs). While self-perceived

improvements in daytime sleepiness are well-documented, the neurobiological underpinnings are not well understood. The purpose of this study was to localize changes in functional connectivity after daily morning blue light therapy (BLT) and to associate these changes with improvements in post-mTBI daytime sleepiness. Methods: 29 individuals with a history of mTBI were randomized to receive either BLT (n=13) or placebo amber light (ALT; n=16). All participants self-reported daytime sleepiness (Epworth Sleepiness Scale (ESS); lower is better) and underwent resting-state functional magnetic resonance imaging at pre- and post-treatment. Whole-brain functional connectivity (FC) was estimated as the correlations between 400 cortical regions of interest (ROIs) assigned to 7 resting-state networks. A two-sample T-test for post-treatment ROI-to-ROI FC identified target connections (FDR corrected p<0.01). Post-treatment ESS scores and FC for these connections were correlated for treatment-related brain-behavior associations (uncorrected p<0.05).

Results: Lower FC after BLT in 4 ROI-to-ROI connections linking the default mode and visual networks was associated with lower ESS scores. Higher FC after BLT in 9 ROI-to-ROI connections linking attention, cognitive control, and visual networks was also associated with lower ESS scores.

Conclusion: BLT resulted in decreased self-reported daytime sleepiness, which was associated with decoupling of the default mode and visual networks as well as increased connectivity between and within attention and cognitive control networks, suggesting potentially improved attention to relevant stimuli and cognitive processes and less internal mentation. These associations may contribute to improved alertness, attention, and cognitive performance following a mTBI. Further work is needed to identify the optimal timing and dosage of BLT to maximize these outcomes.

Support: This study was funded by an award to Dr. Killgore from the US Army Medical Research and Materiel Command (USAMRMC; award number: W81XWH-14-1-0571).

1161

TASIMELTEON SHOWS PERSISTENCE OF EFFICACY IN IMPROVING SLEEP DISTURBANCES IN PATIENTS WITH SMITH-MAGENIS SYNDROME (SMS) IN OPEN-LABEL EXTENSION STUDY

Brooks, J. Gibson, M. Kite, K. Czeisler, E. Fisher, M. Xiao, C. Polymeropoulos, C. Polymeropoulos, M. Vanda Pharmaceuticals Inc., Washington, DC.

Introduction: Smith-Magenis Syndrome (SMS) is a rare (1/15,000 - 25,000 births) neurodevelopmental disorder resulting from an interstitial deletion of chromosome 17p11.2, or from a point mutation in the *RAII* gene. Severe sleep disorder is almost universal in patients with SMS and poses a significant challenge to patients and their families. Tasimelteon improved sleep symptoms in a randomized, double-blind, two-period, crossover study; and here we show that this effect persists for up to four years in an open-label extension. To our knowledge, this is the largest interventional study of SMS patients to date.

Methods: Following the 4-week crossover study, all eligible participants had the option to enroll in an open-label extension. 31/39 (79.4%) of all individuals who participated in the efficacy study have continued on tasimelteon treatment. Participants in the open-label extension provided daily diary sleep quality (DDSQ), and

daily diary total sleep time (DDTST) measures via parental post sleep questionnaire and characterized behavior using the Aberrant Behavior Checklist (ABC).

Results: In the open-label extension, tasimelteon continued to show improvement in the primary endpoints of 50% worst sleep quality (mean = 0.7, SD = 0.94) and 50% worst total nighttime sleep duration (mean = 53.3, SD = 59.01) when compared to baseline. Tasimelteon also improved overall sleep quality (mean=0.7, SD=0.83) and overall total nighttime sleep duration (mean = 51.9, SD=53.03). ABC scores also improved with tasimelteon (mean=-16.3, SD = 15.82).

Conclusion: Tasimelteon continues to demonstrate persistence in efficacy (longest approximately 4 years) with similar magnitudes observed in the 4-week crossover study for sleep quality and total sleep time. Interestingly, daytime behavior also demonstrates long-term improvement in patients with SMS treated with tasimelteon. These results further confirm tasimelteon as a novel therapy for the treatment of sleep disorders in patients with SMS and may provide benefit for behavioral symptoms.

Support: This work was supported by Vanda Pharmaceuticals Inc.

1162

FINANCIAL SAVINGS AND IMPROVED OUTCOMES FROM AN INNOVATIVE, INTEGRATED SLEEP APNEA MANAGEMENT PROGRAM FOR A SAFETY NET POPULATION: 5-YEAR RESULTS

Lim, M. S.

Redwood Pulmonary Medical Associates, Redwood City, CA.

Introduction: Sleep disorders are extremely common in the general population and are associated with an increased risk for fatal accidents, heart disease, stroke, neurocognitive decline, and diabetes. Sleep problems disproportionately affect socioeconomically disadvantaged and under resourced communities. We present here a 5-year analysis of a private-public partnership between a private sleep medicine practice (Redwood Pulmonary Medical Associates, RPMA) and county medical system (the Health Plan of San Mateo) to provide a coordinated, value-based sleep apnea program to adult residents of San Mateo County, CA.

Methods: Referring providers send referrals to a single location (RPMA), and sleep consultations, testing, follow-up care, and CPAP management occur out of the same location by a dedicated staff. Limited channel cardiopulmonary (CP) sleep testing initiated in the office and completed at home was done for most patients. Patients with AHI>5 plus daytime sleepiness were offered nasal CPAP, unlimited mask fittings, and compliance checks.

Results: 2101 CP tests were successfully completed (93.5%). There were 49.6% females and 50.4% males, with an average age of 51.6 years. 31% had severe obstructive sleep apnea (OSA) and 59% were moderate or severe. 1471 (65%) of patients were given prescriptions for CPAP and 471 (32%) were still using CPAP at 5 years, 68.7% of whom were initiated within 3 years of the analysis. Of the patients returning satisfaction surveys, 97% would recommend the program to a relative or friend, and all respondents (99 to date) who were using CPAP felt it had benefitted their health overall. Comparing actual costs of the program to projected fee-for-service costs for the same services, the program saved the Health Plan of San Mateo 1,132,510, or 51.4%.

Conclusion: This program demonstrates the potential clinical and financial benefit of private-public partnerships in administering clinical programs to high risk populations, as private businesses can quickly adapt to new technologies, financial metrics, and standards of care.

Support: No outside financial support was provided

1163

NICKEL, DIME OR DOLLAR: BREAKING DOWN BROKEN NOTIONS OF COST IN SLEEP DIAGNOSTICS

Jambulingam, N.¹ Stretch, R.¹ Butz, D.² Zeidler, M.¹

¹UCLA, VA Greater Los Angeles Healthcare System, Los Angeles, CA, ²University of Michigan Ross School of Business, Ann Arbor, MI.

Introduction: Home sleep apnea tests (HSATs) are convenient alternatives to in-lab polysomnograms (PSGs) but high nondiagnostic rates limit their utility. A clinical decision support tool (CDST) to triage patients to HSAT versus PSG was developed at the Greater Los Angeles VA Healthcare System (GLA-VAHS). It uses a random forest ensemble to reduce non-diagnostic HSAT rates by 46%. While prior studies have found PSGs to be more profitable than HSATs on a *per unit* basis, these analyses do not factor in relative profitability over time. Additionally, no prior studies have quantified the financial impact of a CDST in diagnostic sleep testing.

Methods: We performed an analysis of the overall profitability of HSATs and PSGs in 2018-2019 within GLA-VAHS which has 6 PSG beds. Revenue was calculated using 2019 Medicare reimbursement rates. Contribution margin (CM) analysis was used to factor out the high fixed costs of healthcare infrastructure, instead focusing on variable direct costs (VDCs). CM analysis is especially useful when calculated on a *per diem* basis instead of *per study*, adjusting for number of tests performed in a given day. CM was calculated by subtracting VDCs from revenue under two simulated conditions: with and without the CDST.

Results: PSGs were 2.5 times more profitable than HSATs on a *per unit* basis (CM \$200/study vs. \$81/study). However, on a *per day* basis, PSGs were only 1.4 times more profitable than HSATs at average nightly occupancy rates of 75% (CM \$902/day vs. \$646/ day). Using the CDST to guide testing, 2.2 times more diagnostic HSATs could be performed per day. As a result, HSATs were 1.3 times more profitable than PSGs on a *per day* basis with CDST use (CM \$1,211/day vs. \$902/day).

Conclusion: This analysis demonstrates that implementing a CDST and maximizing utilization of HSATs allow hospitals to better allocate limited sleep lab resources, increase diagnostic throughput and generate higher profits. Analyzing costs using contribution margin avoids erroneous assumptions about profitability and leads to better-informed administrative decisions regarding sleep lab expansion.

Support:

1164

PROSPECTIVE ASSOCIATIONS BETWEEN SLEEP DURATION, VARIABILITY AND TIMING AND DISEASES FROM AN ELECTRONIC HEALTH RECORD BIOBANK IN 24,065 INDIVIDUALS

Dashti, H. S.¹ Cade, B.² Stutaite, G.¹ Saxena, R.¹ Redline, S.² Karlson, E.²

¹Massachusetts General Hospital, Boston, MA, ²Brigham and Women's Hospital, Boston, MA.

Introduction: Implementation of electronic health records (EHR) across healthcare systems linking clinical to survey data has enabled systematic assessments of longitudinal relationships between sleep traits and diseases classified by PheWAS codes where ICD-9/10 codes are collapsed to categories based on clinical similarity. In the Partners Biobank, a hospital-based virtual cohort from Mass General Brigham in greater Boston, MA, we aimed to assess associations between sleep traits and incident diseases.

Methods: Self-reported weekday/weekend bed and wake times from a survey at consent were used to derive sleep traits. Incident diseases were defined as two incident PheWAS codes on separate dates $\geq 1y$ after consent. Cox proportional hazards models compared short (<7h) and long (\geq 9h) sleep duration, with 7-8h (referent group), adjusted for age, gender, race/ethnicity, and employment status, then further adjusted for BMI. Similarly, sleep midpoint (midpoint between weekend wake/bed times), sleep debt (difference in weekend/weekday sleep duration), and social jetlag (difference in weekend/weekday sleep midpoint) were assessed.

Results: The analytical sample consisted of 24,065 adults (mean sleep duration =8.12h) seeking regular care with sleep data. Participants had a total of 7,513,649 ICD codes of which incident 323,946 ICD codes mapped to 137,137 PheWAS codes. Over a median follow-up of 2.73 years (interquartile range: 1.82-3.98),

participants sleeping <7h had a significantly higher risk of incident Acute pain [hazard ratio(95% confidence interval)=1.46(1.2-1.78)], Tobacco use disorder [1.42(1.18-1.71)], Sciatica [1.72(1.3-2.27)], and Edema [1.69(1.25-2.28)]. Each additional hour of later sleep midpoint and increased sleep debt and social jetlag associated with higher risk of incident Major depressive disorder [midpoint:1.30(1.14-1.49); debt:1.23(1.09-1.38); jetlag:1.54(1.27-1.84)]. Associations retained significance upon further adjustment for BMI, except for Edema, and no other associations were observed at the Bonferroni threshold (P=0.0125).

Conclusion: Our findings in a large hospital-based virtual cohort support unique inter-relationships between sleep duration/timing on somatic, behavioral, and mental health outcomes.

Support: H.S.D. and R.S. are supported by NIDDK grant R01DK107859. B.C. is supported by K01-HL135405-01. S.R. and R.S. are partially supported by R35 NHLBI HL 135816.

1165

ONLINE CME-CERTIFIED CASE CHALLENGES IMPROVE COMPETENCE FOR THE DIAGNOSIS AND MANAGEMENT OF PEDIATRIC NARCOLEPSY AMONG PEDIATRICIANS

Finnegan, T.¹ Murray, C. F.¹ Hughes, S.¹ Maski, K.² ¹Medscape Education, New York, NY, ²Boston Children's Hospital, Boston, MA.

Introduction: Narcolepsy is a chronic neurologic sleep disorder that typically starts in childhood. Symptoms of narcolepsy in pediatric patients can differ from adult onset narcolepsy and few treatment options are approved for pediatric narcolepsy. Given the challenges of recognizing the condition in children and selecting an appropriate therapeutic intervention, we investigated whether a case-based educational activity was able to improve the competence of pediatricians to accurately diagnose and manage narcolepsy.

Methods: An online, text-based educational intervention comprised of 2 patient case scenarios was developed. Using a "test and teach" approach, clinicians were presented with multiplechoice questions to evaluate their application of evidence-based recommendations. Each response was followed by detailed, referenced, feedback to teach. Educational effect was evaluated with a repeated-pairs pre- to post-assessment study design in which each individual learner acts as his/her own control. A chi-square test was utilized to identify whether proportions of correct answers at pre and post were significantly different. Cramer's V was used to calculate the effect size of the intervention. Data were collected between April 20, 2019 and September 17, 2019.

Results: The education resulted in an extensive educational effect for pediatricians (n=125; V = .424). Significant improvements were observed in several topics (P < .05 for all comparisons) including: the use of hypocretin cerebrospinal fluid testing as a diagnostic tool for patients with symptoms suggestive of type 1 narcolepsy; appropriate guidance to transition patients with type 1 narcolepsy from one therapeutic regimen to another; and therapeutic selection for a patient with type 2 narcolepsy. Overall, participation in the education resulted in 34% of pediatricians reporting increased confidence in diagnosing and managing sleep disorders in children. Conclusion: This study demonstrated the success of a targeted, online, interactive, case-based educational intervention on improving awareness among pediatricians regarding the diagnosis and management of narcolepsy. The results indicated that pediatricians would benefit from continued education on the care of patients with narcolepsy.

Support: Support for this program came from an unrestricted educational grant from Jazz Pharmaceuticals, Inc.

1166

INVESTIGATING SOCIAL WORKERS' SLEEP HEALTH KNOWLEDGE: OPPORTUNITIES TO PROMOTE SLEEP HEALTH AMONG UNDERSERVED POPULATIONS

Spadola, C. E.¹ Groton, D.¹ Lopez, R.¹ Burke, S. L.² Hilditch, C.³ Pandey, A.⁴ Littlewood, K.⁴ Zhou, E. S.⁵ Bertisch, S. M.⁵ ¹Florida Atlantic University, Boca Raton, FL, ²Florida International University, Miami, FL, ³3Fatigue Countermeasures Laboratory, San José State University Research Foundation, Moffett Field, CA, ⁴University of South Florida, Tampa, FL, ⁵Divison of Sleep Medicine, Harvard Medical School, Boston, MA.

Introduction: Social workers are often front-line psychosocial providers working with underserved populations, many of whom struggle with sleep. They are uniquely positioned to promote sleep health among individuals experiencing health inequities. However, U.S. accredited social work programs do not require sleep health training. We used both quantitative and qualitative methodologies to investigate social work students': a) sleep health knowledge; b) self-reported sleep quality; c) prior sleep health education; and d) client discussions about sleep, in order to inform the development of a sleep health training for social work students.

Methods: Twenty-five social work students were recruited via a listserv email sent at a Florida university. Participants were asked to complete the Sleep Beliefs Scale (SBS) and the Pittsburgh Sleep Quality Index (PSQI) and then to participate in a one-hour long focus group (3 groups with 6-11 students/group) conducted by experienced qualitative researchers.

Results: Mean age was 27.0 ± 11.5 yrs, 92.0% were female, and 48.0% were non-Hispanic white, 28.0% African American, 16.0% Hispanic, 8.0% other. Only 28.0% indicated that they had ever discussed sleep with clients. Knowledge of healthy sleep behaviors (assessed via the SBS) was moderate on a 0-20 scale (13.88, S.D.= 2.7). Participants had an average PSQI score of 8.8 (SD.=4.0), reported sleeping an average of 6.0 hours (SD=1.6), and mean sleep efficiency of 87.0% (SD=12.0). Themes from focus group data highlight students' lack of exposure to sleep health training and a dearth of sleep discussions in clinical practice.

Conclusion: Though social work students acknowledged the importance of sleep health promotion, they reported feeling ill-equipped to promote healthy sleep practices due to lack of sleep education. Sleep health training could allow social workers to confidently promote healthy sleep practices among their clients, recognize when appropriate to refer clients for evaluation for sleep disorders, and improve social workers' own sleep health. An online educational program was subsequently created by study investigators to meet these aims. **Support:** American Academy of Sleep Medicine Foundation

1167

NURSING PERCEPTIONS OF SLEEP ASSESSMENT IN THE INTENSIVE CARE UNIT

Heavner, M. S.¹ Jobe, S. L.² Hurley, J.³ Le, B.⁴ Kantner, C.⁴ Heavner, J. J.³ Shanholtz, C.² Verceles, A.² Wickwire, E. M.² ¹University of Maryland School of Pharmacy, Baltimore, MD, ²University of Maryland School of Medicine, Baltimore, MD, ³University of Maryland Baltimore Washington Medical Center, Glen Burnie, MD, ⁴University of Maryland Medical Center, Baltimore, MD.

Introduction: Sleep disruption in intensive care unit (ICU) patients is highly prevalent and may contribute to adverse clinical outcomes. Although clinical practice guidelines recommend routine assessment of sleep, surveys of ICU clinicians indicate that sleep assessment programs (SAP) are rarely implemented. The purpose of the current project was to characterize sleep-related perceptions, practices, and knowledge among ICU nurses, to identify barriers and facilitators of implementation of a systematic SAP.

Methods: A 29-item, paper-based survey was administered to all nurses (N=220) in a medical ICU at a 750-bed academic medical center and a mixed ICU in a 300-bed community hospital. Voluntary survey completion was conducted over four weeks. Descriptive statistics were employed.

Results: A total of 163 surveys were completed (74.1%). Participants were primarily female (n=135; 82.8%), day-shift nurses (n=83; 50.9%), with 2-5 years of ICU experience (n=70; 42.9%). Respondents said they sometimes (n=52; 31.9%), and often (n=52; 31.9%), assess sleep, and 76.1% (n=124) reported not assessing sleep formally in the last three shifts. Approximately half of the respondents (n=85; 52.1%) were not aware of clinical practice guideline recommendations for sleep in the ICU. Most nurses reported that their unit could benefit from a SAP (n=101; 62.0%) and that they should have a primary responsibility in ensuring that sleep is discussed daily (n=144; 88.3%).

Conclusion: Despite published guidelines recommending routine sleep assessment, ICU nurses report infrequent assessment and a lack of awareness of these guidelines. However, ICU nurses believe implementation of routine sleep assessments would be beneficial to patient care. This suggests that SAP implementation would be positively received by ICU nurses. Future studies evaluating knowledge and site-specific perceptions and practices, as well as nursing staff characteristics, can further guide implementation of SAPs in the ICU.

Support:

1168

PRELIMINARY IMPACT OF A SLEEP HEALTH EDUCATIONAL MODULE FOR SOCIAL WORK STUDENTS

Spadola, C.¹ Groton, D.¹ Lopez, R.¹ Burke, S. L.² Hilditch, C. J.³ Pandey, A.⁴ Littlewood, K.⁴ Zhou, E. S.⁵ Bertisch, S. M.⁶ ¹Florida Atlantic University, Boca Raton, FL, ²Florida International University, Miami, FL, ³San Jose State University Research Foundation, Moffett Field, CA, ⁴University of South Florida, Tampa, FL, ⁵Dana-Farber Cancer Institute and Boston Children's Hospital, Boston, MA, ⁶Brigham and Women's Hospital, Boston, MA.

Introduction: Social workers are well-positioned to promote healthy sleep behaviors among underserved populations; however sleep health training is rarely integrated into social work curriculums. To address this gap, our interdisciplinary team developed a 2-hour online sleep health educational module for social work students. The module was grounded in best e-learning pedagogical principles, and based on qualitative formative research. We tested the initial impact and acceptability of the module.

Methods: We recruited 32 social work students at a Florida University via a departmental listserve. Pre- to post-intervention changes in the Sleep Beliefs Scale (SBS) and the Sleep Practices and Attitudes Questionnaire (SPAQ) were assessed using Wilcoxon Signed-Rank tests. We conducted qualitative research to assess intervention acceptability, and to inform future iterations of the program.

Results: Mean age was 29.5±11.6 yrs, 100% were female, and primarily Non-Hispanic White (41.9%), followed by African American/Black (35.5%), and Hispanic/Latino (22.6%). Results showed pre/post intervention improvements in both the Sleep Beliefs Scale (14.7 ± 2.2 vs. 16.9 ± 2.6 [p=.002]; higher score=higher knowledge) and SPAQ (2.1 ± 0.6 to 1.5 ± 0.6 [p=.001]; lower score=higher importance of sleep) indicating improvements in knowledge surrounding healthy sleep behaviors and the importance of sleep for overall health (respectively). Qualitative data supports the intervention's acceptability and utility. When asked what participants liked best about the module, responses included: "I was educated and am better prepared to offer some insight to my clients and staff"; "..they offer resources we can use for ourselves and our clients;" and "easy to navigage, and full of useful information." Suggestions for improvement included shortening the module's length.

Conclusion: Assessment of an online sleep health educational module indicates a promising impact on sleep health knowledge. A larger study is planned to more definitively evaluate the module's impact and acceptability among social work students.

Support: American Academy of Sleep Medicine Foundation

1169

INNOVATIVE EXPOSURE TO SLEEP MEDICINE FOR MEDICAL STUDENTS

Gupta, S.¹ Golden, E.¹ Howell, M.³ Irfan, M.⁴

¹Hennepin County Medical Center, Minneapolis, MN, ²Hennepin County Medical Center, Minneapolis, MN, ³University of Minnesota, Minneapolis, MN, ⁴Minneapolis Veteran Affairs Medical Center, Minneapolis, MN.

Introduction: Lack of exposure to the field of Sleep Medicine at the medical student level hinders sleep training. Instead of the traditional didactic style, there is a need for innovative collaborative measures to spark interest in the younger generation of learners. The goal of this educational endeavor was to introduce medical students to the field of Sleep Medicine through the platform of Student Interest Group in Neurology (SIGN).

Methods: An interactive session was conducted for SIGN at the University of Minnesota. 24 second-year medical students were divided into 6 groups. The session consisted of introduction, videos of common sleep disorders and interactive briefing afterward. 5-point Likert scale pre and post-session surveys were administered to measure the level of knowledge regarding sleep, familiarity with diagnostic tools, available education, pathways to Sleep Medicine, learner's interest and impact of the session. Wilcoxon matched-pairs signed-rank test was performed to compare pre- and post-surveys.

Results: There was a significant improvement in measures of students' knowledge about sleep diagnostic modalities ($p = 7.8*10^{-5}$), education received ($p= 3.2*10^{-5}$) and pathways to sleep medicine ($p=4.1*10^{-5}$). Survey also showed improvement in students' interest in pursuing a Sleep Medicine career (p=0.07). There was no difference in knowledge about the importance of sleep for health (p=0.69). All of the students found the session to be informative.

Conclusion: Early exposure to sleep disorders in interactive format was well received by the medical students with significant improvement in scores regarding sleep education, awareness of diagnostic modalities, career pathway and interest in sleep medicine (p=0.07). Integration of exposure to Sleep Medicine within the medical curriculum in an innovative format should be done to instigate interest

in this field. Further larger studies are warranted to evaluate the changes in the students' interest in the subspecialty with an introduction in the early stages of their career. **Support:**

1170

TRENDS IN SLEEP APNEA TESTING AMONG VETERANS PARTICIPATING IN A RURAL HEALTH-FOCUSED TELESLEEP MEDICINE PROGRAM

Atwood, C. W.¹ Boudreau, E.² Folmer, R.² Kuna, S. T.³ Pineda, L.⁴ Reichert, J.⁵ Sarmiento, K.⁵ Thompson, W.⁶ Whooley, M.⁵ Zhang, N.⁵ Yarbrough, W. C.⁷

¹VA Pittsburgh Healthcare System, VA Pittsburgh Healthcare System, PA, ²VA Portland Healthcare System, Portland, OR,
³Cpl Michael J Crescenz VA Medical Center, Philadelphia, PA,
⁴VA Phoenix Healthcare System, Phoenix, AZ, ⁵San Francisco VA Medical Center, San Francisco, CA, ⁶Boise VA Medical Center, Boise, ID, ⁷Dallas VA Medical Center, Dallas, TX.

Introduction: The Department of Veterans Affairs has pioneered the use of home sleep apnea testing (HSAT) across many of its medical centers over the past 15 years. Here we report trends regarding diagnostic sleep apnea testing in rural and urban Veterans served by the TeleSleep Program, a VA telehealth initiative focused on increasing access to sleep care for rural Veterans. Rurality is a risk factor for use of polysomnography and is associated with longer wait times for testing and initiation of PAP therapy.

Methods: We used a VA administrative database search of patients enrolled in sleep medicine clinics from fiscal years (FY) 2016-2019 at 7 TeleSleep Hubs: San Francisco, Portland, Phoenix, Boise, Philadelphia, Spokane, and Pittsburgh. Individual encounters were coded locally and transmitted to VA's corporate data warehouse. HSAT codes included 95800, 95801, 95806, G0398, G0399, and G0400. Polysomnography codes included 95807, 95808, 95810, and 95811.

Results: Total number of unique Veterans served increased between FY16 and FY19 from 28,941 to 43,044 (149%); rural Veterans served during this time increased from 9,386 to 14,329 (153%). The total number of annual sleep medicine encounters for all Veterans served increased from 89,870 to 138,127 (154%); rural Veteran visits increased from 29,825 to 50,342 (169%). Unique urban Veterans tested by HSAT increased from 2,158 in FY16 to 4,398 in FY19 (203%) while polysomnography decreased from 5,011 to 3,253 (35%). Unique rural Veterans tested by HSAT increased from 1,102 to 2,768 (251%) and polysomnography decreased by 42% (1,565 to 909 Veterans) during this same time.

Conclusion: Among VA sleep medicine programs with TeleSleep funding, HSAT became the most common approach to diagnostic sleep apnea testing, particularly in rural Veterans. Although polysomnography was widely used at the beginning of the TeleSleep Initiative, use declined as HSAT became more widely implemented. **Support:** VA Office of Rural Health

1171

COMMONLY ENCOUNTERED YET NOT CONFIDENT: GRADUATE PSYCHOLOGY STUDENTS' EXPERIENCE AND CONFIDENCE MANAGING SLEEP DISTURBANCES

Meaklim, H.¹ Monfries, M.¹ Rehm, I. C.¹ Junge, M.² Meltzer, L. J.³ Jackson, M. L.⁴

¹RMIT University, Bundoora, AUSTRALIA, ²Sleep Health Foundation, Blacktown, AUSTRALIA, ³National Jewish Health, Denver, CO, ⁴Monash University, Clayton, AUSTRALIA. **Introduction:** Trainee psychologists receive minimal sleep education during graduate psychology training programs, despite the frequent co-occurrence of sleep disturbances in mental health conditions. This study aimed to explore graduate psychology students' experience working with sleep disturbances and their perceived skills and confidence to assess and treat sleep problems in clinical practice.

Methods: Australian graduate psychology students (N = 163) completed a novel survey developed specifically for the study, inquiring about their experience, skills and confidence to manage sleep disturbances in clinical practice. Students perceived skills to manage sleep disturbances were recorded on a 7-point Likert scale, where 1 = 'strongly disagree' and 7 = 'strongly agree'. Students' confidence to treat specific sleep disorders was also recorded.

Results: Sixty-eight percent of students reported having already worked with a client who experienced a sleep disturbance as part of their training. However, students' perceived skills to assess and treat sleep disturbances were low. Only 14.9% 'agreed' or 'strongly agreed' that they had the skills to assess and diagnose common sleep disorders (M = 3.22, SD = 1.75). Similarly, less than a quarter of students 'agreed' or 'strongly agreed' that they felt comfortable using common sleep-related assessments (23.7%; M = 3.56, SD = 1.96) or empirically-supported interventions for sleep disturbances (22.6%; M = 3.71, SD = 1.83). The majority of students reported they were 'not confident at all' to treat parasonnias (80.3%); hypersonnias (77.9%); OSA (71.3%); circadian rhythm disorders (50%) or insomnia (41%).

Conclusion: Graduate psychology students report low levels of confidence in assessing and managing sleep disturbances in clinical practice, despite over two-thirds of students already working with clients experiencing sleep difficulties. Clinical training in the management of sleep disturbances is required for graduate psychology students.

Support: N/A

1172

SLEEP EDUCATION IMPROVES SCREENING FOR SLEEP DISORDERS AMONG PHYSICIANS AND RESIDENTS IN PRIMARY CARE AND NEUROLOGY SPECIALTIES

Jain, S. V.¹ Kondapalli, K.² Moskowitz, A.² Combs, D.¹ Parathasarathy, s.¹

¹Banner University Medical Center, Tucson, AZ, ²University of Arizona, Tucson, AZ.

Introduction: Sleep disorders are under-diagnosed. The purpose of the study was to evaluate if providing sleep education improves screening and thereby, diagnosis of sleep disorders among physicians.

Methods: The study was approved by the institutional review board. Pediatric (P), neurology (N) and internal medicine (IM) physicians/residents participated in the study. After collecting demographics, including baseline screening rates (BSR), the participants were randomized to educational intervention- either manuscript or oral presentation reviewing insomnia, obstructive sleep apnea and sleep disorders in epilepsy. Questionnaires-baseline knowledge (BQ), post-intervention (PQ) and 9 months after the intervention, screening (SQ) evaluating screening rates (PSR) and usefulness of the study, were collected. The change in knowledge (PQ-BQ) and screening rates (PSR-BSR) were compared between the groups by t-test and ANCOVA after adjusting for BSR. t-tests were used to compare the knowledge and screening rates before

and after the intervention in the entire cohort. Correlations identified the factors associated with improved screening rates.

Results: Thirty and 23 subjects completed the study and SQ, respectively. The average age was 30.4 (standards deviation [SD]= 3.1) with 53% female and practice experience of 2.5 years (SD=1.7) with 60% P, 23.3% N and 16.7% IM participants. There were no significant differences in the characteristics, and knowledge and screening rate change after the intervention between the two groups. However, for individual questions, the oral presentation group had improved knowledge about sleep disorders in epilepsy. The study improved knowledge by 82% for understanding, 77% for managing and 79% for screening for sleep disorders. Knowledge (Mean Difference [MD]= 0.22, p=0.0001) and screening rates (MD=0.29, p=0.001) improved significantly after the intervention in the entire cohort. The improvement in screening rates was associated with presence of screening by the physicians prior to the intervention (r=0.5, p=0.008).

Conclusion: The study showed that sleep education improved knowledge and screening for sleep disorders among physicians.

Support: The study was funded by the American Academy of Sleep Medicine Foundation Focused Project Award.

1173

REDUCING CANCELLATION AND IMPROVING THE PATIENT'S EXPERIENCE THROUGH QUALITY IMPROVEMENT PROCESS

Mullen, L. Cole, A. Parsons, J. Sabla, G. Tiemeyer, K. Simakajornboon, N.

Cincinnati Children's Hospital, Cincinnati, OH.

Introduction: Sleep study volume in our system has increased by 23% from 2017 to 2019 which makes unfilled sleep beds a significant concern. Cancellation rate impacts our sleep bed access. We hypothesized cancellation can be improved through quality improvement process which could ultimately lead to improve patient satisfaction.

Methods: A multi-disciplinary team was assembled to examine potential contributing factors. Using the Model for Improvement we developed, tested and implemented interventions using tools such as PDSA cycles, process map and a simplified FMEA (Failure Modes and Effects Analysis). A Key Driver Diagram helped guide our journey to improve the cancellation rate. We developed a Parent Advisory Group to help us with ideas to identify how we could improve the cancellation rate.

Results: The cancellation was 21% prior to the implementation of our interventions. To improve these measures, we have implemented several interventions. The content of our sleep study preparation handbook was improved, increased distribution of the education handbook and developed a series of sleep study videos which are available for viewing prior to the study to prepare patients and families. In addition, we standardized our process of reminder calls in the call center and sleep lab by defining roles and responsibilities. This improved our ability to answer questions and identify and mitigate barriers they may have. We implemented transportation assistance to patients who have transportation barriers and created a waitlist protocol to assist families with a preferred date. After 2 years of interventions, the cancellation rate has decreased from 21% to 14.7%. Interestingly, as we improved our cancellation rate, the overall patient satisfaction has been improved from 83% to 88%. Conclusion: Using the Model for Improvement, we improved education, communication and scheduling processes, which has reduced cancellation rate and consequently improved patient satisfaction.

Support:

1174

ASSESSING SLEEP DISORDERS IN PRIMARY CARE: A PROVIDER SURVEY ABOUT THE IMPORTANCE OF SLEEP HEALTH

Klingman, K. J.¹ Morse, A.² Williams, N.³ Grandner, M. A.⁴ Perlis, M. L.⁵

¹Upstate Medical University, The State University of New York, Syracuse, NY, ²Geisinger Medical Center, Danville, PA, ³NYU Langone Health, Department of Population Health, New York, NY, ⁴Department of Psychiatry, Tucson, AZ, ⁵Department of Psychiatry, Director of Behavioral Sleep Medicine Program, Philadelphia, PA.

Introduction: Conditions commonly managed by primary care providers (PCPs) such as depression, diabetes, and heart disease, commonly co-occur with sleep disorders. If PCPs could readily identify comorbid sleep disorders in this context, it may provide a pathway to more effective management of both types of disorders. Currently, it is unknown what might encourage or discourage PCPs from routinely screening their patients for sleep disorders.

Methods: PCPs from UPENN and GHS completed surveys regarding sleep health. The 30-item instrument comprised demographic, 14 VAS (0%-100%=strongly disagree-strongly agree), 4 open-ended, 3 yes/no, and 2 multiple-choice questions.

Results: Ninety-nine PCPs responded and were predominately female (61% F, 37%M, 2% other), Caucasian (81%), on-average 45yrs old (25-70) and in primary care for 16yrs (1-43). Fifty-six percent were MDs, 21%DOs, 17%PAs, and 6%NPs. PCPs rated sleep disorders as highly important for cardiopulmonary, mental, and general health (85, 84, & 83%), with no difference (per linear regression, p>0.05) according to system or provider characteristics. PCPs reported high importance for knowing about and diagnosing sleep disorders (88% & 82%) within their practices. Lower comfort levels were reported for discussing (78%) sleep disorders, overseeing/following (62%), diagnosing (60%), or treating (48%) patients. Eighty percent of PCPs stated an efficient sleep disorders screener would be useful for their practice; this perception varied (per logistic regression) according to provider credentials (Wald=0.037) and Hispanic/Latino ethnicity (Wald=0.025). PCPs reported time constraints limit their responsiveness to sleep disorders

Conclusion: A large disparity exists between the importance PCPs place on sleep disorders and their low comfort levels with following, diagnosing, and treating sleep disorders. PCPs endorsed the need to have available an efficient sleep disorders screener to use in their practice.

Support: No funding was received for this study.

1175

SLEEP DISORDERS SCREENING IN PRIMARY CARE: PREVALENCE OF DIAGNOSIS AND TREATMENT IN THE EMR

Klingman, K. J.¹ Morse, A.² Williams, N.³ Grandner, M.⁴ Perlis, M. L.⁵

¹Upstate Medical University, The State University of New York, Syracuse, NY, ²Geisinger Medical Center, Danville, PA, ³NYU Langone Health, Department of Population Health, New York, NY, ⁴Department of Psychiatry, Tucson, AZ, ⁵Department of Psychiatry, Director of Behavioral Sleep Medicine Program, Philadelphia, PA.

Introduction: Undetected and untreated sleep disorders likely precipitate or exacerbate medical and/or psychiatric illnesses. Given this, primary care is an ideal point for managing sleep disorders, yet prior research shows that PCPs diagnose and/or treat sleep disorders at rates far below population prevalences. The purpose of this study was to determine the current rate of detection and treatment of sleep disorders within primary care settings.

Methods: EMR data from two health care systems was analyzed. The proportion of PCPs diagnosing and treating one or more sleep disorders was calculated (per year) for 5 years (2014-2018). Also calculated was the percent of PCP caseload diagnosed and/or treated for sleep disorders.

Results: The two systems comprised n=1021 PCPs. From 2014-2018, the proportion of PCPs diagnosing patients with sleep disorders fluctuated between 58-89%. The proportion treating sleep disorders fluctuated between 50-91%. Non-parametric one-sample run tests (SPSS) indicate these are random distributions (p>0.05). PCPs' use of medications to treat sleep disorders is trending downward over time within one system (per linear regression, p=0.03, R-squared=0.8). Other temporal trends were not evidenced. The average percentage of diagnosed and treated patients per PCP was around 2.5% of their caseloads. Between-system differences were observed.

Conclusion: There is a profound mismatch between percentage of PCPs identifying patients with sleep disorders (60-90%) and the percentage of patient caseload diagnosed and/or treated for sleep disorders (2.5%). This suggests that the majority of PCPs are willing to assess for sleep health but do so in only a small minority of patients. These data, along with our survey data (elsewhere in this volume) suggest that the intention-action gap could be closed if PCPs were appropriately resourced.

Support: There was no funding for this study.

1176

SURVEY OF KNOWLEDGE OF PEDIATRIC TRAINEES REGARDING OBSTRUCTIVE SLEEP APNEA IN CHILDREN

Gummalla, P.¹ Lee, S.² Soriano, S.³ Concepcion, E.³ ¹Stonybrook sleep disorders center, Smithtown, NY, ²Texas children's hospital, Houston, Texas, Texas children's hospital, TX, ³SUNY Downstate hospital, SUNY Downstate hospital, NY.

Introduction: Obstructive sleep apnea (OSA) causes significant morbidity in children. Very few studies were conducted among pediatricians regarding sleep and sleep disorders in children and adolescents. They demonstrated significant gaps in knowledge in the recognition and management of OSA.

Methods: We conducted a survey among the pediatric residents in the screening and diagnosis of OSA. The survey consist of 15 questions related to OSA in children. It aimed at identifying gaps in their knowledge and educating them accordingly. Following the survey, core lectures were included in the pediatric curriculum which aimed at educating them with the causes, diagnosis, management and sequelae of untreated OSA.

Results: 37 pediatric residents took part in the survey. They were from first, second and third year of the pediatric residency training program respectively in an university hospital. 45% of them belong to the first year, 29% of them belong to the second year and 24% of them belong to the third year of training.Only a quarter of them had completed rotations in Pediatric Pulmonology for at least two weeks at the time of the survey. Three questions related to identifying at risk groups, four questions related to identification

of the signs and symptoms, and three questions related to the diagnostic criteria and five questions related to the treatment and follow up of OSA.

46% of the residents accurately answered the questions related to screening and identifying children at risk of OSA. 57% of them identified the signs and symptoms of OSA in children.41% of them answered the questions related to diagnosis appropriately and 49% had correct responses for the questions related to treatment of OSA and follow up.

Conclusion: Less than 50% of the pediatric trainees could accurately identify the at risk groups and symptoms of OSA. These pediatric trainees who are future pediatricians form the referral source for children with OSA and educating them is vital. This reemphasizes the need for the pediatric training programmes to include mandatory training modules related to Pediatric sleep disorders, OSA in particular in the pediatric core curriculum. **Support:** None

1177

COST EFFECTIVENESS OF DIAGNOSTIC APPROACHES TO SLEEP APNEA EVALUATION DURING INPATIENT REHABILITATION FOR MODERATE TO SEVERE TBI

Nakase-Richardson, R.¹ Dismuke-Greer, C.² Jeanne, H.³ Drashser-Phillips, L.¹ Schwartz, D.¹ Calero, K.⁵ Bogner, J.⁶ Whyte, J.⁷ Almeida, E.⁸ Ketchum, J.⁹ Magalang, U.¹⁰

¹James A. Haley Veterans Hospital, Tampa, FL, ²VA Palo Alto Healthcare System, Palo Alto, CA, ³University of Washington, Seattle, WA, ⁴James A. Haley Veterans Hospital, Tampa, FL, ⁵University of South Florida Morsani College of Medicine, Tampa, FL, ⁶Department of PMR, Ohio State University, Columbus, OH, ⁷Moss Rehabilitation Research Institute, Philadelphia, PA, ⁸Craig Hospital Department of Research, Denver, CO, ⁹Craig Hospital, Department of Research, Denver, CO, ¹⁰Division of Pulmonary, Critical Care, and Sleep Medicine and Neuroscience Research Institute, The Ohio State University Wexner Medical Center, Columbus, OH.

Introduction: Recent work has highlighted prevalent obstructive sleep apnea (OSA) after traumatic brain injury (TBI) when patients are vulnerable to disruption of neural repair. The recently completed clinical trial comparing screening and diagnostic tools for OSA during inpatient rehabilitation provided the opportunity to conduct economic modeling of phased approaches using actual trial findings to address one perspective (the payor) on the value of phased testing.

Methods: A cost-effectiveness analysis of four phased approaches to OSA diagnosis including initial utilization of portable sleep monitoring [HSAT] regardless of pre-test probability, determination of pre-test probability using two prediction models [STOPBANG, MAPI], and initial assessment using Level 1 polysomnography was conducted. The analyses were modeled assuming all participants were considered high risk thus a negative screen or portable diagnostic test would result in a participant being referred for Level 1 polysomnography. The cost aversion used in analyses were derived from a recent white paper on the economic modeling of untreated OSA. Trial data from 214 participants were used in analyses (mean age 44 [SD 18], 82% male, 75% white, with primarily motor-vehicle related injury [44%] and falls [33%] with a sample mean emergency department Glasgow Coma Scale of 8 (SD 5).

Results: At AHI ≥15 (33.6%), the prediction models (STOPBANG [-\$5,291], MAPI [-\$5,262]) resulted in greater cost savings and

effectiveness relative to the HSAT approach (-\$5,210) and initial use of Level 1 PSG (-\$5,011). Sensitivity analyses at AHI \geq 5 (70.1%) revealed the initial use of HSAT (-\$6,322.85) relative to the prediction models (MAPI [-\$6,249.71], STOPBANG [-\$6,237) and initial assessment with Level 1 PSG (-\$5,977) resulted in greater savings and cost effectiveness.

Conclusion: The high rates of sleep apnea after TBI highlight the importance of accurate diagnosis and treatment of this comorbid disorder. However, financial and practical barriers exist to obtaining an earlier diagnosis during inpatient rehabilitation hospitalization. Diagnostic cost savings are demonstrated across all phased approaches and OSA severity levels with the most cost-effective approach varying by incidence of OSA.

Support: PCORI (CER-1511-33005), GDHS (W91YTZ-13-C-0015; HT0014-19-C-0004)) for DVBIC, NIDILRR (NSDC Grant # 90DPTB00070, #90DP0084, 90DPTB0013-01-00, 90DPTB0008, 90DPT80004-02).

1178

PREDICTORS OF BEING SEEN BY A BOARD-CERTIFIED SLEEP MEDICINE PROVIDER

Levri, J. M.¹ Jobe, S.¹ Albrecht, J.¹ Scharf, S.¹ Johnson, A.³ Wickwire, E.¹

¹University of Maryland School of Medicine, Baltimore, MD, ²University of Maryland School of Medicine, Baltimore, MD, ³University of Maryland School of Pharmacy, Baltimore, MD.

Introduction: Although several studies have evaluated the impact of board-certification in sleep medicine regarding obstructive sleep apnea treatment outcomes, no studies to date have identified predictive factors to determine which patients are evaluated by board-certified sleep medicine providers (BCSMP) in the clinical practice. Thus, the purpose of this study was to identify predictors of being seen by a BCSMP, relative to nonsleep specialist providers.

Methods: Our data source was a random 5% sample of Medicare administrative claims data from 2006-2013. Sleep disorder diagnoses such as insomnia, obstructive sleep apnea, restless legs syndrome, hypersomnias, and parasomnias, as well as medical comorbidities including cardiovascular, cerebrovascular, mood, pulmonary, and neurological disorders, were operationalized using International Classification of Diseases, Ninth Revision, Clinical Modification codes. Demographic data were obtained from the claims. BCSMP were identified using a novel cross-matching approach based on National Provider Identifier (NPI).

Results: A total of 57,209 Medicare beneficiaries received a sleep disorder diagnosis between 2006-2013, but only 1,279 (2.2%) of these individuals were ever seen by a BCSMP. Within a multivariate logistic regression model, male gender, asthma, and heart failure were significantly associated with being seen by a BCSMP. Additionally, BCSMP were more likely to evaluate patients with two or more sleep diagnoses.

Conclusion: Complexity of sleep disorders and cardiovascular and lung comorbidities were predictive of being seen by a BCSMP. These results demonstrate the importance of BCSMPs in caring for complex sleep medicine patients.

Support: This research was supported by an AASM Strategic Research Award from the AASM Foundation to the University of Maryland, Baltimore (PI: EMW).

1179

BARRIERS TO SLEEP AMONG CHILDREN EXPOSED TO ADVERSITY

Robbins, R.¹ Ripple, C. H.² Fleshman, C.³ Bonuck, K.⁴ Jean-Louis, G.⁵ Hale, L.⁶ McGlinchey, E.⁷ Donskoy, I.⁸ Wolfson, A.⁹ Owens, J.¹⁰

 ¹Brigham & Women's Hospital, Boston, MA, ²Pajama Program, New York, NY, ³Bradley Hospital Sleep Research Lab, Warren Alpert Medical School of Brown University, Providence, RI,
 ⁴University Center of Excellence at Montefiore Rose F. Kennedy Evaluation and Rehabilitation Center, Albert Einstein College of Medicine, The Bronx, NY, ⁵NYU Langone Health, New York, NY, ⁶Stony Brook University, Stony Brook, NY, ⁷Fairleigh Dickinson University, Madison, NJ, ⁸Advocate Children's Hospital, Chicago, IL, ⁹Loyola University Maryland, Baltimore, MD, ¹⁰Boston Children's Hospital, Boston, MA.

Introduction: Children exposed to adversity (e.g., homelessness, poverty) are at risk of poor sleep. Community settings that serve these children, both residential (e.g., foster care group homes, shelters) and non-residential (e.g., early care and education, schools), hold important understandings to the barriers to sleep they experience. We surveyed a heterogeneous national sample of community-based organizations (CBOs) in our Pajama Program sample to explore these barriers.

Methods: Pajama Program, a national 501(c)(3) nonprofit, administered an online needs assessment in May 2016 to staff at 3,911 CBOs. This poster reports on qualitative responses to the question, What are the primary barriers to sleep and bedtime among children your organization serves? Responses from residential and non-residential CBOs were analyzed separately. Two trained coders independently used the constant comparative method to analyze transcripts.

Results: Survey respondents (1,635) provided services in non-residential (42%), residential (18%) and combined (43%) settings. Organizations provided child welfare/foster care (20.6% of programs); transitional housing/shelter (20.5%); social services (15.6%); and early care and education (12.7%) services. Responses to the target openended item were from 127 non-residential and 55 residential programs. Sleep barriers common to both settings included: late/irregular bedtimes; no bedtime routine; lack of sleep education; housing/food insecurity; stress; disrupted/uncomfortable sleep; and adapting to new environments. Trauma was a barrier among residential (vs. non-residential) programs. Non-residential providers noted unstable family situations, bedding insecurity, and poor sleep hygiene.

Conclusion: Our research highlights barriers to sleep among children exposed to adversity, a largely understudied yet high risk group. Barriers reported by residential and non-residential CBOs were more similar than different. was Across program types and settings, CBOs expressed a need for sleep health education interventions for children and caregivers.

Support: Funding for this project was provided by Pajama Program, a national 501(c)(3) non-profit.

1180

THE USE OF NATURAL LANGUAGE PROCESSING TO EXTRACT DATA FROM PSG SLEEP STUDY REPORTS USING NATIONAL VHA ELECTRONIC MEDICAL RECORD DATA

Nowakowski, S.^{1,2} Razjouyan, J.^{1,2} Naik, A. D.^{1,2} Agrawal, R.^{1,2} Velamuri, K.^{1,2} Singh, S.^{1,2} Sharafkhaneh, A.^{1,2}

¹Baylor College of Medicine, Houston, TX, ²Michael E DeBakey VA Medical Center, Houston, TX, ³Michael E DeBakey VA Medical Center, Houston, TX, ⁴Baylor College of Medicine, Houston, TX.

Introduction: In 2007, Congress asked the Department of Veteran Affairs to pay closer attention to the incidence of sleep disorders among veterans. We aimed to use natural language processing (NLP), a method that applies algorithms to understand the meaning and structure of sentences within Electronic Health Record (EHR) patient free-text notes, to identify the number of attended polysomnography (PSG) studies conducted in the Veterans Health Administration (VHA) and to evaluate the performance of NLP in extracting sleep data from the notes.

Methods: We identified 481,115 sleep studies using CPT code 95810 from 2000-19 in the national VHA. We used rule-based regular expression method (phrases: "sleep stage" and "arousal index") to identify attended PSG reports in the patient free-text notes in the EHR, of which 69,847 records met the rule-based criteria. We randomly selected 178 notes to compare the accuracy of the algorithm in mining sleep parameters: total sleep time (TST), sleep efficiency (SE) and sleep onset latency (SOL) compared to human manual chart review.

Results: The number of documented PSG studies increased each year from 963 in 2000 to 14,209 in 2018. System performance of NLP compared to manually annotated reference standard in detecting sleep parameters was 83% for TST, 87% for SE, and 81% for SOL (accuracy benchmark \geq 80%).

Conclusion: This study showed that NLP is a useful technique to mine EHR and extract data from patients' free-text notes. Reasons that NLP is not 100% accurate included, the note authors used different phrasing (e.g., "recording duration") which the NLP algorithm did not detect/extract or authors omitting sleep continuity variables from the notes. Nevertheless, this automated strategy to identify and extract sleep data can serve as an effective tool in large health care systems to be used for research and evaluation to improve sleep medicine patient care and outcomes.

Support: This material is based upon work supported in part by the Department of Veteran Affairs, Veterans Health Administration, Office of Research and Development, and the Center for Innovations in Quality, Effectiveness and Safety (CIN 13-413). Dr. Nowakowski is also supported by a National Institutes of Health (NIH) Grant (R01NR018342).

1181

COMMUNITY-BASED ORGANIZATIONS SEEK SLEEP HEALTH EDUCATION

Fleshman, C.¹ Wolfson, A.² Ripple, C. H.³ Bonuck, K.⁴ Hale, L.⁵ Donskoy, I.⁶ Robbins, R.⁷ McGlinchey, E.⁸ Jean-Louis, G.⁹ Owens, J.¹⁰

¹Bradley Hospital Sleep Research Lab, Warren Alpert Medical School of Brown University, Providence, RI, ²Loyola University Maryland, Baltimore, MD, ³Pajama Program, New York, NY, ⁴University Center of Excellence at Montefiore Rose F. Kennedy Evaluation and Rehabilitation Center, Albert Einstein College of Medicine, The Bronx, NY, ⁵Stony Brook University, Stony Brook, NY, ⁶Advocate Children's Hospital, Chicago, IL, ⁷Brigham & Women's Hospital, Boston, MA, ⁸Fairleigh Dickinson University, Madison, NJ, ⁹NYU Langone Health, New York, NY, ¹⁰Boston Children's Hospital, Boston, MA. **Introduction:** Increasing attention to the importance of sleep among children raises questions about how to implement accessible, effective interventions. Part of answering those questions rests in determining interest in and demand for programming. Pajama Program (PJP), a 501(c)(3), works with nearly 4,000 communitybased organizations (CBOs) nationally that work with children exposed to adversity, including: foster care/child welfare; shelters; low-income schools, after-school, and early care and education programs; and social-service providers. Anticipating its launch of sleep health education programs, PJP and its Good Night Advisory Council of sleep experts designed a CBO needs assessment.

Methods: The survey was distributed electronically to staff at 3,911 CBOs; 1,635 organizations responded (42%).

Results: Across respondents, 65% work with children birth to 18 in settings that were non-residential (39%), residential (18%), or both (43%); most (91%) worked with participants for over one month. CBOs included child welfare/foster care (20.6% of respondents); transitional housing/shelter (20.5%); social services (15.6%); and early care and education (12.7%). Interest in sleep health education was high across all program types: 80 to 89% of programs within each type wanted information for staff and/or caregivers, specifically handouts (among 93% of programs), articles (88%), videos (85%), and workshops (70%). At least 90% of respondents who provided early care and education, parenting, and crisis services were interested in sleep health education for program staff. These program types also had high interest in sleep health education for caregivers, as did child welfare/foster care, school/after school, and shelters (all at least 90% of respondents).

Conclusion: The CBOs in this sample recognize sleep is an issue among the children they serve, but most did not have access to information on sleep health. These results establish the need for sleep health education and suggest preferred modalities. The project is a model for partnerships involving researchers, nonprofits, and community-based organizations.

Support: Funding for this project was provided by Pajama Program, a national 501(c)(3) non-profit.

1182

USING HOME DELIVERY OF HOME SLEEP TESTS TO IMPROVE THE DIAGNOSIS OF OBSTRUCTIVE SLEEP APNEA

Sterner, T. Yankowy, L. Vesnaver, D. Senft, R. WellSpan Health, York, PA.

Introduction: OSA is a highly prevalent and co-morbid condition across the US and the world. Recent data shows between 14% and 49% of middle-aged men have clinically significant OSA and al-though the data shows OSA is less prevalent in women, the consequences of this condition is as severe as in men. Further studies suggest 80% of more than 25 million cases of OSA in the US are undiagnosed. At our institution, several factors contributed to the under diagnosis of OSA: knowledge deficit, complicated order process, inconvenience, fear and cost. We developed a plan to increase the diagnosis of OSA by increasing home sleep testing by 50% within 6 months of initiating a home delivery model.

Methods: Our multifaceted, multidisciplinary and comprehensive plan included a contractual agreement with the manufacturer of our preferred device, WatchPAT, to directly ship the HSAT device to the patient, receive the device back after testing and upload the data for physician interpretation. Integration was established between middle-ware and the HSAT software for flow of information. A collaborative effort with our marketing department to develop a health risk assessment tool specific to sleep apnea,

targeted by health history, resulted in mailings to thousands of patients. A coordinated effort with our Call Center to explain delivery process and schedule testing was done. Paramount to our success was streamlining the ordering process for providers.

Results: 2,122 HSTs were done in the initial 6 months of using home delivery compared to the same 6 month time frame the previous year - an increase of 71%.

Conclusion: Using a broad, collaborative effort among several disciplines within our health care system, we found the access and use of home sleep tests dramatically improved. This was cost effective, saving .5 FTEs, provided a high degree of patient satisfaction and resulted in increased diagnoses. **Support:**

1183

BOARD-CERTIFIED SLEEP MEDICINE PHYSICIANS SEE A GREATER PROPORTION OF COMPLEX SLEEP PATIENTS THAN NON-SPECIALIST PROVIDERS

Jobe, S. L.¹ Albrecht, J. S.² Scharf, S. M.¹ Johnson, A. M.³ Parthasarathy, S.⁴ Wickwire, E. M.^{1,5}

¹Sleep Disorders Center, Division of Pulmonary and Critical Care Medicine, Department of Medicine, University of Maryland School of Medicine, Baltimore, MD, ²Department of Epidemiology and Public Health, University of Maryland School of Medicine, Baltimore, MD, ³Department of Pharmaceutical Health Services Research, University of Maryland School of Pharmacy, Baltimore, MD, ⁴University of Arizona Health Sciences Center for Sleep and Circadian Sciences, Tucson, AZ, ⁵Department of Psychiatry, University of Maryland School of Medicine, Baltimore, MD.

Introduction: Despite a growing literature regarding the impact of board-certification in sleep medicine, little is known about the complexity of patients seen by board-certified sleep medicine physicians (BCSMPs) relative to non-specialists. To address this gap, the purpose of the current study was to evaluate the differences in sleep complaints among Medicare beneficiaries seen by BCSMPs relative to individuals seen by non-specialists.

Methods: Our data source was a random 5% sample of Medicare administrative claims data from 2006-2013. Sleep disorders were operationalized using International Classification of Diseases, Ninth Revision, Clinical Modification codes. Descriptive analyses were performed to estimate the number of sleep disorder diagnoses patients received by provider status. BCSMPs were identified using a crossmatching procedure based on National Provider Identifier (NPI).

Results: A total of 57,209 Medicare beneficiaries received a sleep disorder diagnosis between 2006-2013. Of these, only 2.2% were seen by BCSMPs. Relative to beneficiaries seen by non-specialists, those seen by BCSMPs were more likely to be diagnosed with more than one sleep disorder (p<0.001). Specifically, 91.0% of individuals seen by non-specialists received only one sleep disorder diagnosis, whereas 75.9% of individuals seen by BCSMPs received only one sleep disorder diagnosis. Among beneficiaries seen by non-specialists, the most common sleep disorders were insomnia (48.2%; n=26,967), obstructive sleep apnea (OSA; 31.4%; n=17,554), and restless legs syndrome (8.7%; n=4,871). Among those seen by BCSMPs, the most common sleep disorders were OSA, (70.4%; n=901), sleep apnea with hypersomnia (16.5%; n=211), and insomnia (11.7%; n=150).

Conclusion: BCSMPs see more complex sleep patients than do non-specialists. These results suggest the possibility that more complex patients are referred for sleep specialty care. Further, these results demonstrate the value of board certification in sleep medicine in caring for complex sleep patients.

Support: This research was supported by an AASM Strategic Research Award from the AASM Foundation to the University of Maryland, Baltimore (PI: EMW).

1184

EVALUATION OF A NOVEL SLEEP MEDICINE EDUCATIONAL PROGRAM FOR THE PRIMARY CARE PROVIDER

Lang, R. Keenan, B. T. Kneeland-Szanto, E. Rosen, I. M. Perelman School of Medicine at the University of Pennsylvania, Philadelphia, PA.

Introduction: Inadequate exposure to and insufficient training in sleep medicine across the medical education continuum contributes to a lack of patient access to sleep care. We created a self-directed learning curriculum in sleep medicine aimed at practicing primary care providers.

Methods: In this pilot study, primary care providers, including physicians (PCP) and advanced practice nurses (APP-N), were invited to complete 3 application (app)-based core sleep educational modules in exchange for free continuing education and maintenance of certification credits. The modules were case-based and fully autonomous. Individuals had access to discussion boards moderated by a board-certified sleep physician and were given the option to complete two additional modules on advanced sleep topics. We assessed participants' opinions on the course and any effect on behavior, measured as change in the number of sleep-related orders. Results: Nineteen providers (12 PCP, 7 APP-N) completed the 3 required core modules. Five participants completed at least one additional module. A total of 94.4% reported they would recommend this curriculum to a colleague. Fifteen participants had prior experience with online courses; 93.3% noted that the sleep online modules provided a similar or better experience. Upon completion, 77.8% of learners anticipated this educational activity would contribute to either a great degree or completely to improvements in the health outcomes of their patients. All participants achieved the post-test score of 80% required to receive 1 CME/CNE credit per module completed. The number of sleep consultations or sleep study orders in the 4 months after course completion increased by 24.2%on average compared to the 4 months prior to the course (p=0.0157). Conclusion: Based on Kirkpatrick's model, this novel, app-based curriculum met levels 1-3 (positive reaction, knowledge transfer, and impact on behavior). Larger, longer-term studies are needed to assess the benefits of increasing knowledge in sleep medicine on patient care (Kirkpatrick Level 4).

Support: Funded by NIH NHLBI 5-R25-HL-120874-04

1185

DEVELOPING A CARE PATHWAY FOR INSOMNIA IN OLDER ADULTS AND ADULTS WITH DEMENTIA: RESULTS OF A CONSENSUS MEETING

Benca, R.¹ Ferziger, R.² Wickwire, E. M.³ Bertisch, S.⁴ Biddle, J.⁵ Boustani, M.⁶ Culpepper, L.⁷ Gooneratne, N.⁵ Lett, J.⁸ Manderscheid, R.⁹ Mehra, R.¹⁰ Reynolds, C.¹¹ Grandner, M.¹² ¹University of California, Irvine, Irvine, CA, ²Merck Research Laboratories, Upper Gwynedd, PA, ³University of Maryland, Baltimore, MD, ⁴Harvard Medical School, Boston, MA, ⁵University of Pennsylvania, Philadelphia, PA, ⁶Indiana University, Indianapolis, IN, ⁷Boston University, Boston, MA, ⁸Avar Consulting, Rockville, MD, ⁹National Association of County Behavioral Health and Developmental Disability Directors, Washington, DC, ¹⁰Cleveland Clinic, Cleveland, OH, ¹¹University of Pittsburgh, Pittsburgh, PA, ¹²University of Arizona, Tucson, AZ.

Introduction: Insomnia among older adults and dementia patients carries a high public health burden. Yet, treatment is inconsistent or absent. Standardized, programmatic carepaths can be implemented in clinics/systems/communities to address this after tailoring to local environments. To determine what elements should be included, a consensus meeting was convened, which included discussion, voting on components, and further consensusbuilding among diverse stakeholders.

Methods: Participants represented a wide range of stakeholders and specialties, including academic research, clinical care, industry, government, payors, sleep medicine, primary care, geriatrics, psychiatry, neurology, nursing, pharmacy, quality, and implementation science. 27 statements regarding key components of carepaths for insomnia in elderly and dementia populations were presented and discussed. These represented items addressing identification of patients, screening and assessment, deciding treatment modality and delivery, providing behavioral treatment, providing pharmacotherapy, addressing combined therapy, addressing comorbidities, and incorporating outcome evaluation. All N=20 participants voted individually whether they agreed or disagreed with each statement. Items were scored as 0=strongly agree, 1=agree, 2=disagree, and 3=strongly disagree. Mean scores were evaluated and responses were dichotomized to agree/disagree.

Results: Despite diversity among attendees, median rate of agreement was 95% (IQR=85-95%). Mean score was 0.69 (SD=0.31). 95%CIs were computed for each proportion and compared to the mean. The following elements were significantly different from the mean (p<0.05): medication decision trees (M=0.25), accounting for comorbidities (M=0.26), include outcome evaluation (M=0.30), utilization of EMR (M=0.40), incorporate caregiver (M=0.42), and differ across parts of the system (M=1.79).

Conclusion: Insomnia carepaths for older adults should address identification, screening and assessment, treatment decisions, treatment type and delivery, and evaluation. Organizations should consider these elements when designing carepaths for insomnia among older adults and dementia patients. Consensus-building should begin during the process of prioritizing care path components. **Support:** Merck Research Labs provided support

1186

DEVELOPING AND TESTING A WEB-BASED PROVIDER TRAINING FOR COGNITIVE BEHAVIORAL THERAPY OF INSOMNIA

Taylor, D.¹ Bunnell, B.² Calhoun, C.² Pruiksma, K.³ Dietch, J.⁴ Wardle-Pinkston, S.¹ Milanak, M.² Rheingold, A.² Simmons, R.² Peterson, A.³ Morin, C.⁵ Ruggiero, K.² Brim, W.⁶ Dolan, D.⁶ Wilkerson, A.²

¹University of Arizona, Tucson, AZ, ²Medical University of South Carolina, Charleston, SC, ³UTHSCSA, San Antonio, TX, ⁴University of North Texas, Denton, TX, ⁵Laval University, Quebec, QC, CANADA, ⁶Center for Deployment Psychology, Bethesda, MD.

Introduction: Chronic insomnia is a common, debilitating disorder and a risk factor for significant medical morbidity, mental health problems, and workplace difficulties. Cognitive behavioral therapy for insomnia (CBT-I) is the gold standard treatment for insomnia. However, few providers are trained in CBT-I, in part due to a bottleneck in training availability and the time and cost associated with current training platforms. To address this training deficit, our team developed and evaluated CBTI*web*.org, a web-based provider training course for CBT-I.

Methods: Feedback from alpha- and beta-testing of CBTI*web*.org was collected and used to optimize course content and functionality. Then, a comparison study was conducted in which licensed providers were randomized to complete either the online CBTI*web*. org course (n=21) or an in-person CBT-I training (n=23). During all phases of development, providers completed a Computer System Usability Questionnaire (CSUQ), investigator-developed website usability and content questionnaires, and pre/post-training competency assessments.

Results: Independent samples *t*-tests indicated significant improvements in CSUQ, and website usability and content questionnaires responses from alpha- to beta-testing (all ps < .05). Linear mixed-effects modeling revealed significant within-subject increases in knowledge acquisition (F(34.7) = 65.4, p < 0.001; baseline = 69% correct, post-training = 92% correct) when collapsed across in-person and web-based groups. The interaction group by time interaction was non-significant (F(34.7) = 1.7, p = 0.204), indicating similar gains in knowledge (i.e., equivalence) between the in-person and the CBTIweb.org training formats.

Conclusion: Alpha and beta testers of CBTI*web*.org reported high levels of satisfaction while also noting areas for improvement, which were used to update the site. Findings suggest the final CBTI*web*.org product successfully trained clinicians compared to an in-person workshop, given knowledge acquisition improvements. CBTI*web*.org is an efficient and effective training platform for clinicians to gain knowledge and competence in the most effective treatment for insomnia.

Support: W81XWH-17-1-0165

1187

WHAT TYPES OF ORGANIZATIONS PROVIDE SLEEP-FOCUSED WORKPLACE HEALTH PROMOTION PROGRAMS FOR THEIR EMPLOYEES? AN ANALYSIS OF THE 2017 CDC WORKPLACE HEALTH IN AMERICA SURVEY

Robbins, R.¹ Rosenberg, E.² Barger, L. K.¹ Weaver, M.¹ Quan, S. F.¹ Zeepvat, J.³ Czeisler, C. A.¹ Grandner, M. A.⁴ ¹Division of Sleep and Circadian Disorders, Boston, MA, ²Israeli Ministry of Health, Ramat Aviv, ISRAEL, ³Cornell University, Ithaca, NY, ⁴University of Arizona College of Medicine, Tucson, AZ.

Introduction: There has been a rise in workplace health promotion programs (WHPP)'s in the U.S., designed to improve a variety of employee health behaviors such as exercise and nutrition. Yet, relatively few focus on the third pillar of health: Sleep.

Methods: The CDC collected data from a nationallyrepresentative cohort of companies in 2017. Participants in this Workplace Health in America study completed online surveys reporting the type of WHPP offerings at their worksite and characteristics of their worksite, including occupational field (e.g., agriculture, management, wholesale/retail), workforce size (i.e., small: <100; moderate: 100-499; and large: 500+) and company type (e.g., non-profit, profit-private, profit-public, government). We identified factors associated with an increased likelihood of sleep-focused WHPP using logistic regression adjusted for company size and type. Analyses were weighted for nationally-representative estimates.

Results: Of the N=2,843 companies that provided information, N=261 (11.74%) reported having a sleep program. Worksites with large workforces (OR=4.8, p<0.0005), for-profit public companies (OR=9.0, p<0.0005), in wholesale/retail (OR=3.8, p<0.0005), and those with employer-subsidized full health insurance (OR=12.7, p<0.0005) were more likely to have a sleep-focused WHPP. Other predictors included more long-standing WHPP programs (6 years, OR=4.4, p<0.0005), the presence of employee health in the company's mission (OR=4.5, p<0.0005), leadership buy-in (OR=3.5, p=0.007), and an annual health promotion budget >\$50,000 (OR=11.3, p<0.0005).

Conclusion: In general, workplaces with higher budgets, more wellestablished health promotion programs, and a mission to promote workplace health are more likely to include a sleep program. Also, publicly-traded companies and government were more likely than private companies to have a sleep program. Future research may consider defining barriers among small business and non-profit organizations for implementing sleep-focused workplace health programs.

Support: T32HL007901

1188

TEST CHARACTERISTICS OF A MACHINE LEARNED ELECTRONIC MEDICAL RECORD EXTRACTABLE TOOL FOR OSA CASE IDENTIFICATION IN A COMMUNITY-BASED POPULATION

Patel, S. I.¹ Kukafka, D.² Antonescu, C.² Combs, D.¹ Lee-Iannotti, J.³ Quan, S. F.¹ Parthasarathy, S.¹

¹University of Arizona Health Sciences Center for Sleep and Circadian Sciences, Tucson, AZ, ²Banner Medical Group, Fort Collins, CO, ³Banner University Medical Center - Phoenix and University of Arizona, Phoenix, AZ.

Introduction: Obstructive sleep apnea (OSA) is a significantly underdiagnosed medical condition. A machine learning method known as SLIM (Supersparse Linear Integer Models) that can be extracted from the Electronic Health Record (EHR) has found to be superior to patient-reported sleep-related symptoms to diagnose OSA. Such an evaluation, however, was previously validated in a laboratory-based population. Our aim was to determine the test characteristics for the EHR-extractable SLIM tool in a community-based population.

Methods: Subjects who participated in the Sleep Heart Health Study (SHHS) were included in this analysis. Variable definitions of OSA were determined using an Apnea Hypopnea Index (AHI) threshold of 5 per hour, 15 per hour, or the presence of any comorbidity (hypertension, ischemic heart disease, stroke, mood disorders, impairment of cognition, or sleepiness) when the AHI was between 5 to 15 per hour. Variable hypopnea definitions based upon degree of oxygen desaturation and associated arousals were considered.

Results: In the SHHS dataset, the Receiver Operating Characteristics (ROC) for a SLIM score threshold of 9 for men and 5 for women was good when OSA was defined by AHI > 5 per hour (hypopneas with either $\geq 3\%$ oxygen desaturation or arousals). Specifically, the ROC was 0.72 (95% Confidence Intervals [CI] 0.70; 0.74) with a Positive Predictive Value [PPV] of 0.98 and Likelihood Ratio of a positive test (LR+) of 11.3. The LR+ (6.0) and PPV (0.92) were also good when an AHI of 5 per hour threshold was adopted with hypopneas scored using the minimum 3% oxygen desaturation alone. Similarly, the ROC was good 0.74 (95%CI 0.73; 0.76) with a Positive Predictive Value [PPV] of 0.98 and Likelihood

Ratio of a positive test (LR+) of 11.3. The LR+ (8.9) and PPV (0.81) were also good in the presence of comorbidities when AHI was 5 to 15 per hour using $\geq 4\%$ oxygen desaturation alone.

Conclusion: The EHR-extractable tool can be an actionable tool for case-identification of patients needing a referral for sleep study in a community-based population. Such an approach could facilitate an automated, rather than manual, OSA screening approach aimed at managing population health. **Support:** HL138377

Support: HL

1189

RECRUITING, TRAINING, AND IMPLEMENTING SLEEP HEALTH EDUCATORS IN COMMUNITY-BASED RESEARCH TO IMPROVE SLEEP HEALTH

Aird, C.¹ Seixas, A.¹ Moore, J.¹ Nunes, J.² Gyamfi, L.¹ Garcia, J.¹ Blanc, J.¹ Williams, N.¹ Zizi, F.¹ Jean-Louis, G.¹ ¹NYU Grossman School of Medicine, New York, NY, ²City College/ CUNY, New York, NY.

Introduction: Adherence to OSA assessment and treatment is low among racial/ethnic minorities, particularly among blacks. Navigating patients along the continuum of care from assessment to treatment adherence requires motivation, social support, and self-efficacy. Previous studies indicate that community health educators can provide motivation, social support, and skills to patients to better navigate the complex OSA care continuum. However, recruiting, training, and implementing sleep health educators in clinical or research settings is complex. For the current study, we describe how we recruit, train, and implement sleep health educators in research and clinical settings and assess what makes a sleep health educator successful.

Methods: We recruited and trained twenty-five self-identified black sleep health educators for a randomized clinical trial (R01MD007716) focused on increasing OSA assessment and treatment adherence among blacks. During recruitment, we assessed key personality attributes that translate to being an effective sleep health educator, via behavioral and personality surveys, focused groups, and process forms filled out by educators. Sleep health educators underwent an 8-week training program on sleep health and motivational interviewing. In order to be certified, sleep health educators had to pass a written and scenario-based assessment. During the implementation phase of the trial, we assessed how many interviews each health educator conducted and whether individual characteristics were related to how many interviews.

Results: Of the trained educators, 80% were female, ranging from 25 to 58 years old. They all completed at least high school. All educators rated the program highly and were very satisfied with dispensing tailored sleep health education. Educators who displayed the highest knowledge about sleep health, provided frequent emotional and strategic support, committed to helping their assigned participants, and who rated their rapport highly with their assigned participants were most effective in getting their participant to adhere to OSA assessment and treatment.

Conclusion: Sleep health educators can be vital to increasing OSA assessment and treatment adherence among blacks. In order to ensure success, sleep health educators must undergo a thorough recruitment, training, and implementation and dissemination process.

Support: K01HL135452, R01MD007716, R01HL142066, K01HL135452, and K07AG052685

1190

ENGAGING PATIENTS AND FAMILY MEMBERS TO UNDERSTAND WHAT MATTERS MOST LIVING WITH OBSTRUCTIVE SLEEP APNEA

Mou, J.¹ Silva, A.¹ Figetakis, K.² Ho, S. S.³ Williams, M.³ Mebust, K. A.³ Xia, Y.⁴ Xie, J.⁵ Wang, J.⁶ Chin, N.⁷ Vondran, R.⁸ Vondran, R.⁸

¹MultiCare Institute for Research & Innovation, MultiCare Health System, Tacoma, WA, ²Woodcreek Healthcare, Puyallup, WA, ³Neurophysiology & Sleep Program, MultiCare Health System, Tacoma, WA, ⁴University of British Columbia -Vancouver, Vancouver, BC, CANADA, ⁵University of North Carolina - Chapel Hill, Chapel Hill, NC, ⁶Charles Wright Academy, Tacoma, WA, ⁷University of Chicago, Chicago, IL, ⁸Sound Oxygen Service Inc, Puyallup, WA.

Introduction: As a common but modifiable chronic condition, obstructive sleep apnea (OSA) has been identified as the top secondary cause of many other diseases including cardiovascular diseases and type 2 diabetes. Diagnosing and managing OSA provides neurological, cardiovascular and metabolic benefits, however real-world studies indicate disconnections between evidence and outcomes. Using an engagement approach and qualitative design, this project aims to better understand care and research gaps in OSA in a community healthcare setting.

Methods: Methods: Patient and family representatives were identified and recruited through OSA support meetings hosted by MultiCare Sleep Medicine Centers, to form a board of 12, with three key patient advocates. Six meetings, each facilitated by one or two members of the board, were held to encourage focus group discussion and accommodate interactive conversations on the topic. Discussions were audio recorded and edited to exclude patients' identifiable information, then transcribed. Manual open coding was completed by two coders for each transcription to develop a codebook, followed by auto-coding and inductive content analysis using Nvivo 11.

Results: All enrolled patients had diagnosed moderate-to-severe OSA and were prescribed with continuous positive airway pressure (CPAP) therapy. Two participants were African American and one was multiethnic. Patients' age ranged from early 30s to 80s. Seven main themes were identified: OSA diagnostic issues; treatment experiences and options; comorbidities; patient community and support needs; long-term management challenges beyond "compliance"; knowledge of OSA, CPAP and care; and patient-driven research. The first few weeks after CPAP initiation appeared to be a critical time window that impacted patients' adaptation and use. Conclusion: Our study revealed barriers and facilitators in OSA diagnosis and treatment. Results showed highly prevalent chronic co-morbidities and the needs to care for patients in the comorbid scenario. It was highlighted that a paradigm of patient-centered care and research is lacking and warranted. Participants also called for better coordination between sleep medicine, primary care, other specialists, durable device suppliers and insurance. Key research efforts are expected to focus on the first 30-day post CPAP dispense to improve compliance.

Support: Patient Centered Outcomes Research Institute (PCORI) (Contract #: 7717241)

1191

DEVELOPMENT OF A CLINICALLY-VALIDATED QUESTIONNAIRE AND SCORING ALGORITHM DESIGNED TO IDENTIFY COMMON SLEEP PROBLEMS AMONG ADULTS

Galaska, B. Bakker, J. M. Sert Kuniyoshi, F. Bush, M. Salazar, J. Jasko, J. G. Friedman, A. L. White, D. P. Philips North America, Monroeville, PA.

Introduction: Although sleep is critical to maintaining health and quality of life, inadequate sleep duration and/or quality is common. It can be difficult to distinguish sleep problems that may be addressed through adjustments to lifestyle versus issues that may represent a more serious condition requiring medical intervention. SmartSleep Analyzer is a cloud-based questionnaire and scoring algorithm designed to categorize respondents according to likely sleep problems as follows: obstructive sleep apnea (OSA), snoring, trouble falling asleep or staying asleep, delayed sleep phase disorder (DSPD), shift work disorder (SWD), chronic sleep restriction (CSR), or no sleep problem. Primary, secondary, and tertiary categorizations are provided, where applicable. The objective of this study was to validate the questionnaire scoring algorithm categorization/s against a sleep physician assessment.

Methods: From 2,316 available records, 90 complete questionnaires were randomly selected for this analysis. The questionnaire scoring algorithm categorization was compared against the consensus assessment of three independent sleep physicians who each reviewed the answers to all questions before arriving at a diagnosis. Results: The questionnaire respondents (70% female) were aged 42.2±14.5 years, had a mean BMI of 32.0±7.7 kg/m², and selfreported sleep duration of 6.5±1.4 hours/night. The primary, secondary, or tertiary categorization of the questionnaire scoring algorithm matched the primary consensus categorization of the physicians 90.6% of the time (95% confidence interval (CI): 82.6 to 95.7). When OSA and snoring were grouped, agreement increased to 98.9% (95% CI: 94.0 to 100). In all analyses undertaken, the accuracy of questionnaire scoring algorithm against the physicians exceeded the accuracy of the physicians when compared to each other.

Conclusion: These results demonstrate that our questionnaire and scoring algorithm performs well in identifying sleep problems that may impact adult respondents, using physician-review as the comparison standard.

Support: Philips

1192

THE ACCURACY OF A NOVEL SLEEP RING DEVICE FOR ESTIMATING SLEEP ONSET WITH GOOD AND POOR SLEEPERS

Scott, H. Whitelaw, A. Canty, A. Lovato, N. Lack, L. Flinders University, Adelaide, AUSTRALIA.

Introduction: THIM is a new ring-like sleep device that, if found to accurately measure sleep onset, could be used for a variety of clinical purposes. These include administering a brief but effective treatment for insomnia called Intensive Sleep Retraining, facilitating the optimal 10-minute power nap, and administering Multiple Sleep Latency Tests (MSLTs) outside of the sleep laboratory. This study assessed the accuracy of THIM for measuring sleep onset latency compared to polysomnography (PSG).

Methods: Twenty healthy individuals aged 23.6 years (SD = 4.89) underwent overnight PSG recording whilst using THIM on two

nights in the sleep laboratory, one week apart. On each night, participants completed sleep onset trials for four hours whilst monitored via PSG. In these trials, participants attempted to fall asleep whilst responding to vibrations emitted from THIM. Once they failed to respond to two consecutive stimuli, THIM woke them with an intense vibration. Participants had a short break before attempting the next trial.

Results: On average, THIM overestimated sleep onset on the first night by 0.24 minutes (SD = 0.90). On the second night, THIM overestimated sleep onset by 0.82 minutes (SD = 1.31) and this discrepancy was not significantly different to that obtained on the first night, p = .08. The accuracy of THIM did not differ between good sleepers (Insomnia Severity Index (ISI) score < 7) or poor sleepers (ISI score 8-15), p = .98.

Conclusion: The findings suggest that THIM is accurate at estimating sleep onset latency for both good and poor sleepers. The next step is to test THIM outside of the laboratory environment. The goal is to develop an accurate yet practical device that can translate laboratory-based procedures to the home environment, to the benefit of patients and clinicians wanting to improve sleep. **Support:** The project was funded in-part by the manufacturers of THIM, Re-Time Pty. Ltd., with additional funding provided by Flinders University.

1193

ACCURACY OF A COMMERCIAL WEARABLE IN DETECTING SLEEP STAGES COMPARED TO POLYSOMNOGRAPHY IN ADULTS: CONSIDERING SLEEP CLASSIFICATION METHODS AND EFFECTS OF EVENING ALCOHOL CONSUMPTION

Menghini, L.¹ Alschuler, V.² Claudatos, S.² Goldstone, A.² Baker, F.² Cellini, N.¹ Colrain, I.² de Zambotti, M.²

¹Department of General Psychology, University of Padua, Padova, ITALY, ²Center for Health Sciences, SRI International, Menlo Park, CA.

Introduction: Commercial wearable devices have shown the capability of collecting and processing multisensor information (motion, cardiac activity), claiming to be able to measure sleep-wake patterns and differentiate sleep stages. While using these devices, users should be aware of their accuracy, sources of measurement error and contextual factors that may affect their performance. Here, we evaluated the agreement between Fitbit Charge 2^{TM} and PSG in adults, considering effects of two different sleep classification methods and pre-sleep alcohol consumption.

Methods: Laboratory-based synchronized recordings of device and PSG data were obtained from 14 healthy adults ($42.6\pm9.7y$; 6 women), who slept between one and three nights in the lab, for a total of 27 nights of data. On 10 of these nights, participants consumed alcohol (up to 4 standard drinks) in the 2 hours before bedtime. Device performance relative to PSG was evaluated using epoch-by-epoch and Bland-Altman analyses, with device data obtained from a data-management platform, Fitabase, via two

methods: one that accounts for short wakes (SW, awakenings that last less than 180s) and one that does not (not-SW).

Results: SW and not-SW methods were similar in scoring (96.76% agreement across epochs), although the SW method had better accuracy for differentiating "light", "deep", and REM sleep; but produced more false positives in wake detection. The device (SW-method) classified epochs of wake, "light" (N1+N2), "deep" (N3) and REM sleep with 56%, 77%, 46%, and 62% sensitivity, respectively. Bland-Altman analysis showed that the device

significantly underestimated "light" (~19min) and "deep" (~26min) sleep. Alcohol consumption enhanced PSG-device discrepancies, in particular for REM sleep (p=0.01).

Conclusion: Our results indicate promising accuracy in sleep-wake and sleep stage identification for this device, particularly when accounting for short wakes, as compared to PSG. Alcohol consumption, as well as other potential confounders that could affect measurement accuracy should be further investigated.

Support: This study was supported by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) grant R21-AA024841 (IMC and MdZ). The content is solely the responsibility of the authors and does not necessarily represent the official views the National Institutes of Health.

1194

AN OPEN-LABEL STUDY OF TREATING TRAUMATIC NIGHTMARES WITH AN INVESTIGATIONAL SMARTWATCH BASED SYSTEM

Stephan, J. T.¹ Davenport, N.² Evans-Lindquist, M. K.² Hiltner, R. K.² Karlin, D. R.³

¹NightWare, Minneapolis, MN, ²Minneapolis Veterans Affairs Health Care System, Minneapolis, MN, ³HealthMode, Inc., New York, NY.

Introduction: Nightmares are a common problem affecting 2-8% of the general population with the prevalence of comorbid nightmares in those with post-traumatic stress disorder (PTSD) being significantly higher at 72%. The negative sequelae of nightmares are myriad including impaired quality of life, sleep deprivation, insomnia, daytime sleepiness, fatigue, and suicidal ideation. This study investigated a novel approach for the treatment of nightmare disorder in military veterans.

Methods: All participants in this study were veterans receiving care at the Minneapolis Veterans Affairs Health Care System (VAHCS), diagnosed with PTSD, and had self-reported nightmares. At the baseline assessment, eligible participants were given a digital smartwatch preloaded with an application designed to arouse but not awaken the user out of the nightmare. Paired t-test analysis was used to compare the participants' baseline and follow-up responses.

Results: A significant decrease in participant Pittsburgh Sleep Quality Index (PSQI) scores (13.6 to 7.4, p < 0.001), PTSD Checklist for DSM-5 (PCL-5) scores (45.8 to 25.1, p < 0.0025) and Patient Health Questionnaire (PHQ-9) scores (12.2 to 6.1, p < 0.002) was observed over the sixty day trial. All 9 participants experienced a decrease in the Epworth Sleepiness Scale (ESS) upon completion of the trial, with an average decrease of -2.5 ± 1.4.

Conclusion: These results demonstrate that a novel digital therapeutic smartwatch application is effective in improving sleep quality, reducing the burden associated with PTSD symptoms, and lowering depressive symptoms in those with nightmare disorder within an open label study. These results have initiated further investigation into efficacy of digital therapeutic smartwatch applications in nightmare intervention and improved sleep quality. **Support:** NightWare, Inc.

1195

CLOSED-LOOP ELECTROENCEPHALOGRAM-BASED MODULATED PINK NOISE TO FACILITATE FALLING ASLEEP

Garcia-Molina, G. Kalyan, B. Aquino, A. Philips Sleep and Respiratory Care, Pittsburgh, PA. **Introduction:** During the wake to sleep transition, the EEG exhibits a reduction in the power in the beta (15-30 Hz) band and an increase in the power of the theta (4-8 Hz) band. In previous publications we reported that the log-ratio " ρ =10×log(β/θ)" quasi-monotonically decreases by an order of magnitude as sleep initiates.

Methods: We developed a closed-loop, real-time system that processes a single EEG signal (FPz-M2) to modulate the volume of (pink-noise) sound according to " $\rho = 10 \times \log(\beta/\theta)$ ". The volume was calibrated such that it progressively decreases as sleep initiates. The EEG was acquired using the Philips AliceTM PSG station connected to a laptop where the algorithm was implemented. The sound was played through a wearable headband connected to the laptop's audio-output. The algorithm processes 6-second EEG windows to estimate: 1) a signal quality index, 2) the average " ρ ", and 3) the sleep stage using a deep-learning stager. The volume changes with "p" according to a sigmoidal model. From the time where N2 or N3 sleep has been continuously detected for 3 minutes, the volume decays to zero in an exponential fashion. Seven subjects without any sleep disorder diagnosis (3F/4M; 33.6 ± 8.7 years old) participated in a home-based trial and recorded 5 sleep sessions. The first familiarization session was followed by randomized 2-session blocks: Block 1: closed-loop volume modulation (active), and Block 2 open-loop (sham) constant volume decrease.

Results: A 2.2-minute decrease (p=0.1) in average sleep latency was found in the active condition (11.6 \pm 5.0m) w.r.t. the sham condition (13.8 \pm 6.1m). A 5.2-minute decrease (p=0.08) in average N3 latency was found in the active condition (29.3 \pm 10.4m) w.r.t. the sham condition (34.5 \pm 13.6m). The log-ratio decreased significantly faster (p<0.05) and more monotonously in the active condition suggesting a faster sleep-deepening due to the sound modulation.

Conclusion: Closed-loop modulation of the volume of pink-noise based on the EEG's β/θ ratio may promote a faster sleep onset and a faster transition into deeper NREM sleep. The statistically trending results reported in this research grant further experimental validation with a larger number of subjects. **Support:** Philips Sleep and Respiratory Care

Support: Philips Sleep and Respiratory

1196

MACHINE LEARNING DERIVED-INTERPRETATIVE ALGORITHM BETTER DIFFERENTIATES SLEEP AND WAKE EPOCHS AND ESTIMATES SLEEP PARAMETERS FROM WRIST ACTIGRAPHY DATA

Haghayegh, S.¹ Khoshnevis, S.¹ Smolensky, M. H.¹ Diller, K. R.¹ Castriotta, R. J.²

¹The University of Texas at Austin, Austin, TX, ²University of Southern California, Los Angeles, CA.

Introduction: Several different interpretive algorithms (IAs) are available for scoring actigraphy-obtained body movement data for sleep and wake epochs. Although most have high sensitivity in detecting sleep epochs, they identify wake epochs poorly. We derived a machine learning (ML) based IA that improves differentiation of sleep and wake epoch to better estimate sleep parameters. **Methods:** Forty-one adults (18 females) 26.6 ± 12.0 years old underwent at-home single-night sleep assessment. Motionlogger[®] Micro Watch Actigraph recorded in zero crossing mode body movement per 30s epoch, with automated sleep scoring by single-channel electroencephalography (EEG) device (Zmachine[®] Insight+) as reference. The popular Cole-Kripke IA was applied to score body movement time series data of the following combination of current

1, preceding 4, and following 2 minute long epochs. Data of 21 subjects were utilized to train/derive the ML IA (logistic regression), and data of the other 20 subjects were used to test performance of it and the Cole-Kripke IA.

Results: In reference to the EEG, the Cole-Kripke actigraphy IA showed sensitivity of 0.98 ± 0.02 , specificity of 0.48 ± 0.19 , and kappa agreement of 0.53 ± 0.16 in detecting sleep epochs, while the ML-derived IA showed corresponding values of 0.90 ± 0.06 , 0.71 ± 0.14 , and 0.57 ± 0.11 . The Cole-Kripke IA, relative to EEG, method significantly (P<0.05) underestimated sleep onset latency (SOL) by 18.0 min and wake after sleep onset (WASO) by 35.1 min, and overestimated total sleep time (TST) by 53.1 min and sleep efficiency (SE) by 9.6%. The ML-derived IA, relative to EEG significantly underestimated SOL by 15.1 min, but comparably (P>0.05) estimated WASO, TST, and SE.

Conclusion: The ML-derived IA, in comparison to Cole-Kripke IA, when applied to sleep-time wrist actigraphy data significantly better differentiates wake from sleep epochs and better estimates sleep parameters.

Support: This work was supported by the Robert and Prudie Leibrock Professorship in Engineering at the University of Texas at Austin.

1197

UTILITY OF FITBIT CHARGE 2 FOR SLEEP MONITORING IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Kim, D. SHIN, W. Byun, J. KyungHee University Hospital at Gangdong, Seoul, KOREA, REPUBLIC OF.

Introduction: The wearable device may be useful in monitoring sleep. Many studies reported reliable data in detecting sleep-wake states and sleep stage proportion in healthy adults, However, only a few validation studies were performed evaluating sleep using the wearable devices in patients with obstructive sleep apnea(OSA), which showed insufficient accuracy. We aimed to evaluate the reliability of multi-sensory wristband (Fitbit Charge 2) in patients with OSA.

Methods: This was a preliminary analysis of a prospective singlecenter observational study. Consecutive patients underwent standard Polysomnography (PSG) for evaluation of OSA with Fitbit Charge 2. Sleep data from PSG and Fitbit charge 2 were compared using paired t-tests and Bland-Altman plots.

Results: A total of eighty-six patients were analyzed. Four of them had poor data quality, 18 of them did not show sleep stages. Compared with the PSG, Fitbit Charge 2 showed higher total sleep time (419.1 \pm 194.0 vs 269.8 \pm 22.6, p<0.001) and sleep efficiency (95.8 \pm 2.5 vs 84.6 \pm 7.1, p<0.001). Those with sleep stage data showed higher sleep efficacy (87.7 \pm 5.5 vs 82.37.5, p=0.024) and a lower proportion of N1 sleep (33.7 \pm 19.9 vs 65.3 \pm 38.8, p=0.01).

Conclusion: Fitbit Charge 2 showed limited utility in monitoring sleep in patients with obstructive sleep apnea. **Support:** none

1198

EFFICACY OF DIRECT THERMOELECTRIC FOREHEAD COOLING FOR TREATING INSOMNIA SYMPTOMS

Schirm, J.¹ Bellamy, B.¹ Nofzinger, E.^{1,2}

¹Ebb Therapeutics, Pittsburgh, PA, ²University of Pittsburgh, Pittsburgh, PA.

Introduction: Prior studies have shown beneficial effects of forehead cooling in insomnia patients using a device that circulates cooled fluids through a forehead pad. The current study aimed to determine if a device that cooled via direct thermoelectric contact to the forehead via a Peltier cooler would have similar effects in individuals with insomnia symptoms. Such a technology may allow for form factors that may have advantages for some individuals.

Methods: An intelligent, portable and battery-operated forehead cooling system using thermoelectric coolers (TECs) with a user selectable temperature range between 14C and 18C was used in the study. Individuals with insomnia symptoms (N=30, 25 female) were recruited and studied at 2 geographic locations. Each participant received pre- and post- treatment insomnia severity as well as daily sleep diary assessments over 1 week of baseline and 4 weeks of treatment.

Results: Participants' insomnia symptoms improved over baseline in insomnia severity index (M + SD = 19.7 + 3.8 pre- vs 9.4 + 5.3 post-treatment, t = -9.3, p<0.00001), in sleep latency (M + SD = 43.0 + 40.8 minutes pre- vs 20.7 + 22.7 minutes posttreatment, t = 6.8, p<0.00001), in minutes awake after sleep onset (M + SD = 63.0 + 59.2 minutes pre- vs 24.5 + 34.5 minutes posttreatment, t = 8.0, p<0.00001) and in sleep quality (0-10 scale with 10 = best, M + SD = 4.1 + 1.9 pre- vs 6.8 + 2.2 post-treatment, t = -13.4, p<0.00001).

Conclusion: Forehead cooling via direct thermoelectric contact to the forehead via a Peltier cooler had beneficial effects on subjective insomnia symptoms. These promising preliminary data suggest the need for further large scale randomized controlled trials to establish the efficacy of forehead-cooling using direct thermoelectric contact to the forehead via a Peltier cooler on insomnia symptoms. **Support:** Ebb Therapeutics, Pittsburgh, PA 15222

1199

THE ACCURACY OF A NEW SLEEP RING DEVICE FOR TRACKING SLEEP AND WAKEFULNESS OVERNIGHT USING ACTIGRAPHY

Scott, H. Lovato, N. Lack, L.

Flinders University, Adelaide, AUSTRALIA.

Introduction: THIM is a new consumer ring-like device that can passively monitor sleep overnight using actigraphy. This project aimed to develop the THIM sleep tracking algorithm (Study 1), and test its accuracy against polysomnography (PSG) with another independent sample of good and poor sleepers (Study 2).

Methods: *Study* 1: 25 healthy individuals (15 females) aged 25.38 years (SD = 6.39) slept overnight in the sleep laboratory with THIM, the Philips Spectrum, the Fitbit Flex, and PSG recording simultaneously. The THIM sleep tracking algorithm was developed by optimising sensitivity and specificity with PSG. *Study* 2: An additional 20 individuals (14 females) aged 23.22 years (SD = 5.02) slept overnight in the sleep laboratory with the same devices as in Study 1.

Results: *Study 1:* THIM showed high agreement with PSG for estimating sleep (sensitivity = .91) and reasonably high agreement for wakefulness (specificity = .59). There were no significant differences between PSG and THIM for total sleep time, t(24) = 0.76, p = .46, or sleep efficiency, t(24) = 0.56, p = .58. *Study 2:* THIM showed high agreement with PSG for estimating sleep (sensitivity = .89) and wakefulness (specificity = .59). Compared to PSG, THIM significantly underestimated total sleep time, t(19) = 2.10, p = .049, and sleep efficiency, t(19) = 2.20, p = .04, by an average of 21.35 minutes (*SD* = 45.52) and 4.44% (*SD* = 9.04), respectively.

Conclusion: Together, these studies suggest that THIM is reasonably accurate for monitoring sleep overnight in healthy individuals. Slight modifications to the algorithm and additional sensors could be added to THIM to improve its accuracy. Future research will examine the accuracy of THIM with larger sample sizes and particularly for people with insomnia, with the goal being to incorporate sleep tracking into a mobile-based treatment program for insomnia.

Support: The project was funded in-part by the manufacturers of THIM, Re-Time Pty. Ltd. Additional funding was provided by Flinders University.

1200

SEASONAL VARIATION IN RHR MEASURED BY A WEARABLE RING

Koskimaki, H.¹ Kinnunen, H.¹ Ronka, S.² Smarr, B.³ ¹Oura Health Ltd, Oulu, FINLAND, ²Bitfactor, Oulu, FINLAND, ³Oura Health Ltd, San Fransisco, CA.

Introduction: Resting heart rate (RHR) associates with cardiovascular fitness, acute and chronic health status, and mental stress. Nightly sleep provides an excellent measurement time because ambient conditions are constant and confounding factors are controlled. Relatively low RHR is seen as a mark of better health, performance, and recovery levels. Over the past few years, development of wearable devices has made it possible to follow the course of individual RHR as long-term time series, which enables observation of how behavioural, societal and seasonal factors affect RHR at population scale.

Methods: In this study, mean individual nightly RHR of each calendar day was studied from all Oura ring users starting from July 2016 (n=1415) and ending at March 2019 (n=57.278). The subjects were 36 percent female and 64 percent male, mostly in working age: 20-30-year (15%), 30-50 years (60%), and 50-60-years (16%). Majority of the ring users live in the Northern Hemisphere, and minority (n = 1.500) in the Southern Hemisphere.

Results: With users from Northern Hemisphere, the yearly RHR peaks in December and lowers between April and August. The magnitude of seasonal variation was observed to be roughly 2 bpm: In 2018, March averages 1.6 bpm higher than August, and December averages 2.1 bpm higher than August. Looking at subjects from New Zealand and Australia, exactly opposite phase was observed, so that both hemispheres followed the length of the day. Apparently, RHR yearly fluctuations are based on biological factors like Sun light and ambient temperature. Both hemispheres also demonstrated weekly peaks at weekends (+1.6 bpm), and a most district peak at each New Year night. **Conclusion:** The long-term RHR was shown to be affected by biological, societal, and lifestyle factors. We find that long-term wearable data from the Ring can reveal health related physiological effects across large populations, including seasonality.

Support: This work was supported by Oura Health Ltd.

1201

PERFORMANCE EVALUATION OF A NOVEL CONTACTLESS BREATHING MONITOR AND MACHINE LEARNING ALGORITHM FOR SLEEP STAGE CLASSIFICATION IN A HEALTHY POPULATION

Lauteslager, T.¹ Kampakis, S.¹ Williams, A. J.² Maslik, M.¹ Siddiqui, F.¹

¹Circadia Technologies Ltd, London, UNITED KINGDOM, ²Sleep Disorders Centre, Guy's and St Thomas' NHS Foundation Trust, London, UNITED KINGDOM. **Introduction:** Although polysomnography (PSG) remains the gold standard for sleep assessment in a lab setting, non-EEG signals such as respiration and motion are directly affected by sleep stages and can be used for sleep stage prediction. Importantly, these signals can be obtained in a low-cost and unobtrusive manner, allowing for large scale and longitudinal data collection in a home environment. The Circadia C100 System (FDA 510(k) clearance expected Q1 2020) is a novel 'nearable' device that uses radar for contactless monitoring of respiration and motion. The current study aims to validate the performance of the associated sleep analysis algorithm.

Methods: A total of 41 nights of sleep data were recorded from 33 healthy participants using the device, alongside PSG. Data were recorded both in a sleep lab and home environment. PSG data were scored by RPSGT-certified technicians. Respiration and movement features were extracted, and machine learning algorithms were developed to perform sleep stage classification and predict sleep metrics. Algorithms were trained and validated on PSG data using cross-validation.

Results: An epoch-by-epoch true positive rate of 56.2%, 79.4%, 55.5% and 72.6% was found for 'Wake', 'REM', 'Light' and 'Deep' respectively. No statistical differences in performance were found between home-recorded and lab-recorded contactless data. Mean absolute error of total sleep time (TST), wake after sleep onset (WASO), and sleep efficiency (SE) was 13.2 minutes, 11.3 minutes and 3%, respectively. The contactless monitor was found to outperform both medical grade and clinical grade actigraphy based devices: The Philips Actiwatch Spectrum Plus and the Fitbit Alta HR.

Conclusion: Current results are encouraging and suggest that the contactless monitor could be used for long-term sleep assessment and continuous evaluation of sleep therapy outcomes. Further clinical validation work is ongoing in subjects diagnosed with sleep disorders such as obstructive sleep apnea. **Support:** -

1202

VALIDATION OF A NOVEL WEARABLE HOME SLEEP TESTING DEVICE FOR ASSESSMENT OF OBSTRUCTIVE SLEEP APNEA

YEH, E.¹ WONG, E.¹ STROHL, K.¹ GU, W.² TSAI, C.² LEUNG, L.² YAR, W.¹ CHIANG, A.¹ ¹University Hospitals, CLEVELAND, OH, ²Belun Technology Company Limited, Hong Kong, HONG KONG.

Introduction: There is a substantial need for an accurate and easyto-use tool for obstructive sleep apnea (OSA) assessment. Belun Ring Platform (BRP), a novel photoplethysmography (PPG)-based home sleep apnea testing system with a proprietary deep learning algorithm, has been shown to have good sensitivity and specificity in predicting OSA in subjects without significant comorbidities and medications known to affect heart rate (HR). In this study, we further tested its performance in subjects referred for in-lab polysomnography (PSG) assessment of sleep disorders without excluding those with non-arrhythmia comorbidities or the subjects on HR-affecting medications.

Methods: PSG was recorded simultaneously with the Ring in the sleep lab and the studies were manually scored by certified sleep technicians according to the AASM Scoring manual version 2.4. Exclusion criteria include age <18, unstable cardiopulmonary status, recent hospitalization within 30 days, significant arrhythmias, baseline HR <50 or >100, home oxygen use, pacemaker/

defibrillator, post-cardiac transplantation or Left ventricular assist device.

Results: A cohort of 78 individuals (26 males and 52 females, age 50.5) were studied with 26 taking HR-affecting medications. Of these, 35 (45%) had AHI < 5; 14 (18%) had AHI 5-15; 15 (19%) had AHI 15-30; 14 (18%) had AHI > 30. The Ring-REI correlated well with the PSG-AHI (r =0.83, P <0.001). The accuracy, sensitivity, specificity in categorizing AHI >15 were 0.808, 0.931, and 0.735 respectively. The positive predictive value, negative predictive value, positive likelihood ratio, and negative likelihood ratio were 0.675, 0.947, 3.509, and 0.094 respectively. The use of HR-affecting medications did not significantly affect the sensitivity and specificity of BRP in predicting OSA (P =0.16 and 0.44 respectively).

Conclusion: BRP is promising as a reasonable tool for OSA assessment and can potentially be incorporated into a broad spectrum of clinical practices for identification of patients with OSA.

Support: This study is supported by a Grant from Belun Technology Company Limited.

1203

WORTH THE WEIGHT: WEIGHTED BLANKET IMPROVES SLEEP AND INCREASES RELAXATION

Danoff-Burg, S. Rus, H. M. Cruz Martir, L. Raymann, R. J. SleepScore Labs, Carlsbad, CA.

Introduction: Weighted blankets are designed to create deep touch pressure stimulation, to simulate the feeling of being held or hugged. The deep pressure provided by a weighted blanket, along with consistent sensory input from its weight, is thought to work by lowering stress and reducing physiological arousal, thereby improving sleep. We tested whether a weighted blanket would improve sleep.

Methods: 30 participants with objectively measured sleep onset and sleep maintenance issues participated in a 6-week field study, using a non-counterbalanced pre-post intervention design. Intervention consisted of the use of a weighted blanket every night at home. The blanket was filled with microbeads and weighed approximately 10 percent of each participant's body weight. Sleep was measured objectively using ResMed S+ every night and by daily self-report. Paired t-tests and multilevel regression were used to test for statistical significance.

Results: Objective sleep data from the 28 participants who completed all study requirements showed 7% improvement of Wake After Sleep Onset, 2% decrease in Light Sleep, and slight improvement in Sleep Efficiency (1.5%) and Sleep Maintenance (1.4%) during intervention (all ps < 0.05). Additionally, participants felt they fell asleep faster (13% faster), experienced better sleep quality (14% better), felt more rested in the morning (17% more rested), and felt they slept better through the night without waking up (36% improvement). They also reported feeling 13% less stressed at bedtime and 17% more relaxed while trying to fall asleep.

Conclusion: Using a weighted blanket reduces self-reported feelings of stress, enhances feelings of relaxation, and can improve sleep and reduce time awake at night in people with sleep onset and sleep maintenance issues.

Support: Gravity Blanket

1204

ANALYZING USER JOURNEY DATA IN DIGITAL HEALTH: PREDICTING DROPOUT FROM A DIGITAL CBT-I INTERVENTION

Bremer, V.¹ Chow, P.² Funk, B.¹ Thorndike, F.³ Ritterband, L.²

¹Leuphana University, Lunenberg, GERMANY, ²University of Virginia, Charlottesville, VA, ³Pear Therapeutics, Boston, MA.

Introduction: Intervention dropout is an important factor for the evaluation and implementation of digital therapeutics, including in insomnia. Large amounts of individualized data (logins, questionnaires, EMA data) in these interventions can combine to create user journeys - the data generated by the path an individual takes to navigate the digital therapeutic. User journeys can provide insight about how likely users are to drop out of an intervention on an individual level and lead to increased prediction performance. Thus, the goal of this study is to provide a step-by-step guide for the analysis of user journeys and utilize this guide to predict intervention dropout, illustrated with an example from a data in a RCT of digital therapeutic for chronic insomnia, for which outcomes have previously been published.

Methods: Analysis of user journeys includes data transformation, feature engineering, and statistical model analysis, using machine learning techniques. A framework is established to leverage user journeys to predict various behaviors. For this study, the framework was applied to predict dropouts of 151 participants from a fully automated web-based program (SHUTi) that delivered cognitive behavioral therapy for insomnia. For this task, support vector machines, logistic regression with regularization, and boosted decision trees were applied at different points in 9-week intervention. These techniques were evaluated based on their predictive performance.

Results: After model evaluation, a decision tree ensemble achieved AUC values ranging between 0.6-0.9 based on application of machine earning techniques. Various handcrafted and theory-driven features (e.g., time to complete certain intervention steps, time to get out of bed after arising, and days since last system interaction contributed to prediction performance.

Conclusion: Results indicate that utilizing a user journey framework and analysis can predict intervention dropout. Further, handcrafted theory-driven features can increase prediction performance. This prediction of dropout could lead to an enhanced clinical decision-making in digital therapeutics.

Support: The original study evaluating the efficacy of this intervention has been reported elsewhere and was funded by grant R01 MH86758 from the National Institute of Mental Health.

1205

ACTIVITY TRACKERS AS A TOOL IN SLEEP RESEARCH: DETERMINING DISCREPANCIES IN TRACKERS VS. PSG

Pollet, E. P.^{1,2} Pollet, D. P.² Long, B.¹ Qutub, A. A.¹

¹University of Texas at San Antonio, San Antonio, TX, ²Academy Diagnostics Sleep and EEG Center, San Antonio, TX.

Introduction: Fitness-based wearables and other emerging sensor technologies have the potential to track sleep across large populations longitudinally in at-home environments. To understand how these devices can inform research studies, limitations of available trackers need to be compared to traditional polysomnography (PSG). Here we assessed discrepancies in sleep staging in activity trackers vs. PSG in subjects with various sleep disorders.

Methods: Twelve subjects (age 41-78, 7f, 5m) wore a Fitbit Charge 3 while undergoing a scheduled sleep study. Six subjects had been previously diagnosed with a sleep disorder (5 OSA, 1 CSA). 4 subjects used CPAP throughout the night, 2 had a split night (CPAP 2nd half of the night), and 6 had a PSG only. Activity tracker staging was compared to 2 RPSGTs staging.

Results: Of the 12 subjects, eight subjects' sleep was detected in the activity tracker, and compared across sleep stages to the PSG (7 female, 1 male, ages 41-78, AHI 0.3-87, RDI 0.5-94.4, sleep efficiency 74%+/-18, 4 PSG, 1 split, 3 CPAP). The activity tracker matched either tech 52% (+/- 13). The average difference in score tech and activity tracker staging for sleep onset (SO) was 16 +/- 15 minutes and wake after sleep onset was 43.5 +/- 44 minutes. Sensitivity, specificity, and balanced accuracy were found for each sleep stage. Respectively, **Wake**: 0.45+/-0.27, 0.97+/-0.03, 0.71+/-0.12, **REM**: 0.41+/-0.30, 0.90+/-0.06, 0.60+/-0.28, **Light**: 0.71+/-0.09, 0.58+/-0.19, 0.65+/-0.10, **Deep**: 0.63+/-0.52, 0.88+/-0.05, 0.59+/-0.49.

Conclusion: From this study of 12 subjects seen at a sleep clinic for suspected sleep disorders, activity trackers performed best in wake, REM and deep sleep specificity (>=88%), while they lacked sensitivity to REM and wake (<=45%) stages. The tracker did not detect sleep in 4 subjects who had elevated AHI or low sleep efficiency. Further analysis can identify whether discrepancies between the Fitbit and PSG can be predicted by distinct patterns in sleep staging and/or identify subject exclusion criteria for activity tracking studies.

Support: This project in on-going with the support of Academy Diagnostics Sleep and EEG Center and staff.

1206

AUTOMATED SLEEP APNEA ASSESSMENT BASED ON MACHINE LEARNING AND WEARABLE TECHNOLOGY Chin. H^{\perp} Log. P^{2} Ku. P^{\perp} Lin. X^{\perp}

Chiu, H.¹ Lee, P.² Ku, B.¹ Liu, Y.¹

¹Mediatek Inc., Hsinchu city, TAIWAN, ²Centre of Sleep Disorder, National Taiwan University Hospital, Taipei City, TAIWAN.

Introduction: Obstructive sleep apnea (OSA) is a condition characterized by repeated episodes of partial or complete obstruction of the respiratory passages during the sleep. Traditional polysomnography (PSG) for OSA estimation is bulky and time-consuming for daily use. Therefore, this study aims to develop a novel photoplethysmography (PPG) and accelerometer based smart watch for OSA detection, in which a high-performance and low-complexity automated OSA detection was embedded for long-term in-home measurement.

Methods: The developed watch measured PPG signals from wrist radial artery and body motion from accelerometer as well. 121 patients (92 males, 29 females) were recruited from the normal community and Center of Sleep, National Taiwan University Hospital, Taiwan in this study. All OSA scoring were analyzed by three registered PSG technologists. The AHI of the cohort was 10.1 ± 18.3 (0 to 82.7). An automated OSA detection algorithm was designed based on machine-learning (ML) technique, in which was iteratively updated according to each 30-second epoch of the collected data. Subsequently, obstructive and hypopnea events were detected according to the OSA detection algorithm.

Results: To better valid the effectiveness, this study focused on the estimation performance of the subjects with AHI>15. Based on hold-out validation, the average sensitivity and precision in the AHI>15 cohort were 77.2% and 58.6%, respectively, with a Cohen's kappa of 0.46. The interclass correlation between the watch and technologists was 0.81 (95%CI: 0.61-0.91). The result showed that the proposed automated OSA detection could achieve consistent result with technologist during standard sleep testing.

Conclusion: This study developed a wrist-based watch based on ML technique to assess OSA severity. We compared the performance with clinical technologists for the OSA detection. Further, the

sensitivity and precision were generally acceptable while subjects were with AHI>15. The proposed wrist-based watch could provide reliable performance for OSA estimation, and may be of a light for future in-home sleep studies.

Support: This study was supported by Mediatek Inc.

1207

KEEP IT SIMPLE: WIRELESS PATCHES FOR HOME SLEEP DIAGNOSTICS

Zandieh, S. O.^{1,2} Reuveny, A.³ Pearson, S.⁴ Mordechai, A.³ Wang, C.³ Aesmani, D.³ Backeris, P.³ Ancoli-Israel, S.⁵ ¹Saint Barnabas Medical Center, West Orange, NJ, ²Weil Cornell Medical College, New York, NY, ³Cornell Tech, New York, NY, ⁴Valley Medical Center, Ridgewood, NJ, ⁵University of California, La Jolla, CA.

Introduction: Despite growing use of home sleep testing for the diagnosis of obstructive sleep apnea (OSA), there are significant barriers including limited availability, high cost, and complex wires making it difficult for patients to use on their own. The objective of this study was to evaluate a new flexible, thin, and wireless adhesive patch (proprietor sensors, TatchSleep Pro [TSP]) for the detection of OSA and compare the validity to overnight polysomnography (PSG). The TSP transmits data wirelessly to a smartphone app which in turn uploads the data to a cloud server. Data is presented to a sleep professional via a dedicated interface to score and analyze the results.

Methods: Patients (n=9; mean age=47 (SD=10); mean BMI=33 (SD=6.6); 4 males) undergoing a PSG evaluation for OSA also wore 2 TSP patches applied on the chest and abdomen. The TSP patches detected respiratory effort, derived airflow, derived pressure, body position and movement. Two sets of respiratory data (with common SpO2 and heartrate as a reference from PSG) were created, one from PSG and one from the TSP. The data were then scored by a certified sleep technician, blinded to the conditions. Linear regression analyses were used to compare the two derived apnea-hypopnea indices (AHI). In the morning, all participants were asked about their experience using the TSP. The study was approved by the Western IRB.

Results: There was a significant correlation for AHI between the TSP and PSG (R2=0.94; p<0.01). All participants found the TSP comfortable to wear and indicated that removing the patches was 'easy' or 'very easy,'

Conclusion: These preliminary results suggest the wireless TSP may be an effective, cost-efficient way to evaluate OSA. Despite small sample, results show promise as a new innovative product for home sleep testing.

Support: Supported by Tatch Inc.

1208

SLEEP STAGE PREDICTION AND SLEEP DISORDERED BREATHING DETECTION USING RAW ACTIGRAPHY AND PHOTOPLETHYSMOGRAPHY FROM WEARABLE CONSUMER DEVICE

Olsen, M.¹ Sorensen, H.² Jennum, P.³ Mignot, E.⁴

¹Technical University of Denmark, Palo Alto, CA, ²Technical University of Denmark, Lyngby, DENMARK, ³Danish Center for Sleep Medicine, Glostrup, DENMARK, ⁴Stanford University, Palo Alto, CA.

Introduction: Wearable, multisensory consumer devices that estimate sleep are prevalent and hold great potential. Most validated actigraphic prediction studies of sleep stages (SS) have only used low resolution (30 sec) data and the Cole-Kripke algorithm. Other algorithms are often proprietary and not accessible or validated. We present an automatic, data-driven deep learning algorithm that process raw actigraphy (ACC) and photoplethysmography (PPG) using a low-cost consumer device at high (25Hz) and low resolution to predict SS and to detect sleep disordered breathing (SDB) events.

Methods: Our automatic, data-driven algorithm is a deep neural network trained and evaluated to predict SS and SDB events on 236 recordings of ACC data from a wrist-worn accelerometer and PPG data from the overlapping PSG. The network was tested on raw ACC and PPG data, which was collected at 25 Hz using the HUAMI Arc2 wristband from 39 participants that underwent a nocturnal polysomnography (PSG).

Results: Overall accuracy (Acc), recall (Re), specificity (Sp), and kappa (κ) per subject on the test dataset the prediction of wake, NREM, REM was Acc=76.6%, Re=72.4%, Sp=78.0%, kappa=0.42. On average, we found a 7% higher performance using the raw sensor data as input instead of processed, low resolution inputs. PPG was especially useful for REM detection. The network assigned 55.6% of patients to the correct SDB severity group when using an apnea-hypopnea index above 15.

Conclusion: Current results show that SS prediction is significantly improved when using the raw sensor data; it indicates that the system holds promise as a potential pervasive monitoring device for patients with chronic sleep disorders. In contrast the system did not show potential as a sleep apnea screening tool. Additional studies are ongoing to examine the effects of pathology such as sleep apnea and periodic leg movement on SS prediction.

Support: Technical University of Denmark; University of Copenhagen, Copenhagen Center for Health Technology, Klarman Family Foundation.

1209

EFFECT OF WEARABLES ON SLEEP IN HEALTHY INDIVIDUALS: A RANDOMIZED CROSS-OVER TRIAL AND VALIDATION STUDY

Berryhill, S.¹ Morton, C. J.¹ Dean, A.¹ Berryhill, A.¹ Provencio-Dean, N.¹ Patel, S. I.¹ Estep, L.¹ Combs, D.² Mashaqi, S.¹ Gerald, L. B.³ Krishnan, J.⁴ Parthasarathy, S.⁵

¹UAHS Center for Sleep & Circadian Sciences; Division of Pulmonary, Allergy, Critical Care & Sleep Medicine; University of Arizona, University of Arizona, AZ, ²Department of Pediatrics, University of Arizona, Tucson, AZ, ³Asthma and Airways Disease Research Center, Tucson, AZ, ⁴Breathe Chicago Center and Division of Pulmonary, Critical Care, Sleep, & Allergy, University of Illinois, Chicago, Illinois, Chicago, IL, ⁵University of Arizona Health Sciences Center for Sleep and Circadian Sciences, University of Arizona, AZ.

Introduction: To determine whether a wearable sleep-tracker improves perceived sleep quality in healthy subjects. To test whether wearables reliably measure sleep quantity and quality compared to polysomnography.

Methods: A single-center randomized cross-over trial of community-based participants without medical conditions or sleep disorders. Wearable device (WHOOP, Inc.) that provided feedback regarding sleep information to the participant for 1-week and maintaining sleep logs versus 1-week of maintaining sleep logs alone. Self-reported daily sleep behaviors were documented in sleep logs. Polysomnography was performed on one night when wearing

the wearable. PROMIS Sleep disturbance sleep scale was measured at baseline, 7, and 14 days of study participation.

Results: In 32 participants (21 women; 23.8 ± 5 years), wearables improved nighttime sleep quality (PROMIS sleep disturbance; B= -1.69; 95% Confidence Interval -3.11, -0.27; P=0.021) after adjusting for age, sex, baseline, and order effect. There was a small increase in self-reported daytime naps when wearing the device (B = 3.2; SE 1.4; P=0.023) but total daily sleep remained unchanged (P=0.43). The wearable had low bias (2.5 minutes) and low precision (5.6 minutes) errors for measuring sleep duration and measured dream sleep and slow wave sleep accurately (Intraclass coefficient 0.74 ± 0.28 and 0.85 ± 0.15 , respectively). Bias and precision error for heart rate (bias -0.17%; precision 1.5%) and respiratory rate (bias 1.8%' precision 6.7%) were very low when compared to that measured by electrocardiogram and inductance plethysmography during polysomnography.

Conclusion: In healthy people, wearables can improve sleep quality and accurately measure sleep and cardiorespiratory variables. **Support:** WHOOP Inc.

1210

DEVELOPMENT OF AN AUDITORY NEUROFEEDBACK DURING SLEEP ONSET PROCESS

Douch, M.¹ Soubrier, M.¹ Pinaud, C.¹ Harris, M.² Thorey, V.¹ ¹Dreem Algorithms Team, Paris, FRANCE, ²Dreem Science Team, New York, NY.

Introduction: Biofeedback is proposed as an alternative method to help patients with insomnia reducing their anxiety. Some studies have shown that auditory neurofeedback can be effective at reducing sleep-onset latency. However, the AASM sleep stage classification only describes the sleep-onset as a binary state (i.e. wake or N1) which makes it not adapted for neurofeedback. We introduced a simple 4-stages classification for sleep-onset, on 10 seconds EEG epoch. The aim of this study was to develop an automatic method to detect these stages, and an online algorithm embedded in the Dreem headband (DH) that adapted the auditory feedback based on the current stage.

Methods: Fourteen subjects underwent an overnight PSG monitoring, from which the first sleep-onset period was extracted. We defined the simple 4-stages classification for sleep-onset on 10 seconds EEG epoch as following: SO1) > 75% of the epoch covered by alpha frequencies SO2) between 25% and 50% of the window covered with alpha frequencies, SO3) Alpha frequencies covered less than 25% and theta frequencies covered less than 30% of the epoch, and SO4) Theta frequency covered more than 30% of the epoch. For the manual scoring, 4 sleep scorers have been given the instructions and a Q&A session after scoring the first two records. For the algorithm, a sound triggering algorithm was linked to a neural network trained on the scored data, to dynamically adapt the sound to the sleep-onset stage.

Results: The scorers reached an average agreement of 68 + 15% over all the records. The neural network reached an accuracy of 68%. Per state the accuracy was: $71 \pm 32\%$ (S1), $52 \pm 22\%$ (S2), $54 \pm 23\%$ (S3), $79 \pm 21\%$ (S4). The automatic neurofeedback was able to adapt sound stimulations in real-time based on stages and was well perceived among first testers.

Conclusion: The results of this preliminary work show that we can reach a higher agreement by reducing the epoch duration and use this classification to produce automatic biofeedback during the sleep onset period. Further studies using a data-driven method should be conducted.

Support: This study supported by Dreem sas.

1211

ASSESSING THE ACCURACY OF A DRY-EEG HEADBAND FOR MEASURING BRAIN ACTIVITY, HEART RATE, BREATHING AND AUTOMATIC SLEEP STAGING

Thorey, V.¹ Guillot, A.¹ El Kanbi, K.¹ Harris, M.² Arnal, P. J.² ¹Dreem Algorithms Team, Paris, FRANCE, ²Dreem Science Team, New York, NY.

Introduction: The development of new sleep study devices, adapted for daily use, is necessary for diagnosis of sleep disorders. However, this requires to be both suitable for daily use and capable of recording accurate electrophysiological data. This study assesses the signal acquisition of a comfortable sleep headband, using dry electrodes, and the performance of its automatic sleep staging algorithms compared to the gold-standard clinical PSG scored by 4 sleep experts.

Methods: 42 participants slept at a sleep center wearing both the Dreem headband (DH) and a PSG simultaneously. We measured 1) the EEG signal similarity between both devices, 2) heart rate, breathing frequency and respiration rate variability (RRV) agreement, and 3) the performance of the headband automatic sleep scoring compared to PSG sleep experts manual scoring.

Results: Results demonstrate a strong correlation between the EEG signals acquired by the headband and those from the PSG, and the signals acquired by the headband enable monitoring of alpha ($r = 0.75 \pm 0.11$), beta ($r = 0.74 \pm 0.14$), delta ($r = 0.78 \pm 0.16$), and theta ($r = 0.63 \pm 0.15$) frequencies during sleep. The mean absolute error for heart rate, breathing frequency, and RRV was 2.2 \pm 0.8 bpm, 0.3 \pm 0.2 cpm and 3.1 \pm 0.4 %, respectively. Automatic Sleep Staging reached an overall accuracy of 84.1 \pm 7.5% (F1 score: 83.0 \pm 8.4) for the headband to be compared with an average of 86.4 \pm 5.5% (F1 score: 86.5 \pm 5.5) for the 4 sleep experts.

Conclusion: These results demonstrate the capacity of the headband to both precisely monitor sleep-related physiological signals and process them accurately into sleep stages. This device paves the way for high-quality, large-scale, longitudinal sleep studies. **Support:** This Study has been supported by Dreem sas.

1212

A SYSTEMATIC ASSESSMENT OF ENGAGEMENT, FUNCTIONALITY, AESTHETICS, INFORMATION, AND RECOMMENDATION FEATURES IN SLEEP MOBILE APPLICATIONS

Hollimon, L.¹ Moore, J.¹ Richards, S.¹ Robbins, R.² Grandner, M.³ Chung, A.¹ Chung, D.¹ Jean-Louis, G.¹ Seixas, A.¹

¹NYU Grossman School of Medicine, New York, NY, ²Harvard University, Cambridge, MA, ³University of Arizona, Tucson, AZ.

Introduction: Initial download and use of sleep tracking is very high, but prolonged use is very low. Poor prolonged use may be attributable to several factors such as engagement, functionality, aesthetics, information, and recommendation. We appraised these five factors in 16 consumer- and research/medical- grade digital sleep devices.

Methods: Three reviewers independently assessed 16 consumer- and medical-grade sleep digital devices using the Mobile Application Rating Scale (MARS) App quality ratings which measures engagement (engagement, entertainment, interest, customization, interactivity, target group), functionality (functionality, performance, ease of use, navigation, gestural design), aesthetics (layout, graphics, visual appeal), information (Accuracy. Goals, Quality of information, Quantity of information, Visual information, Credibility, and Evidence base) and recommended on a Likert scale, with 1- Inadequate to 5 Excellent. Each subcategory is rated on a 1-5 Likert scale which is summed for each category: engagement (30), functionality (25), aesthetics (15), information (35) and recommended (yes or no).

Results: Devices that had the highest engagement score were Fitbit (27), Apple Watch (27), Garmin (27), and Dreem 2 headband (25.5). Apple Watch (30) had highest score; while Fitbit (13), Apple Watch (13), Garmin (13), Samsung Gear (13) had highest aesthetic score. While for information, ActiGraph (35), SOMNOwatch plus (35), CleveMed SleepView Monitor (35), CleveMed Sapphire PSG (35), SOMNOscreen plus (35), Nox T3 Sleep Monitor (35) and Nox A1 PSG System (35) had the highest ratings. The Dreem 2 headband has the potential induce prolong use among users with and without sleep disorders, based on high scores on engagement (25.5), Functionality (20.5), and Information (26.5).

Conclusion: Consumer- and research-grade digital devices that measure sleep have varying levels of engagement, functionality, aesthetics, information and recommendations to facilitate prolong use. Consumer grade devices had higher engagement, functionality and aesthetics scores, while research grade devices had higher information and recommendation scores. If consumer- and research-grade devices are to have prolonged use, standardization is needed across the five MARS components.

Support: K01HL135452, R01MD007716, R01HL142066, and K07AG052685

1213

SLEEPFECT TRACKER: A CROSSPLATFORM MOBILE RESEARCHKIT APP FOR SLEEP SELF-MANAGEMENT

Menon, P.¹ Seixas, A.² Pathan, Z.¹ Suhail, M.¹ Jean-Louis, G.² Ayoub, S.¹ Naqeeb, B.¹ Wani, B.¹ Mishra, S.¹ Khan, S.¹ ¹Applied Informatics Inc., New York, NY, ²NYU Grossman School of Medicine, New York, NY.

Introduction: We created Sleepfect Tracker, a researchkit-based cross platform app to explore the feasibility and acceptability of a sleep tracking app for sleep self-management.

Methods: We developed Sleepfect Tracker app on Appbakery, a DIY app making platform using ResearchKit for iOS and ResearchDroid for Android users. Sleepfect allows participants to track their sleep behavior (weekly via sleep diary) and environment (monthly), as well as their total sleep time and step counts data via Apple's HealthKit, Android step count sensors, or Fitbit (wearable). Three hundred and ninety-five (395) individual from around the globe downloaded the app and 163 unique users answered surveys on their sleep behavior, environment, and architecture. In total we collected 6429 sleep and 2882 step data points and provided insights into user sleep behavior and sleep environment. We also analyzed whether sleep duration was associated with steps. **Results:** Regarding immediate sleep environment and behavior, 11.1% worked or studied in bed, 27.0% reported having pets in bed, 39.7% red in bed prior to sleep, 40.3% watched TV in bed prior to sleep, 11.3% drank alcohol prior to bed, 9.8% smoked prior to bed or wake during night, 8.1% ate snack at bedtime, and 6.5% ate when they awake at night. 74.3% of the participants used electronic devices in their bedroom. Of the participants who used electronic devices in the bedroom, 52.1% had an average sleep duration of 6-8 hours and 29.8% reported sleeping 4-6 hours. Of the participants who did not use electronic devices in bedroom, 30.1% slept 4-6 hours, 31.8% slept 6-8 hours, and 32.45% slept 8-10 hours, on average. The relationship between steps count and sleep hours was trending, r=.16, p=.07.

Conclusion: Users can evaluate their sleep habits, monitor daily sleep-related behaviors through Sleepfect tracker. The app demonstrated initial usability and feasibility, but long-term usability and effectiveness must be evaluated. Further investigations on which functions will be more useful to help user to improve their sleep and engage users should be considered.

Support: K01HL135452, R01MD007716, R01HL142066, and K07AG052685

1214

HIGHER BEDROOM TEMPERATURE ASSOCIATED WITH POORER SLEEP: DATA FROM OVER 3.75 MILLION NIGHTS

Raj, A. Ruder, M. Rus, H. M. Gahan, L. O'Mullane, B. Danoff-Burg, S. Raymann, R. Sleepscore Labs Carlsbad, CA.

Introduction: Bedroom temperature can influence nocturnal thermoregulation and sleep. To date, limited, small experimental studies have shown that bedroom temperatures outside the recommended range of 65 and 70°F can negatively impact sleep. However, this association has not been studied in a large-scale data set. Using over 3.75 million nights of objectively measured data, we analyzed the associations between habitual bedroom temperatures and sleep.

Methods: Over 3.75 million nights of sleep and bedroom temperature data were collected using S+ by ResMed technology from 34,096 Individuals (57% male, 20-90 years, mean age 48.7 + / -14.5 years, all US residents). Multilevel regression analyses were used to analyze associations between bedroom temperature and sleep. A stricter alpha level of 0.001 was used to account for the large number of observations in the dataset.

Results: Bedroom temperature was above 70°F for 69% of nights, with the average temperature ranging between 68.8 and 76.2°F. For each 1°F increase in bedroom temperature between 60-85°F, sleep efficiency decreased by 0.06%. Likewise, higher bedroom temperatures were linked to shorter Total Sleep Time duration (-0.45 mins/°F), longer Sleep Onset Latency (+0.04 mins/°F), and longer Wake After Sleep Onset (+0.11 mins/°F), all ps<0.001.

Conclusion: Analyzing data from over 3.75 million nights, we found that many people sleep in a bedroom warmer than the optimal temperature. Further, higher bedroom temperatures - even within the recommended range for optimal sleep - are associated with poorer sleep and higher wakefulness. Bedroom thermostats and cooling options should be considered to achieve optimal sleeping temperature conditions.

Support: N/A

1215

IMPROVING SLEEP DISORDERS AND CHRONIC NECK PAIN BY ADJUSTING HEIGHT OF THE PILLOW THROUGH CERVICAL POSTURE MANAGEMENT

Yamada, S.¹ Yamada, K.²

¹16 Gou Orthopaedic Clinic, Sagamihara City, JAPAN, ²16 Gou Orthopaedic Clinic, Kanagawa-ken, JAPAN.

Introduction: Recent studies have suggested that chronic musculoskeletal system pain, including chronic neck pain, accounts for approximately 70% of all sleeping disorders. Simultaneously, sleeping disorders increase pain sensitivity and create a vicious cycle of chronic neck pain. Patients with chronic pain experience a variety of somatic symptoms (e.g., Stomach or bowel problems, Back pain, Pain in your joints, headaches, chest pain, shortness of breath, dizziness, fatigue, and Trouble sleeping) that are difficult to treat. However, treatment of both neck chronic pain and sleeping disorders through cervical posture management by adjusting height of the pillow used while sleeping has yet to be considered.

Methods: Patients who visited our hospital with chief complaints of chronic neck pain were rated according to the Numerical Rating Scale (NRS) and were asked to answer the Somatic Symptom Scale-8 (SSS-8), to ascertain the degree of somatic symptoms. Out of all the patients, only 84 scored at least 8 out of 32 in the SSS-8. Based on the individual results of the 84 respondents, they were given customized pillows to be used for 3 months. The pillows were adjusted using the SSS method developed at our clinic. We adjusted the height of the pillow by 5mm increments to check the cervical inclination angle at approximately 15 degrees in supine position, lateral position with the center line from face to neck at left-right symmetry and finally confirming smooth turning over. The respondents performed NRS and SSS-8 after 2 weeks and 3 months of using the pillow.

Results: The results of this study showed that at 0 weeks / 2 weeks / 3 months. NRS score was 6.8 / 5.1 / 4.1 (p <0.01), and the overall SSS-8 score was 13.2 / 9.9 / 8.2 (p <0.01), showed a marked improvement. By symptom, all symptoms except Stomach or bowel problems showed significant improvement. Trouble sleeping showed the highest improvement at 2.6 / 1.7 / 1.3 (p <0.01).

Conclusion: Chronic neck pain and sleeping disorders improved in the patients. These results suggested that cervical posture management by adjusting height of the pillow is an effective treatment method.

Support: None

1216

MARKEDLY DECREASED ICTAL DISCHARGES WITH POSITIVE AIRWAY PRESSURE (PAP) TREATMENT IN A PATIENT WITH REFRACTORY EPILEPSY AND SEVERE OBSTRUCTIVE SLEEP APNEA

Massoud Mona, MD, Schütz Sonja G., MD, MS University of Michigan

Introduction: Individuals with epilepsy are at increased risk for obstructive sleep apnea (OSA). Treatment of OSA with positive airway pressure (PAP) can result in decreased seizure frequency in patients with epilepsy, possibly due to decreased sleep disruption and normalization of oxygenation saturation levels when using PAP. The following case report demonstrates an example in which treatment of OSA with PAP resulted in a dramatic improvement of electroencephalographic seizures.

Report of Case: A 32 year-old man with refractory epilepsy presented to our office complaining of loud snoring, witnessed apneas, and excessive daytime sleepiness. Despite prior left frontal and left anterior temporal lobectomy, vagus nerve stimulator treatment, and excellent treatment adherence to four different antiepileptic medications, the patient continued to have 1-3 clinical seizures per day. Physical examination was notable for obesity (BMI=39.4 kg/ m2), high arched palate and Mallampati class 4 airway. Long-term eletroencephalogram (EEG) monitoring was significant for frequent interictal discharges during wakefulness and sleep, as well as frequent subclinical seizures during sleep characterized by C3/P3 polyspike bursts followed by brief rhythmic fast activity. A diagnostic polysomnogram revealed severe OSA (AHI=69/hour). The patient subsequently underwent a PAP titration study with extended EEG montage, which was notable for a marked decrease in polyspike discharges on effective PAP pressures, from an average of 24 polyspike discharges per hour on PAP pressures < 15 cm of water to an average of 3 polyspike discharges per hour on PAP pressures \geq 15 cm of water. PAP treatment was initiated following the titration study.

Conclusion: This case demonstrates that effective treatment of OSA can lead to a reduction of abnormal EEG discharges in a patient with intractable epilepsy. Additional clinical follow up is needed to assess whether PAP treatment also resulted in decreased frequency of clinical seizures.

1217

OBESITY HYPOVENTILATION SYNDROME IN A 4-YEAR-OLD CHILD

Wang, Grace¹, Guevarra, Jay², Bronstein, Jason²

¹Pediatric Pulmonology, Penn State Children's Hospital. Hershey, PA, ²Sleep Medicine, Icahn School of Medicine at Mount Sinai Hospital. New York, NY.

Introduction: four-year-old boy with morbid obesity was referred to pediatric sleep for nocturnal hypoxemia during inpatient admissions. He was found to have daytime hypoventilation, likely secondary to obesity hypoventilation syndrome (OHS).

Report of Case: During two inpatient admissions (wheezing, gastroenteritis), he desaturated to the 70s during sleep. At home, he received blow-by oxygen as he could not tolerate other interfaces. He underwent adenotonsillectomy. However, snoring, day-time sleepiness, hyperactivity and aggressive behavior persisted.

Birth history was unremarkable, though he became progressively more obese over time. His father had obesity and obstructive sleep apnea. Physical exam was notable for elevated blood pressure of 122/68 mmHg (above 99th percentile), weight and height above 99th percentile, and BMI of 32 kg/m2 (z-score \sim 4.3).

Despite extensive counseling, family declined polysomnography and labs. Awake end-tidal CO2s were elevated at 47 mmHg. Echocardiogram showed half-systemic PA pressures, right ventricular hypertrophy, and right atrial dilation.

Family began desensitization protocol in preparation for future PAP therapy and polysomnography. Pediatric endocrinology consultation revealed low suspicion for hormonal/metabolic concerns. He entered a pediatric weight loss program.

Conclusion: This 4-year-old boy demonstrated daytime hypoventilation, systemic and pulmonary hypertension, likely consequences of his severe obesity. OHS is defined as BMI >95th percentile in children and awake hypercapnia (PaCO2 > 45 mmHg) in absence of alternative hypoventilation causes (e.g. pulmonary, cardiac, neurologic, pharmacologic). Presenting symptoms may include hypersomnolence, morning headaches, cognitive deficits, and signs of cor pulmonale1. The literature consists primarily of case reports; prevalence of pediatric OHS is unknown. Obesity afflicts 18.5% of children in the United States2. Given the severity of OHS sequelae, maintaining a high index of suspicion is crucial. Consider further work-up in patients with unexplained low oxygen saturations, signs of pulmonary hypertension (unexplained dyspnea on exertion, pedal edema), polycythemia, and elevated bicarbonate1.

1218

Obstructive Sleep Apnea Due To Oromandibular Dystonia And Treated With Botulinum Toxin

Hsieh Caleb, MD MS^{1,2}, Hsu Nancy, MD³, Thomas Aaron, MD², Chang Melisa, MD^{1,2}, Ryden Armand, MD², Zeidler Michelle, MD^{1,2}

¹UCLA Department of Medicine, Department of Sleep Medicine. Los Angeles, CA, ²West Los Angeles VA Medical Center, Department of Sleep Medicine. Los Angeles, CA, ³Scripps Clinic

Torrey Pines. Lo Jolla, CA

Introduction: Abnormalities of the upper airway are an underrecognized cause of obstructive sleep apnea (OSA). Oromandibular dystonia (OMD) is characterized by involuntary contractions of the masticatory, facial, pharyngeal or laryngeal muscles usually resulting in pain, dysarthria, dysphagia, or impaired mastication. We present a patient with OMD manifest as episodic OSA and sleep maintenance insomnia treated effectively with botulinum toxin injections of the temporalis and masseter muscles.

Report of Case: A 51-year-old man was referred for fatigue and insomnia. History was notable for prior facial trauma requiring jaw surgeries with titanium prostheses and chronic OMD requiring periodic botulinum toxin injections of the temporalis and masseter muscles.

An initial home sleep test (HST) 43 days after the last botulinum toxin injection showed a respiratory event index of 6.5 events/hour. Given severity of symptoms, positive airway pressure (PAP) was initiated; however, due to poor tolerance and persistent symptoms, he was referred for attended polysomnography (PSG). The PSG was done 13 days after an injection and showed apnea-hypopnea index (AHI) of 0 events/hour. Because the patient continued to endorse episodically severe symptoms that he felt paralleled the

Case Reports from Clinical Trainees

severity of OMD and waning of botulinum toxin effect, a repeat PSG was performed at a subjective botulinum toxin effect nadir. This PSG done at 83 days post-injection demonstrated AHI of 84 events/hour. PAP therapy was thus resumed, and the patient's symptoms improved with increased frequency of botulinum toxin treatments.

Conclusion: To our knowledge, this is the first report of OSA due to chronic dystonia of the facial muscles. Botulinum toxin has demonstrated benefit in the treatment of OMD with efficacy generally lasting three to six months. While PAP remains the first-line treatment for OSA, in patients with structural or functional abnormalities of the upper airway, it is important to also consider treatment of the underlying anatomical defect.

1219

A CASE OF A NIGHT TIME AFFAIR

Ali Hamed, MD, Stevens Suzanne, MD University of Kansas Medical Center, Kansas City, KS

Introduction: Sleep associated seizures especially Nocturnal Frontal Lobe Epilepsy (NFLE) represents a spectrum of challenging clinical manifestations presenting as complex nocturnal movements/ behaviors, making the diagnosis often difficult.

Report of Case: A 64 y/o male, with history of ongoing complex movements occurring during his sleep, with no history of strokes or neurological deficits. Had extensive neurologic workup (all negative) including routine electroencephalogram (EEG), prolonged inpatient EEG (12 hours), and MRI of the brain. Home sleep study showing moderate obstructive sleep apnea (OSA) AHI 24/hour successfully treated with CPAP therapy (residual AHI 1.7/hour) with improved nighttime symptoms initially. Wife recalls events as happening only at night while sleep, as patient often confused upon waking up in the morning, at times appear to sit up and smack his lips. No nighttime hallucinations, sleep paralysis, or acting out dreams were reported. Had two episodes associated with tongue biting and loss of bladder control. Another episode happened after a daytime nap, patient went outside and was mowing his lawn, went "completely blank ", appeared confused. No daytime or nighttime seizures were ever noticed. Patient do not recall any of the above events. Repeat EEG was normal. MRI/MRA of the head /neck showed small tiny focus in left frontoparietal lobe, suggesting remote cortical ischemic injury. Polysomnography (PSG) with seizure montage showed Interictal epileptic discharges (IEDs) foci recorded in the frontal/frontopolar leads without accompanying body movements. Interictal spike and wave activity seen during stage N2.

Initially treated with carbamazepine (had skin reaction) switched to levetiracetam with complete resolution of his symptoms.

Conclusion: This case illustrates the importance of reviewing the clinical history, behavior semiology, and diagnostic ancillary testing such as polysomnography with EEG monitoring in distinguishing nocturnal epileptic seizures from other nocturnal complex behavior disorders and parasomnias.

1220

A CASE OF HYPOGLOSSAL NERVE STIMULATOR CERVICAL LEAD CUFF DISLODGEMENT

Ali Hamed, MD, Duthuluru Sowjanya, MD Department of Pulmonary, Critical Care and Sleep Medicine University of Kansas, Kansas City, KS

Introduction: Hypoglossal Nerve Stimulation (HGNS) has become an alternative therapy for moderate to severe obstructive sleep apnea (OSA) patients intolerant to PAP therapy. HGNS devices typically comprise of implantable pulse generator (IPG) placed surgically in an infraclavicular subcutaneous pocket. An electrode cuff attached to the IPG wraps around the distal portion of the of the hypoglossal nerve. This device has an implantable chest sensor that monitors the respiratory efforts.

Report of Case: A 76-year-old male with history of severe OSA (AHI 39 /hour) was intolerant to PAP therapy. HGNS was implanted in the right infraclavicular pocket under general anesthesia without any complications. Patient had successful tongue motion to stimulus per protocol intra- operatively in the OR. However, no tongue movement was noted despite maximum stimulation up to 4.5 V at follow up clinic visit. Follow up C spine x ray showed very low-lying HGNS cervical lead cuff, and possible dislodgement. Patient was taken back to the OR. Intraoperatively it was noted that the previously placed cervical lead cuff has folded back and was lying on the surface of the submandibular gland /digastric anchor site. It was dissected free and replaced on the distal inclusion branches of the hypoglossal nerve with loupe magnification and EMG confirmation (tongue deviation at 1.5 volts). Patient developed tongue neuropraxia with difficulty swallowing, difficulty speaking and right sided tongue deviation lasting for months, that gradually improved. Patient had successful HNS activation 6 months later using 2.2 V.

Conclusion: (HGNS) failure secondary to cervical lead cuff dislodgement is a rare complication and should be taken in consideration. Post-operative imaging and comprehensive clinical examination are crucial in detecting such problems. Temporary tongue neuropraxia post Hypoglossal nerve stimulator placement is another possible complication.

1221

FLUMAZENIL FOR THE TREATMENT OF HYPERSOMNOLENCE AND FATIGUE IN A PATIENT WITH EHLERS-DANLOS SYNDROME WITH MULTIPLE CARDIAC COMORBIDITIES

Pepito Donna Lea, MD¹, Markun Leslie, MD², Sampat Ajay, MD² ¹UC Davis School of Medicine, Department of Pulmonary Critical Care and Sleep Medicine ²UC Davis School of Medicine, Department of Neurology

Introduction: Ehlers-Danlos syndrome (EDS) is a genetically inherited connective tissue disorder which has a high prevalence of sleep conditions, including obstructive sleep apnea, insomnia, fatigue, and hypersomnia., Chronic fatigue is an important factor in the impaired quality of life in patients with EDS. Successful treatment of fatigue and hypersomnia with traditional wake-promoting medications and stimulants is limited, due to the high prevalence of postural tachycardia, orthostatic intolerance, and other cardiac conditions in these patients. We present a case of EDS with underlying cardiac comorbidities, hypersomnia and fatigue who had significant improvement in excessive daytime sleepiness after treatment with flumazenil.

Report of Case: A 19 yoF with PMH of Ehlers-Danlos syndrome, postural tachycardia syndrome (POTS), autonomic instability and well-controlled depression presented with symptoms of fatigue and excessive daytime sleepiness (ESS of 17/24) despite obtaining 10-15 hours of sleep each day. Polysomnogram followed by MSLT was notable for borderline excessive sleepiness without other abnormalities (PSG: AHI 3/hr, SpO2 nadir 92%; MSLT: 0 SOREMS, mean sleep latency 10 minutes). Prior autonomic and cardiac work-up revealed POTS (maximum HR 180

bpm), orthostatic intolerance and aortic root dilatation. Physical exam and previous laboratory work up for fatigue were unremarkable. A trial of flumazenil 6 mg lozenge every 4-6 hours, as needed for sleepiness, was initiated. On her subsequent visit 6 months later, patient reported 50% improvement in symptoms of fatigue and sleepiness, with decrease in ESS from 17 to 5. No adverse effects to flumazenil were reported.

Conclusion: Flumazenil is a gamma-aminobutyric acid (GABA)-A receptor antagonist that has been previously documented to provide sustained clinical benefit in treatment-refractory hypersomnolence. This case report highlights a successful alternative treatment option for hypersomnolence in patients with EDS and cardiac comorbidities, in which traditional wake-promoting agents and stimulants may be contraindicated.

1222

DOES CREATIVITY & "DOSE" ENHANCE OUTCOMES IN IMAGERY REHEARSAL THERAPY? A CASE OF SUCCESSFUL IRT

Roth Alicia J, Ph.D. & Drerup Michelle, Psy.D., DBSM Sleep Disorders Center, The Cleveland Clinic, Cleveland OH

Introduction: Imagery Rehearsal Therapy (IRT) is an efficacious treatment for Nightmare Disorder. In IRT, patients practice pleasant guided imagery techniques, then use these skills to re-script recurring nightmares, which lowers the frequency and intensity of overall nightmare activity. However, the most efficacious methods and dosage of guided imagery and nightmare re-scripting is undetermined.

Report of Case: The patient was a 70-year-old male with Nightmare Disorder. Patient denied any precipitating event or trauma associated with nightmare onset. He has a longstanding history of depression and OSA (uses CPAP). He was taking Seroquel, which reduced severity of nightmares but not frequency. Trials of other medications for nightmares had failed (including prazosin, Depakote, and trazodone).

Patient presented as highly distressed, exhibited distrust towards medical providers, and was skeptical about the effectiveness of IRT. Despite his skepticism, patient self-initiated very detailed and media-enhanced methods for pleasant guided imagery and nightmare re-scripting, including written narratives, voice recordings, and created a movie of his re-scripted nightmare with pictures set to music. He listened to the recordings 2-3x/day. Themes of nightmares included lack of mastery over problems; patient's re-scripted dreams put him back in control of frightening scenarios.

Nightmare logs at baseline showed sleep quality=1.9/5; average=2.0 nightmares/night; average intensity= 6.2/10. At week 15 of treatment, sleep quality=3/5; nightmares/night average=0.25; average intensity=6/10. Sleep disturbance also improved (ISI=18-moderately severe clinical insomnia to 11-subthreshold insomnia); mood was stable (PHQ=5-mild depression).

Conclusion: Previous studies have suggested that IRT increases patients' sense of mastery or perceived self-efficacy over nightmares (Rousseau et al., 2018). Additionally, higher verbal memory in persons with trauma-related nightmares has been shown to improve nightmare frequency and severity in IRT (Scott et al., 2017). In this case study, self-efficacy may have been activated by the highly detailed and media-enhanced imagery the patient created. Further empirical research on the mechanisms for enhancing IRT is warranted.

1223

CENTRAL SLEEP APNEA AND CHIARI 1 MALFORMATION IN A PEDIATRIC PATIENT WITH KLIPPEL-FEIL SEQUENCE

Martirosyan Zara, MD and Malhotra Sonal, MD, MPH

Introduction: Klippel- Feil Sequence (KFS) is a rare congenital condition that classically presents with a triad of congenital cervical spine fusion, reduced cervical spine flexion and low posterior hairline. KFS has been associated with several comorbidities including congenital heart defects, hearing loss, renal dysfunction, Chiari malformation and sleep disordered breathing (SDB). The co-occurrence of SDB and chiari malformation type 1 (CM1) has been reported in multiple individuals. However, the pathological basis of the connection between CM1 and SDB in the setting of KFS is not clearly understood.

Report of Case: Here we present a pediatric case report of a patient with KFS, SDB, drooling and dysphagia. The drooling and dysphagia prompted an MRI that revealed CM1. Baseline polysomnogram (PSG) showed mild central sleep apnea with apnea-hypopnea index (AHI) 6.06 comprised of central apnea index 4.81 and obstructive apnea index 1.28. Posterior fossa decompression of this patient, following neurology recommendations, resolved majority of the symptoms, namely the drooling, nocturnal cough, dysphagia and sleep disturbances. However unexpectedly repeat polysomnogram eight weeks after posterior fossa decompression revealed worsening central sleep apnea despite the patient being clinically asymptomatic.

Conclusion: Taken together this case highlights the point that while it is critical to recognize the association of SDB in the setting of KFS, decompression alone may not be sufficient to completely alleviate SDB and/or certain neurological symptoms such as nocturnal coughing. Our observations are consistent with the hypothesis that SDB in a setting of KFS is multifactorial and pathophysiology is not clearly understood.

1224

TREATMENT OF SEVERE OSA USING LOW FLOW OXYGEN IN A NEONATE WITH ROBIN SEQUENCE

Saeed Sidra, Amos Louella

Medical College of Wisconsin, Milwaukee, WI

Introduction: Robin Sequence (RS) involves the clinical triad of micrognathia, glossoptosis and cleft palate. There is a spectrum of severity, but most neonates with RS have upper airway obstruction, resulting in severe obstructive sleep apnea, sometimes requiring surgical interventions such as tongue-lip adhesion, mandibular distraction, or tracheostomy. We present an infant with RS and severe obstructive sleep apnea which was managed with supplemental oxygen.

Report of Case: Our patient was born at 39 weeks gestation with RS. He had a normal DNA microarray. He was discharged after a 3 week NICU hospitalization for poor feeding. Over the next 2 months, he had poor weight gain and worsening obstructive breathing and was evaluated by craniofacial surgery at that time. Room air polysomnography (PSG) was recommended and revealed an AHI of 21, REM AHI of 48, supine AHI of 25, prone AHI of 19, mean SPO2 of 98%, minimum SPO2 of 61%, and normal capnography with 0% of the time spent > 50 mmHg. A repeat sleep study on 1/4LPM oxygen in the supine position revealed an AHI of 1.7, mean SPO2 of 99%, minimum SPO2 of 92%, and normal capnography. He was discharged on supplemental oxygen. At 4 months of age, he had good weight gain. At 10 months of age,

C. Case Reports

room air PSG revealed persistent OSA with an AHI of 7.2, REM AHI of 21, mean SPO2 of 97%, minimum SPO2 of 81%, and normal capnography. At age 3 yrs, his PSG on room air showed resolution of his OSA with an AHI of 0.6, mean SPO2 of 97%, minimum SPO2 of 87% and normal capnography.

Conclusion: This case illustrates the spectrum of severity of RS and the utility of low flow oxygen to treat OSA in this patient population.

1225

SEXSOMNIA: A CASE OF SLEEP MASTURBATION AND SLEEPING IN AN OPEN WORK ENVIRONMENT

Kim David S. DO¹, Foster Brian E. DO², Rizzo Meagan M. MD^{2,3}, Collen Jacob F. MD^{2,3}, Soca Rodolfo MD^{2,3}

¹Department of Internal Medicine, Walter Reed National Military Medical Center, Bethesda, MD, ²Sleep Medicine Center, Walter Reed National Military Medical Center, Bethesda, MD, ³Department of Medicine, Uniformed Services University of the Health Sciences, Bethesda, MD

Introduction: Sleep sex or sexsomnia is classified in the International Classification of Sleep Disorders as a non-rapid eye movement (NREM) parasomnia. The overall prevalence is unknown but several conditions such as obstructive sleep apnea (OSA), shift work, and/or insufficient sleep have been reported as factors affecting the frequency of NREM parasomnias. Parasomnias, with sexsomnias in particular, can cause significant emotional distress to patients and their families. We present a case of co-morbid sexsomnia and OSA in a patient serving in the military that had the complicating factor of sleeping in open quarters.

Report of Case: A 40-year-old male in active duty military without significant past medical history or medication use presented to clinic with a two year history of disruptive sleep masturbation reported by wife. Patient's wife reported no instances of attempted sexual intercourse. The patient had no recollection of the episodes. The frequency and nature of the episodes were causing personal and marital distress. Patient was also concerned about work responsibilities, since he was periodically required to sleep in open environments. After initial clinic evaluation, patient underwent a video polysomnography (vPSG) with an extended EEG montage. No parasomnia activity was captured on vPSG but patient was diagnosed with moderate OSA. He was started on continuous positive airway pressure (CPAP) therapy, resulting in decreased number of episodes with relapses corresponding to nights without CPAP usage.

Conclusion: Sexsomnia cases require careful history taking and evaluation. The nature of the episodes and the sleep environment of the patient must be examined for any medico-legal ramification as there are court precedents of sexual assault charges made in sexsomnia-related cases. The military environment is unique with group-sleeping conditions, often in austere environments. If sexsomnia were to be misinterpreted as indecent exposure, under the military code of conduct, this could have significant career implications.

1226

A CASE OF HYPOGLOSSAL NERVE STIMULATION INDUCED CHEYNE-STOKE CENTRAL SLEEP APNEA

Rezayat Talayeh, DO, MPH & Chang Melisa, MD VA Greater Los Angeles, Los Angeles, CA, University of California, Los Angeles **Introduction:** Treatment of obstructive sleep apnea (OSA) with positive airway pressure (PAP), mandibular advancement devices (MAD) and oral surgery have been reported to lead to emergent central sleep apnea (CSA). In this case report the emergence of CSA in a Cheyne-Stokes pattern following the use of hypoglossal nerve stimulator as a treatment modality for OSA is discussed.

Report of Case: A 70-year-old man with a history of hypothyroidism and severe OSA diagnosed via a home sleep apnea test with a respiratory event index (REI) of 38 events/ hr was intolerant of PAP therapy and an MAD did not effectively treat his OSA. He was deemed an appropriate candidate for hypoglossal nerve stimulation following a drug induced sleep endoscopy. Following implantation and activation, he developed a lip droop and was ruled out for a stroke. A polysomnogram was completed which showed significant improvement in his sleep apnea at a voltage range of 1.4-17V. At 1.8V he developed REM- supine central events. When the voltage was further increased to 1.9-2.0V non-REM supine central events arose. These events appeared to have Cheyne-Stoke morphology with a cycle duration of over 50s. He was set to an amplitude of 1.6 V with a positional belt for treatment of his OSA without any emergent CSA.

Conclusion: This patient developed central sleep apneas with Cheyne-Stoke morphology following treatment of obstructive sleep apnea using a hypoglossal nerve stimulator. The central events began at higher voltage settings (greater than 1.8V). He had no history of heart failure or arrhythmias. This higher voltage may lead to overshoot of the tongue out of the airway resulting in hyperpnea, hypocapnia and central apnea but the underlying pathophysiology for the Cheyne-Stoke pattern in the absence of heart failure remains unknown.

1227

A CASE OF A FEARFUL SLUMBER

Asis Aristotle, MD, Wilson Annise Georgette, MD, Alapat Philip Mani, MD,

Department of Pulmonary, Critical Care and Sleep Medicine, Baylor College of Medicine, Houston, TX

Introduction: Isolated sleep paralysis (ISP) occurs when rapid eye movement (REM)-based atonia intrudes into wakefulness, outside the context of narcolepsy, substance abuse, mental disorder or other medical conditions. No "gold standard" assessment and diagnostic instrument currently exists.

Report of Case: A 63-year old female with hypersomnia and positive airway pressure (PAP)-controlled obstructive sleep apnea was referred for recurrent episodes of paralysis during sleep-wake transitions, lasting 15-20 seconds, occurring every 2-3 years since the age of 15, and associated with fear and anxiety. Episodes were more frequent in the last 2 years after significant sleep deprivation and starting a weight loss supplement, BIO-X4, which contains green tea and probiotics. No cataplexy, or history of traumatic brain injury and stroke were identified. Epworth Sleepiness Scale score was 14 on armodafinil. Reported sleep amounts were regularly scheduled 6-7-hour periods, with no suggestion of circadian dysfunction. In 2016, polysomnogram showed Apnea-Hypopnea index of 2.6/hour, Respiratory Disturbance Index of 13.8/hour with oxygen nadir of 92% in the setting of hypersomnia. Continuous PAP of 11 cmH20 was initiated after a successful titration with controlled residual AHI during follow-ups. Multiple Sleep Latency Test during the same time revealed mean sleep latency of 5.5 minutes and no sleep-onset REM with 5 naps. Brain imaging and electroencephalogram were both normal as well as drug panel, blood

C. Case Reports

counts, metabolic profile and thyroid function. Decreased episodes and severity of recurrent ISP were reported after discontinuation of the supplement. Apart from anxiety related to the episodes, the patient denied any interference with daytime function.

Conclusion: Isolated sleep paralysis is an important sleep disorder that requires proper evaluation to rule out competing diagnoses and consideration of therapeutic interventions. Likely associated with a lack of understanding and available literature, the prevalence in the general population is likely higher than what is currently perceived.

1228

A CASE OF PULMONARY HYPERTENSION AND SEVERE OBSTRUCTIVE SLEEP APNEA IN A PEDIATRIC PATIENT WITH DOWN SYNDROME

Asis Aristotle M.D., MD¹, Kaplan Kevin M.D.²

¹Department of Pulmonary, Critical Care and Sleep Medicine, Baylor College of Medicine, Houston, TX, ²Department of Pediatric Pulmonary and Sleep Medicine, Texas Children's Hospital, Houston, TX

Introduction: Children with Down syndrome (DS) are at risk for obstructive sleep apnea (OSA) and related health consequences. Adherence to treatment plans, especially positive airway pressure (PAP), can be difficult for people with DS. We present a case of a pediatric patient with DS who developed pulmonary arterial hypertension (PAH) due to poor adherence of OSA therapy.

Report of Case: A 4-year-old male with a past medical history of DS was diagnosed with severe OSA and hypoventilation at 18 months of age - AHI 27, (oAHI 27), CO2 above 50 mmHg for 53.2% of total sleep time (TST)). A titration study showed that on a bi-level pressure (BPAP) of 11/6 cm H2O his OSA was improved - AHI 5.9 (oAHI 3.7), O2 nadir of 93%. The family attempted PAP at home but discontinued after 2 weeks. He underwent an adenoidectomy at 22 months of age and tonsillectomy and repeat adenoidectomy at 40 months of age. Patient presents to the emergency center with respiratory distress. Chest films showed cardiomegaly. Echocardiogram revealed evidence of worsening PAH (tricuspid regurgitation of 3.5-3.8 m/s and flattening of the interventricular septum). He was admitted to the pediatric intensive care unit for further management of his PAH attributed to his underlying uncorrected OSA and hypoventilation. Repeat polysomnogram showed continue severe OSA and hypoventilation - AHI 30.3 (oAHI 30.3) CO2 above 50 mmHg for 89% of TST. Repeat titration study showed that a pressure of 11/7 cm H2O improved his OSA- AHI 6.4 (oAHI 6.4) CO2 max of 52 mmHg. Treatment with BPAP was initiated prior to discharge. Repeat echocardiogram 2 months after admission showed improvement of the PAH while on BPAP without additional vasoactive drug therapy.

Conclusion: Children with DS are at high risk for OSA. Although treatment can be difficult, proper management of the OSA essential to preventing potential serious health consequences.

1229

MANAGEMENT OF SEVERE SLEEP RELATED DISORDERED BREATHING IN A PEDIATRIC CASE OF VAGAL NERVE STIMULATION

Ryba-White Benjamin, Molero Helena, Irfan Muna Hennepin Healthcare, University of Minnesota, Minneapolis Veterans Affairs Medical Center **Introduction:** Vagal nerve stimulator (VNS) can be an effective treatment for refractory epilepsy but can lead to sleep related disordered breathing in the form of obstructive and central sleep apnea. We describe a pediatric case with VNS developing severe obstructive sleep apnea (OSA) and the course of management.

Report of Case: A 10-year-old boy with history of cerebral palsy, medically intractable epilepsy secondary to Lennox-Gastaut syndrome, and noted improvement in seizure frequency with VNS placement, presented to the pediatric sleep clinic with concern for sleep disordered breathing in the setting of snoring and witnessed gasping at night. The polysomnogram (PSG) showed severe OSA with apnea/hypopnea index (AHI) of 21.8 events/hour and associated hypoxemia. He subsequently underwent adenotonsillectomy. Post procedural PSG demonstrated persistent severe OSA. The obstructive events had rhythmicity temporally coincident with the VNS cycling. Subsequent PSG, after turning VNS off, showed a reduction in AHI to 11 events/hour without hypoxemia. Positive airway pressure (PAP) was initiated during this PSG, however titration was rendered unsuccessful due to VNS activation. A final titration PSG was performed with optimal resolution of OSA with VNS turned off. Since the patient did not have nocturnal seizures, it was recommended to turn the VNS off at nighttime with CPAP use. Patient was noted to have less sleep disruptions at night and more alertness during the day.

Conclusion: This case poses a challenging situation where patient's epilepsy responded to VNS but the device contributed to OSA and affected PAP titration. One strategy to improve OSA in such cases is to change VNS parameters at the risk of changing stimulation settings effective for seizure control. Turning VNS off during sleep provided a simpler solution while ensuring effective treatment. Thus, clinicians should be vigilant about concomitant OSA treatment in the setting of vagal nerve stimulation, which can improve quality of life.

1230

AN UNUSUAL PRESENTATION OF DEMENTIA WITH LEWY BODIES IN A SEPTUAGENARIAN

Geil Eric S, Agudelo Christian, Ascher Kori, Sun Xiaoyan, Velez-Ruiz Naymee, Ramos Alberto R UHealth Sleep Medicine Program, Miller School of Medicine,

University of Miami, FL Department of Neurology, Miller School of Medicine, University of Miami, FL

Introduction: Dementia with Lewy bodies (DLB) can be associated with degeneration of the hypocretin system and reduced hypocretin levels. However, reports of DLB initially presenting with narcolepsy with cataplexy symptoms are sparse.

Report of Case: A 77-year-old man presents with two years of new onset hypersomnia, tremors, memory difficulties, and gait instability. Before his sleep evaluation, he was started on levetiracetam for suspected seizures and levodopa for suspected Parkinson's disease, without improvement. During his evaluation for hypersomnia, he reported loud snoring, witnessed apneas, sleep maintenance difficulty, hypnagogic hallucinations of well-formed images, and vivid dreams with dream enactment behavior occurring almost nightly. He had witnessed episodes of cataplexy described as transient lower extremity weakness with associated bifacial weakness, inability to speak, and no loss of consciousness, self-injury, loss of bowel/bladder control, or post-event symptoms. These events completely resolved after 1-minute, occurred while sitting or standing, and were spontaneous or triggered by anger or anxiety. His Epworth Sleepiness Scale was 18 and Ullanlinna Narcolepsy Scale was 35, suggestive of narcolepsy with cataplexy. Physical examination did not reveal

Parkinsonism or resting tremor. He subsequently underwent a baseline polysomnogram that showed an apnea-hypopnea index of 32.7 events/ hour. During an in-laboratory titration study, he had REM sleep without atonia. A 30-minute EEG was interpreted as normal and a typical cataplexy-like event captured during continuous EEG monitoring had no ictal correlate. Despite adherence to positive airway pressure therapy, he continued to have hypersomnia, cataplexy, and visual hallucinations during transitions from wake to sleep. He was tapered off levetiracetam and levodopa with a plan for further evaluation of probable DLB.

Conclusion: Narcolepsy with cataplexy symptoms is an uncommon presentation of probable DLB as seen in this case with two core clinical features of DLB (RBD and visual hallucinations), suggesting neurodegeneration of the hypocretin system.

1231

SLURRED SPEECH- UNUSUAL PRESENTATION OF CATAPLEXY IN CHILDHOOD NARCOLEPSY

Reddy Abhishek M.D., Maddox Mary Halsey, M.D. University of Alabama at Birmingham, Department of Pediatric Pulmonary and Sleep Medicine

Introduction: Narcolepsy, a chronic neurological disorder of excessive daytime sleepiness, cataplexy, sleep paralysis, and hypnagogic/ hypnopompic hallucinations, frequently presents in late childhood/ early adolescence. Cataplexy, the most specific symptom, presents as transient loss of muscle tone causing weakness. Approximately 80% of children with narcolepsy present with cataplexy1.

Report of Case: Eight year old African American boy presented to sleep clinic with concerns for excessive daytime sleepiness and slurred speech. Teachers initially referred to speech therapy because of slurred speech. There, speech got noticeably worse with frustration. Subsequently, sleepiness developed, and his pediatrician referred him for polysomnogram (PSG) and Mean Sleep Latency test (MSLT). After unremarkable PSG, MSLT demonstrated mean sleep latency of 10 minutes with no SOREM sleep. Patient came for evaluation in Sleep Medicine clinic where family reported worsening speech with extreme emotions and profound sleepiness, Epworth of 20. Because of the presentation and lack of REM sleep on MSLT, brain MRI was ordered and was normal. HLA typing for narcolepsy positively showed HLA haplotype associated with narcolepsy. Patient was diagnosed with narcolepsy with cataplexy and started on Modafinil 50 mg. Modafinil was progressively increased to 200 mg in a.m. and 100 mg at noon. To address cataplexy symptoms, he started Venlafaxine 12.5 mg and increased to 25 mg. Epworth score improved on Modafinil, his cataplexy symptoms, including speech difficulties, subsided on Venlafaxine. Patient's communication improved, as did school performance and social life, and patient is currently thriving in 10th grade. Current Epworth is 5 and current medications include Modafinil 200 mg in the morning, 100 mg at noon and Venlaflaxine 25 mg at night. Conclusion: This case report highlights the importance of recognizing unusual presentations of cataplexy and the impact of treatment. These symptoms can be easily missed and erroneously attributed to developmental delay or behavioral issues.

1232

IMPROVEMENT IN EXCESSIVE DAYTIME SLEEPINESS IN TWO NARCOLEPTIC PATIENTS ON ORAL CONTRACEPTIVES

Macias Maria, MD, Singh Suprya, MD, Patel Amee, DO Baylor College of Medicine, Texas Children's Hospital **Introduction:** Narcolepsy is a chronic disorder characterized by excessive daytime sleepiness (EDS). We present two patients with narcolepsy with improvement in symptoms with the use of oral contraceptives.

Report of Case: Patient A is a 15-year-old female with a family history of narcolepsy who presents with excessive daytime sleepiness and cataplexic episodes (eye twitching, abnormal sensations of the face, drop attacks). Her pediatric daytime sleepiness scale score (PDSS) was 29. The polysomnography (PSG) did not record sleep disordered breathing. Multiple sleep latency test (MSLT) recorded 4 out 4 sleep onset rapid eve movement period (SOREMPS) and mean sleep onset latency (SOL) of 6.2 minutes. She was diagnosed with Narcolepsy Type 1 and started on stimulants as family was not interested in Xyrem. Over two years, her PDSS the doses of the stimulants were increased due to significant daytime sleepiness. Caregiver was not interested in Xyrem. Patient demonstrated increased sleepiness during menstrual cycles. She was treated with OCPs for menorrhagia. Her EDS improved, PDSS decreased from an average of 24.5 to 17.5 and the dose of the stimulants was decreased.

Patient B is an 11-year-old female with excessive daytime sleepiness and fragmented sleep with multiple awakenings. She took frequent naps during the day. Her PDSS was 29. The PSG showed mild OSA and the MSLT recorded 3 out of 4 SOREMPS and SOL was less than 1 minute. She was diagnosed with Narcolepsy Type 2. During her treatment course, several medication regimens were trialed but were not effective including stimulants, Modafinil, and Armodafinil. Caregiver was not interested in Xyrem. At the age of 13 years, she started OCPs for dysmenorrhea. Her EDS improved and PDSS decreased from an average of 29 to 16.75.

Conclusion: We present two patients with narcolepsy who continued to have EDS in spite of treatment with wake promoting agents and daytime naps. Their EDS and PDSS improved after initiating OCP therapy Pubertal changes may have a significant influence on narcolepsy patients. The use of OCPs may be beneficial in conjunction to wake promoting agents.

1233

MITOCHONDRIAL MYOPATHY MAKING IT HARD TO SLEEP! OSA MANAGEMENT IN MITOCHONDRIAL MYOPATHY WITH A VARIANT IN SNAPC4 AND PURA GENES

Okorie Ugorji, MD, Monceaux Brittany, MD, Smalley Megan, MD; Roberts Edmond, MD, Liendo Cesar, MD, Chernyshev Oleg, MD Ochsner-LSU Shreveport; Shreveport, LA 71130

Introduction: No standard of care exists for management of sleep disorders/sleep disordered breathing (SDB) and Mitochondrial myopathies. Our case report describes our experience with this condition in a pediatric patient.

Report of Case: A ten-year-old middle eastern male with PMH of Mitochondrial myopathy was referred to Sleep Medicine by ENT with reported snoring, witnessed apneic spells and daytime fatigue. Flexible Video Laryngoscopy did not reveal tonsillar or adenoid hypertrophy so it was postulated that his OSA was a result of his craniofacial abnormalities and global hypotonia.

Genetic workup showed Mitochondrial complex II & III deficiency from a variant in SNAPC4 gene and PURA gene leading to failure to thrive, severe developmental delay, generalized muscle weakness, bradycardia requiring pacemaker, global hypotonia requiring nocturnal oxygen for chronic respiratory failure and G-tube placement due to difficulty with feedings.

Diagnostic PSG showed severe OSA with AHI of 11.3, RDI 11.8, REM AHI 56.0, REM RDI 56, and minimum oxygen saturation 85%. Subsequent PAP titration led to initiation of BIPAP therapy with settings of Auto-BiPAP EPAP min 5, IPAP max 20; PSmin 4; PSmax 6 cm H2O and continuation of nocturnal home oxygen. Sleep fragmentation improved to an arousal index of 3.1 with BIPAP. The patient and caregiver presented to the Sleep Medicine clinic 1 month after the PAP titration study with objective report showing >4 hours use >70% of the time and subjective satisfaction with BIPAP therapy with improvement in snoring and apnea.

Conclusion: Mitochondrial disorders lead to a deficiency of ATP affecting all organ systems and is most recognizable in the form of neuromuscular impairments. Neuromuscular impairments can translate into SDB issues such as OSA. Patients with genetic conditions such as mitochondrial myopathy should be routinely screened and evaluated for SDB and treated if warranted to significantly improve morbidity, mortality and quality of life.

1234

CONSTRICTIVE PERICARDITIS, AN UNLIKELY CAUSE OF CENTRAL SLEEP APNEA

Introduction: Central sleep apnea (CSA) and Cheyne stokes respiration (CSR) is a well-recognized complication of heart failure across multiple New York Heart Association functional classes. We present a case of CSA associated with constrictive pericarditis and normal systolic function.

Report of Case: 43-year-old male with past medical history of severe obstructive sleep apnea noted to have high residual apnea-hypopnea index (AHi) while using automatic continuous positive airway pressure, suspected to be central events. The patient also had progressive dyspnea on exertion, chest pain, and bilateral transudative pleural effusions. Pulmonary function testing was normal. He underwent a split night PSG which demonstrated severe OSA with AHi 81/ hour during baseline. With CPAP titration, AHi was 30/hr, with predominately central events. Transthoracic echocardiogram demonstrated reduced stroke volume (LVOT VTI .085), dilated IVC, and EF 55%, septa! bounce, and annulus reversus (possible constriction). Right and left heart catheterization showed equalization of the diastolic pressures (RA 35 mm Hg, RV 48/30, PA 49/30, PCWP 29, Cl 1.6, SVR 1118 dynes, PVR 216 dynes), no coronary disease, and codominant system. Technetium 99 pyrophosphate scan demonstrated symmetric uptake in left and right ventricles, but abnormal thickening of pericardium suspicious for constrictive pericarditis. His symptoms progressed despite aggressive medical therapy and he was ultimately taken to surgery for pericardiectomy where it was noted his pericardium was extremely thickened and densely adhered to the myocardium, consistent with constrictive pericarditis. There was immediate improvement in hemodynamic status post-operatively. At post discharge follow up, he was NYHA class I, with resolution of all signs of heart failure. Repeat polysomnogram demonstrated persistent OSA, but resolution of CSA at baseline and with CPAP.

Conclusion: Constrictive pericarditis has not previously been reported as a cause of CSA or CSR. This patient had complete resolution of his heart failure symptoms with definitive resection of his pericardium.

1235

REM SLEEP ASSOCIATED HYPOVENTILATION

Rojanapairat Oragun MD, Beggs Abigail MD, Chang Melisa MD, Thomas Aaron MD Pulmonary, CriUcal Care and Sleep Medicine Section, West Los Angeles Veterans Affairs Healthcare center and the David Geffen School of Medicine at UCLA

Introduction: Hypoventilation is a spectrum of respiratory disorders that is frequently found in patients with chronic obstructive pulmonary disease, restrictive lung disease (eg obesity, neuro-muscular, severe interstitial lung disease, and chest wall disease), chronic sedative use, and hypothyroidism. Rapid eye movement (REM) sleep hypoventilation may be the first manifestation of hypoventilation prior to development of non-REM sleep hypoventilation and eventual awake alveolar hypoventilation.

We present a case of hypoventilation during REM sleep with mild restriction on pulmonary function testing, prior to the development of obesity hypoventilation syndrome (OHS).

Report of Case: 68-year-old male with past medical history of diastolic heart failure, class Ill obesity (BMI 46), hypertension, chronic kidney disease lllb, and diabetes mellitus underwent split night polysomnography for evaluation of snoring, witnessed apneas and excessive daytime sleepiness. The study was significant for an apnea hypopnea index of 105/hour, and REM sleep sustained desaturation to a nadir of 72% without apneas or hypopneas, suspicious for hypoventilation. The derangements during REM sleep did not correct during PAP titration despite CPAP and supplemental oxygen. End tidal capnography was not available for the study. Follow up PFT demonstrated normal spirometry, mild restrictive lung volumes, ERV 27%, and severely depressed DLCO which corrected for alveolar volume. Daytime arterial blood gas did not reveal hypercapnia or hypoxemia (7.37/39/78/23). He underwent successful nocturnal titration with average volume assured pressure support with the final settings of IPAP 24-30, EPAP 20, VT 560 (8 ml/kg IBW), rate of 12 breaths per minute and no supplemental oxygen.

Conclusion: This patient demonstrates REM sleep hypoventilation without overt OHS during all stages of sleep, which likely would progress to OHS over time. OHS is associated with increased rates of chronic heart failure, pulmonary hypertension, hospitalizations for respiratory failure, and mortality. Early recognition and treatment are important in improving morbidity and mortality.

1236

A CASE OF SYSTEMIC AMYLOIDOSIS AND SLEEP-RELATED HYPOVENTILATION

Castner Lauren M., D.O. and Garwood Mark D., M.D. University of Michigan, Ann Arbor, MI

Introduction: Amyloidoses are a group of systemic diseases characterized by misfolded protein fragment deposition within the organs, including the heart, kidney, liver, gastrointestinal tract, nervous system, pulmonary system, and soft tissues1. Obstructive and central sleep apnea are known to occur frequently in those with cardiac amyloidosis. This case discusses a patient with systemic amyloidosis and chronic hypercarbic, hypoxic respiratory failure.

Report of Case: A 66 year old female with a history of systemic amyloidosis, non-ischemic cardiomyopathy, hypertension, and obstructive sleep apnea was admitted for acute on chronic heart failure. Despite intravenous diuresis, she remained hypoxemic, requiring 1 liter per minute of oxygen.

She was found to have bilaterally reduced diaphragmatic excursion and a restrictive ventilatory defect on spirometry. She had a preceding history of chronic carbon dioxide retention with elevated CO2 levels for greater than a year (52-74 mmHg).

Sleep medicine was consulted to assist in evaluation of the patient's obstructive sleep apnea and hypoxic, hypercarbic respiratory failure. Baseline polysomnogram revealed sleep related hypoventilation with transcutaneous CO2 (TCO2) ranging between 77-86 mmHg without clear obstructive sleep apnea. A bilevel positive airway pressure (BPAP) titration was then performed (TCO2 54-69 mmHg) and while the patient's obstructive sleep apnea was well treated, sleep-related hypoventilation and central apneas persisted. Average volume assured pressure support (AVAPS) was initiated for management of sleep related hypoventilation. In follow up, the patient is feeling well, off oxygen, with daytime TCO2 38 mmHg.

Conclusion: This case demonstrates a rare complication of systemic amyloidosis in the setting of respiratory failure attributed to amyloid infiltration of the diaphragm. In the few previously reported cases of neuromuscular respiratory failure in systemic amyloidosis there is rapid progression and high mortality3, which highlights the importance of assessing for sleep disordered breathing and additional causes of respiratory failure in a patient with a complex systemic disease.

1237

POSITIONAL CENTRAL SLEEP APNEA IN A CHILD: NOT ALWAYS OBSTRUCTIVE

Powell Weston MD, PhD, Chen Maida MD Seattle Children's Hospital

Introduction: Spinal cord compression at the craniocervical junction can cause central sleep apnea (CSA) in children, due to mechanical disruption of respiratory control centers. Polysomnography (PSG) pre/post surgical decompression is indicated to evaluate treatment response.

Report of Case: A 5-year-old with Wolf-Hirschhorn syndrome, developmental delay, partial agenesis of the corpus callosum, hypotonia, incomplete segmentation of C1 with subluxation leading to stenosis, cleft palate, and obstructive sleep apnea (OSA) presented for post-surgical PSG. Prior PSGs revealed oAHI 55/hr and 7/hr, cAHI 3/hr and 2/hr at 1 and 10 months of age, respectively. At 46 months, patient underwent suboccipital decompression at C1 for severe craniocervical stenosis. Postoperative PSG revealed emergence of CSA with cAHI 8/hr and stability of OSA with an oAHI 8/hr. Positional analysis revealed worsening in lateral position (lateral cAHI 10.6/hr, supine cAHI 4.6/hr). Central events were up to 33 seconds long with nadir desaturation of 76% in lateral position compared to 20 seconds and nadir 88% in supine position. Head CT and MRI showed incomplete ossification of the C1 vertebra with subluxation of the left lateral mass leading to absence of CSF flow at the craniocervical junction. Repeat decompression and fixation is planned by neurosurgery; in the interim supine sleep was recommended and family declined BiPAP.

Conclusion: In our case, CSA worsened with left lateral positioning and improved with supine positioning. Lateral positioning likely increased subluxation of the left lateral mass seen on CT/MRI, and eased subluxation in supine sleep causing position-dependent dynamic impingement of the respiratory control centers at the level of subluxation. The absence of findings prior to decompression may reflect increased instability after decompression or increased stenosis with growth. Our case highlights the importance of positional analysis for central as well as the more conventional obstructive sleep apnea to understand pathogenesis and guide therapy.

1238

CENTRAL SLEEP APNEA WITH CHEYNE STOKES BREATHING IN A PEDIATRIC PATIENT WITH HEART FAILURE

Powell Weston T., MD, PhD, Chen Maida, MD, MacKintosh Erin, MD

Seattle Children's Hospital

Introduction: Central sleep apnea due to Cheyne-Stokes breathing (CSA-CSB) commonly occurs in adult patients with chronic heart failure, but has rarely been described in children. We describe a case of CSA-CSB in a pediatric patient with dilated cardiomyop-athy and acute heart failure.

Report of Case: A 12-year-old is admitted to the intensive care unit in the setting of new diagnosis of dilated cardiomyopathy leading to acute systolic and diastolic heart failure requiring inotropic infusions. After admission she is noted to have self-resolving desaturations on continuous pulse oximetry while asleep. Sleep medicine is consulted for further evaluation. She has desaturations during naps and night-time sleep that are not associated with any snoring, congestion, cough, choking, or gagging. She underwent adenotonsillectomy 7 years prior. Her father has dilated cardiomyopathy. Current medications are spironolactone, furosemide, ranitidine, loratadine, enoxaparin, milrinone and epinephrine infusion. Physical exam reveals an obese girl with absent tonsils, clear breath sounds, and tachycardia. Cardiac MRI showed severely dilated left ventricle with global hypokinesia and depressed function (EF 7%). Polysomnography reveals AHI 24.2/hr, with oAHI 0/hr and cAHI 24.2/hr. No snoring, flow limitation, or thoracoabdominal paradox is seen. Cheyne-Stokes respiration is present leading to diagnosis of CSA-CSB.

Supplemental oxygen is provided to blunt desaturations. While waiting for titration PSG she underwent placement of a left ventricular assist device and orthotopic heart transplantation. Following heart transplantation she had resolution of desaturations while asleep without supplemental oxygen; family declined repeat polysomnography.

Conclusion: Central sleep apnea with Cheyne-Stokes breathing is associated with increased mortality in adult patients with heart failure and provides important prognostic information if identified. The prevalence of central sleep apnea and its implications are unknown in pediatric patients and our case highlights the need to consider sleep disordered breathing as a cause of desaturations in patients with acute heart failure.

1239

MIND BLOWN: EXPLODING HEAD SYNDROME AS A SIDE EFFECT OF MARIJUANA

Missak Christopher DO¹, George Jenie MD¹,

Gurubhagavatula Indira MD, MPH^{1,2}

¹Division of Sleep Medicine, University of Pennsylvania, ²Crescenz VA Medical Center, Philadelphia

Introduction: Marijuana use is increasing the United States and has been associated with increased sensory perception, euphoria, and altered cognition. Exploding head syndrome is a parasomnia characterized by loud explosion-like noises occurring prior to or during sleep. This the first report to link marijuana use with the occurrence of EHS.

Report of Case: A 45-year-old man with depression, anxiety and multisubstance abuse reported two years of neuropsychiatric symptoms including: "fireworks going off in my brain," visual

hallucinations, and sudden arousals characterized by panic and vertigo. He reported bouts of occupational stress and severe anxiety previously treated with clonazepam for six months, but discontinued it due to poor clinical response. Normal findings were reported on a previous work-up including: MRI, EEG, and head CT Scan.

During his visit, he reported that he had been smoking marijuana three times a day for 24 years. One month prior to his visit he discontinued marijuana, stopped clonazepam, and started using mirtazapine with improvement in all aforementioned symptoms. During the same month, he relapsed and smoked marijuana once with a sudden return of all previously described symptoms on the night following its use. These symptoms then resolved and did not recur again until 9 months later, after another episode of weekend marijuana use. An in-lab polysomnogram revealed an apneahypopnea index 2.2 per hour, increased alpha intrusion throughout the study, and no epileptiform activity. No parasomnias were observed during rapid-eye-movement (REM) or non-REM sleep.

Conclusion: The etiology of EHS remains elusive. A review of the research has proposed five major theories and case reports suggest a complex etiology. Given the temporal correlation with marijuana use and symptom resolution with its cessation, we presume that EHS resulted from a complex interplay between neurons susceptible to cannabinoids and their derivatives.

1240

OBSTRUCTIVE SLEEP APNEA MANAGEMENT IN WEILL-MARCHESANI SYNDROME: A CASE REPORT

Monceaux Brittany MD, Smalley Megan MD, Okorie Ugorji MD, Roberts Edmond MD, Liendo Cesar MD, Asghar Sheila MD, Chernyshev Oleg MD PhD

Division of Sleep Medicine, Department of Neurology, Louisiana State University Health Science Center Shreveport, LA, USA

Introduction: Weill-Marchesani Syndrome (WMS) is a rare systemic genetic connective tissue disorder which usually presents with symptoms of short stature, limited joint movement, and eye problems such as glaucoma and microspherophakia. This genetic condition is associated with fibrous tissue hyperplasia. WMS is inherited as autosomal dominant or autosomal recessive patterns in families leading to a variability in presenting phenotype. Few papers have been written on airway management during anesthesia but as far as we know, this is the first case report on obstructive sleep apnea management in a patient with WMS.

Report of Case: A 9 year old boy with a past medical history of Methylene THF Reductase deficiency, von Willebrand's Disease, seizure disorder, premature birth, developmental delays and Weill-Marchesani syndrome was referred to Sleep Medicine due to tonsillar hypertrophy (3+), snoring and witnessed apneas. Upon physical examination, patient had mid-facial hypoplasia, retropositioning of the mandible, high arched palate, Mallampti class IV, maxillary hypoplasia and mandibular hypoplasia. He had been evaluated by ENT which determined the patient to be too high risk due to his medical conditions for T&A. The patient had a polysomnogram in 2018 indicating OSA with an apnea-hypopnea index of 4.2 and a minimum oxygen saturation of 91%. After a CPAP titration study, the patient was started on Auto CPAP of 5-15 cmH2O and has shown improvement in symptoms based on subjective and objective compliance report. Patient has been able to tolerate PAP therapy well with 100% compliance greater than 4 hours per night.

Conclusion: This case is the first illustrating OSA in a patient with Weill-Marchesani Syndrome. In WMS, the causes of OSA are not only due to tonsillar hypertrophy, but multifactorial, including craniofacial abnormalities. Given the high risk of surgical complications in WMS patients, PAP therapy appears to be a reasonable option for OSA management.

1241

A CASE OF CHRONIC HYPOVENTILATION OF UNKNOWN ETIOLOGY WITH IMPROVEMENT FROM A WAKE PROMOTING AGENT

Cole Melissa, MD, Isaacs Thomas, MD, Patel Amee, DO Baylor College of Medicine; Texas Children's Hospital

Introduction: We present a patient with chronic insomnia, excessive daytime sleepiness, and sleep-related hypoventilation that improved with a wake promoting agent in conjunction with BPAP. Report of Case: A 15-year-old male with Cystic Fibrosis (single F508del mutation, positive sweat test, FEV1% of 98%) and Autism who presented with frequent headaches, chronic insomnia, and daytime sleepiness (PDSS of 22). Current medications included albuterol as needed. Sleep history was significant for restless leg symptoms. Labs revealed low serum ferritin. Sleep study two years prior showed an AHI of 3.4 and PMLD of 18.4. After completion of iron therapy and initiation of gabapentin, his chronic insomnia and serum ferritin improved. However, patient continued to have aggressive behavior, headaches, and snoring. Sleep study was repeated and revealed mild OSA with hypoventilation (AHI of 6.41, >28% of total sleep time with TCO2 above 50 mm Hg). Initial serum bicarbonate was 29 mmol/L and progressively increased to 34 mmol/L. Due to evidence of persistent chronic hypoventilation, patient was trialed on BPAP. Subsequent sleep study with BPAP showed an AHI of 0, but with worsening sleep related hypoventilation (TCO2 of 56mmHg, > 70% total sleep time with TCO2 above 50 mm Hg). Genetic, endocrine, and neurological work up for hypoventilation was negative. Due to persistent daytime sleepiness (PDSS of 24), MSLT was performed and showed evidence of hypersomnia. Modafinil was then initiated. With the combination of nocturnal BPAP use and Modafinil, daytime sleepiness improved (PDSS of 15) and he had normalization of CO2 and bicarbonate levels.

Conclusion: We present a patient with CF, with minimal lung disease, who demonstrated a negative work up for sleep related hypoventilation, in which dysregulation of control of breathing improved with the use of a wake promoting agent in conjunction with BPAP.

1242

AN ADULT WITH ROHHAD DISEASE

Ahmad Hamna¹, Gohar Ashraf¹

¹Department of Pulmonary, Critical Care and Sleep Medicine

Introduction: Rapid-onset obesity with hypothalamic dysfunction, hypoventilation and autonomic dysregulation (ROHHAD) is an extremely rare and fatal disease presenting in early childhood.

Report of Case: A 25-year-old male, seen in our adult sleep clinic to establish care at the age of 21. He was previously followed by pediatric sleep department. He was diagnosed with ROHHAD syndrome in early childhood after presenting at the age of 18 months. At the time, he was diagnosed with central hypoventilation with multiple endocrinopathies. He was noted to have mild mental retardation and behavioral problems. He was initially worked up for multiple genetic disorders and was eventually diagnosed with ROHHAD syndrome. As a pediatric patient, he had a CPAP titration study that was unsuccessful with persistent central apneas. Eventually, he was treated with auto-SV (with minimum and maximum pressure support of 1 and +15 respectively, an EPAP max of +20, and EPAP min of +5). He has since been doing well with ASV. He also had insomnia and hypersomnia and is being treated with Modafinil for excessive daytime sleepiness and Zolpidem for the insomnia.

Conclusion: The mortality related to cardiorespiratory arrest in ROHHAD disease has been estimated to be 40-50%. To our knowledge, case reports of adult patients with ROHHAD in literature, are few and far between.

1243

A GUINNESS BOOK CHALLENGE IN UPPER AIRWAY STIMULATION ACTIVATION

Zakhary Nardine DO, and Strohl Kingman MD

Case Western Reserve University/University Hospitals Cleveland Medical Center

Introduction: Upper airway stimulation is a option for CPAP-intolerant patients. Device activation is typically ~4 weeks after the implant procedure.

Report of Case: A 61yo male with severe OSA had an upper airway stimulation device placed by ENT. At that time, stimulation produced bilateral tongue protrusion. In the immediate post-operative period, after closure, a hematoma, at the inferior chest incision, was discovered and drained with cauterization of the bleeding vessel. Seven weeks after implant, patient reported to our sleep clinic for activation of the device; and at that time, there was no sensation or activation up to the maximum amplitude of 5mV. The device reported an acceptable respiratory waveform, and triggering on and off sets but without sensory outcomes. Changing of the electrode configuration with advanced settings had no effect. Impedance values were acceptable. Tongue movements were grossly intact. At 2 months, ENT evaluation found mild hypoglossal nerve neuropraxia. To assess for a device related issue, x-rays of the neck and chest were performed and showed proper placement of the device. At 3.5 months, neuropraxia had resolved but device activation was unsuccessful, with no sensory or motor activation to 5mV stimulation. Plans were made for a procedure during which the lead electrode or implantable pulse generator would be assessed or replaced. At 4 months after implantation, in a multidisciplinary appointment with Sleep, ENT and the device representative, with a 3 electrode negative pole and the generator as the + pole, at 2.3mV, the device was activated. At the present time, the patient is exploring higher and lower mV settings and a PSG titration is scheduled.

Conclusion: This is the longest recorded duration (3.5+ months) of unsuccessful post-operative activation; and it occurred ~ 2 months after clinical signs of hypoglossal nerve neuropraxia had resolved.

1244

A CURIOUS CASE OF ASV FAILURE

*Chaudhary Nishant, MD¹, Ayache Mirna, MD², Carter John MD*³ ¹CWRU – UH Hospitals, Metro Health Medical Center, ²CWRU – Metro Health Medical Center, ³CWRU – Metro Health Medical Center

Introduction: Positive airway pressure-induced upper airway obstruction has been reported with the treatment of obstructive sleep apnea (OSA) using continuous positive airway pressure (CPAP) along with an oronasal interface. Here we describe a case of persistent treatment emergent central sleep apnea (TECSA) inadequately treated with adaptive servo ventilation (ASV), with an airflow pattern suggestive of ASV-induced upper airway obstruction.

Report of Case: A 32-year-old male, with severe OSA (apnea hypopnea index: 52.4) and no other significant past medical history, was treated with CPAP and required higher pressures during titration sleep studies to alleviate obstructive events, despite a Mallampati Class II airway and a normal body mass index. Drug-Induced Sleep Endoscopy (DISE) showed a complete velopharynx and oropharynx anterior posterior (AP) collapse, long soft palate, which improved with neck extension. CPAP therapy, however, did not result in any symptomatic benefit and compliance reports revealed high residual AHI and persistent TECSA. He underwent an ASV titration sleep study up to a final setting of expiratory positive airway pressure 9 cm H2O, pressure support 6-15 cm H2O (auto-rate), with a full-face mask due to high oral leak associated with the nasal interface. The ASV device detected central apneas and provided mandatory breaths, but did not capture the thorax or abdomen, despite normal mask pressure tracings. Several such apneas occurred, with significant oxyhemoglobin desaturation.

Conclusion: We postulate that the ASV failure to correct central sleep apnea as evidenced by the absence of thoracoabdominal inspiratory effort, occurred due to ASV-induced upper airway obstruction. Further treatment options for this ASV phenomenon are to pursue an ASV-assisted DISE and determine the effectiveness of adjunctive therapy including neck extension, nasal mask with a mouth closing device and a mandibular assist device.

1245

CHEYNE-STOKES RESPIRATION AND CIRCULATION TIME—A CASE TO ADD TO THE MYSTERY

Bhura Sajeer, MD^a, Rupani Nawaz, MD^a, Massoud Mona, MD^a. Chowdhuri Susmita, MD, MS^{b,c}, Shamim-Uzzaman Q. Afifa, MD, MS^{a,d}

^aUniversity of Michigan, Ann Arbor, MI, ^bJohn D. Dingell VA Medical Center, Detroit, MI, ^cWayne State University, Detroit, MI, ^dVA Ann Arbor Healthcare System, Ann Arbor, MI

Introduction: Lung-to-finger circulation time (LFCT), the time taken for the circulation to reach the fingertips from the lungs, has been shown to correlate inversely with cardiac function (Hosokawa et al. 2015). LFCT can be measured on PSG as the time from the start of rebreathing after a central respiratory event to the nadir of the oxygen desaturation on the SpO2 signal. Patients with congestive heart failure (CHF) have been found to have increased LFCT, with even longer LFCT in patients with Cheyne-Stokes Respirations (CSR). We are reporting a case where LFCT increased throughout a single night by more than 40%.

Report of Case: A 66-year-old diabetic male with ischemic cardiomyopathy (LVEF 25%), CVA with residual L-sided paresthesias, CKD-IV and hypertension was diagnosed with severe CSA and OSA (AHI 31.5, CAI 13.9, OAI 0.0, MAI 0.2, hypopnea index of 17.4, minimum SpO2 of 80%; supine AHI 56.1). CSR was not mentioned on this diagnostic study. On his titration study, CPAP and BPAP failed to treat CSA, but ASV effectively treated his sleepdisordered breathing; however, he could not be started on ASV due to HFrEF. On a repeat titration after ICD implantation, CSR was prominent in the supine position, on both CPAP and BPAP S/T therapy. Cycle length progressively increased from 40 seconds at

C. Case Reports

the beginning to 57 seconds at the end of the recording, an increase of 42% throughout the night. Circulation time also increased from 31 seconds to over 40 seconds. Scorable central respiratory events resolved but periodicity persisted.

Conclusion: To our knowledge, this is the first case of CSR with such progressive prolongation in cycle length during a single night. It again raises the question of the role of BPAP S/T in CSR with heart failure, and clarification of the potential use of supplemental oxygen in such situations.

1246

RESOLUTION OF CYCLICAL VENTRICULAR ECTOPY WITH ASV THERAPY IN A PATIENT WITH CHEYNE-STOKES RESPIRATIONS

Rupani Nawaz, MD, Massoud Mona, MD, Bhura Sajeer, MBBS, Kaplish Neeraj, MD

University of Michigan, Ann Arbor, MI

Introduction: Nocturnal cardiac arrhythmias, ranging from ventricular ectopy to heart blocks, have been commonly reported in patients with obstructive sleep apnea syndrome (OSAS). Potential mechanisms for these rhythm disturbances include OSA-associated hypoxemia, arousal trigger increased sympathetic activity and alterations in intrathoracic pressures leading to cardiac mechanical structural changes. A beneficial effect of CPAP treatment on rhythm abnormalities in patients with obstructive sleep apnea has also been demonstrated. However, the relationship of cardiac arrhythmias and central sleep apnea is not well established.

Report of Case: We report an 82-year-old male with CAD and Atrial fibrillation s/p PPM who presented for management of his sleep disordered breathing (SDB). Upon review, his original sleep studies performed at an outside facility revealed obstructive sleep apnea and central sleep apnea with Cheyne-Stokes Respirations. The patient presented to us on treatment with an auto-adjusting PAP (APAP) of 7-15 cmH2O with an average delivered pressure of 12 cmH2O. A re-titration study was recommended and demonstrated persistent central sleep apnea with Cheyne-Stokes breathing despite treatment with CPAP 12-18 cmH2O. During this time, EKG monitoring revealed an atrial paced rhythm with frequent premature ventricular beats (PVBs) which occurred in a cyclical pattern. After initiation of Adaptive Servo-Ventilation (ASV), periodic breathing was well controlled and cyclical ventricular ectopy had completely resolved.

Conclusion: Although CPAP therapy has been shown to improve nocturnal arrhythmias in patients with obstructive sleep apnea, the same relationship, to our knowledge, has not been reported in patients with central sleep apnea and Cheyne-Stokes breathing. This case demonstrates the improvement of cyclical ventricular ectopy with ASV therapy.

1247

RARE HYPOVENTILATION SYNDROME IDENTIFIED IN OBESE ADOLESCENT

Dr. Minor Courtney, MD

Children's Hospital of Colorado, University of Colorado Anschutz Medical Campus

Introduction: With the obesity epidemic persisting in the United States today, it is no surprise that morbidly obese patients with poor sleep are often linked to the diagnoses of obstructive sleep apnea and/or obesity hypoventilation syndrome before testing is even completed. It is important to spread awareness among

pediatric sleep providers of the rare hypoventilation syndromes for which most physicians likely have never seen a case. Missed diagnoses may lead to not only increased morbidity and mortality in patients, but under-reporting of cases will further delay crucial research needed to identify the precise pathophysiology that is currently unknown.

Report of Case: Authors encountered a 16 year-old male who had re-presented to pediatric sleep clinic after a two-year period of being lost to follow-up. The patient's past medical history included Angelman's syndrome, idiopathic central adrenal insufficiency, autonomic instability, seizure disorder, aggressive conduct disorder, morbid obesity, and hypoventilation without discrete obstructive sleep apnea as diagnosed on sleep study at age 10. Central hypoventilation was designated as idiopathic. The family had now returned to care due to concern for worsening episodes of bradypnea, bradycardia, and cyanosis requiring aggressive tactile stimulation. Review of records over the interim revealed multiple ER visits for stress dose steroids, as well as hospitalizations for hypothermia, bradycardia, and seizures. Consideration was taken that his multiple diagnoses were consistent with the unified diagnosis of Rapid-onset obesity with hypothalamic dysfunction, hypoventilation, and autonomic dysregulation (ROHHAD), and his hypoventilation thus could be accurately characterized as a syndrome of central hypoventilation rather than idiopathic or related to obesity. In addition, his progressive aggression and severe developmental delay could more accurately be attributed to chronic intermittent hypoxia, thus prompting more urgent need for non-invasive ventilation.

Conclusion: With approximately 100 reported cases, ROHHAD is a rare, clinically distinct entity from other etiologies of hypoventilation. Improved awareness of the diagnosis would hopefully lead to earlier diagnosis and improved anticipatory guidance able to be provided to the family.

1248

HYPERSOMNIA WITH PSEUDO-CATAPLEXY ASSOCIATED WITH SCHIZOAFFECTIVE DISORDER: A CASE REPORT

Khogeer Feras, MD¹, Sachdeva Alok, MD¹ ¹Michael S. Aldrich Sleep Disorders Center, Department of Neurology, University of Michigan, Ann Arbor, Michigan

Introduction: Psychiatric illness is an important potential cause of excessive daytime sleepiness (EDS), hypersomnia, and narcolepsy-like symptoms. We report a case of hypersomnia and pseudo-cataplexy associated with schizoaffective disorder.

Report of Case: A 28-year-old man with obstructive sleep apnea (OSA) and schizoaffective disorder presented to our sleep medicine clinic complaining of EDS, cataplexy-like attacks, sleep paralysis, depressed mood, and dream intrusion. Questions about cataplexy often provoked brief episodes of speech arrest, eye closure, and neck flexion lasting a few seconds.

An MSLT with CPAP showed a mean sleep latency of 3 minutes and 3 sleep onset REM periods (SOREMPs); however, actigraphy showed that the SOREMPs occurred during the patient's habitual sleep period and the patient discontinued a REM-suppressing medication within a few days of the study. The HLA-DQB1*0602 allele was absent.

Following circadian adjustment, a repeat MSLT with CPAP showed zero sleep and no SOREMPs. The preceding polysomnogram showed a total sleep time (TST) of 7.8 hours, no epileptiform activity, adequately-treated OSA, and a REM latency

of 53 minutes. Prior to these studies, two weeks of sleep diary and actigraphy showed a TST greater than 10 hours. Ultimately, treatment of the patient's psychiatric disorder resulted in a reduction of his EDS and TST.

Conclusion: Patients with psychiatric illness can present with hypersomnia, EDS, and narcolepsy-like symptoms such as pseudocataplexy. Misdiagnosing such a patient with narcolepsy can lead to treatments capable of worsening the primary underlying psychiatric disturbance. MSLT results must be interpreted in the context of a patient's clinical history and sleep schedule; if there is concern for a false positive result, repeat testing should be considered.

1249

REVERSE PLATYPNOEA-ORTHODEOXIA SYNDROME: A RARE CAUSE OF HYPOXEMIA DURING IN LAB POLYSOMNOGRAPHY

Sessums Mary T., M.D.¹, Guzman Maria P., M.D.¹, Dredla Brynn K., M.D.²

¹Mayo Clinic, Jacksonville, FL USA, ²Neurology and Sleep Medicine, Mayo Clinic, Jacksonville, FL, USA

Introduction: Sleep related hypoxemia carries a broad differential diagnosis. Right-to-left shunting is a known cause of hypoxemia that is not correctable with supplemental oxygen. A patent foramen ovale (PFO) is an intra-cardiac shunt that results in hypoxemia and in rare instances can lead to platypnea-orthodeoxia syndrome (POS). POS is characterized by dyspnea and hypoxemia in the upright position, which improves when supine. We present a case of nocturnal hypoxemia and PFO with a unique clinical presentation consistent with reverse POS discovered on PSG.

Report of Case: 57-year-old male with Class I obesity and a remote diagnosis of mild sleep-disordered breathing not treated with CPAP was referred to Sleep Medicine for excessive daytime sleepiness, snoring, and witnessed apneas. Polysomnography revealed basal oxygen saturation of ~88% during sleep. There was no evidence of apnea or hypopnea. Hypoxemia was not correctable with supplemental oxygen, suggestive of shunt physiology. Oxygen saturation was normal during upright exercise. Pulmonary hypertension was ruled out. Transesophageal echocardiogram revealed PFO with right-to-left shunt. Overall presentation was consistent with reverse POS secondary to PFO, which will be treated with percutaneous trans-catheter closure.

Conclusion: This is a rare case of reverse POS secondary to PFO, with shunt physiology initially suspected during Sleep Medicine evaluation based on PSG. Reverse POS is characterized by dyspnea and hypoxemia while supine, as opposed to upright as in classic POS. The precise pathophysiology of reverse POS is unclear. This case emphasizes the need to consider reverse POS in patients with supine hypoxemia refractory to oxygen therapy. Such findings on PSG should prompt further workup for causes of right-to-left shunt. This diagnosis should not be overlooked, as the underlying abnormality is often correctable. In this patient, we expect hypoxemia to resolve with percutaneous trans-catheter closure of PFO.

1250

AGRYPNIA EXCITATA IN A PATIENT WITH PARANEOPLASTIC AUTOIMMUNE ENCEPHALITIS

Sanchez, JL¹, Saeed, S¹, Battistini, H.¹ ¹Department of Sleep Medicine - Medical College of Wisconsin

Introduction: Agrypnia Excitata (AE) is a syndrome characterized by loss of sleep with permanent motor and autonomic hyper activation. This case describes this peculiar syndrome in a patient with paraneoplastic autoimmune encephalitis.

Report of Case: DG is a 35 yr old male with a history of anti-Ma2 limbic encephalitis secondary to cystic teratoma of the left testis diagnosed 6 months prior to presenting in Sleep Clinic. His parents described significant sleep disturbances including short sleep and wake periods throughout the day and night with no apparent pattern, acting out dreams, motor activity during sleep including pulling at his clothes or using his hands to manipulate invisible objects. Additionally they described low-grade fevers. and severe hyperphagia. Polysomnogram showed absence of slow-wave sleep and what appeared to be an admixture of stage 1 non-rapid eye movement (NREM) with rapid-eye movement (REM) sleep. Multiple sleep-latency testing (MSLT) demonstrated a mean sleep latency of 5.2 minutes and four sleep-onset REM periods (SOREMPs). Magnetic resonance imaging of the brain revealed persistent inflammation of the mesial temporal lobes and hippocampal region. Cerebral spinal fluid testing showed persistent anti-Ma2 antibodies. Based on this clinical presentation we made a diagnosis of Agrypnia Excitata.

Conclusion: Agrypnia Excitata is a syndrome characterized by loss of the normal sleep-wake rhythm. Sleep consists of the disappearance of spindle-delta activities, and persistent stage 1 NREM sleep mixed with recurrent episodes of REM sleep. The second hallmark of AE is persistent motor and autonomic hyperactivity observed during wake and sleep. AE has been described in three distinct clinical syndromes: Morvan Syndrome (autoimmune encephalitis), Fatal Familial Insomnia, and Delirium tremens. The pathogenesis of AE consists of intra-limbic disconnection releasing the hypothalamus and brainstem reticular formation from cortico-limbic inhibitory control. In autoimmune encephalitis, antibodies that act on voltage-gated potassium channels within the limbic system have been implicated in the pathophysiology.

1251

MANAGEMENT CONSIDERATIONS IN AN ADOLESCENT WITH KLEINE-LEVIN SYNDROME

George Alisha, MD¹, Chopra Nikki, MD¹, Afolabi-Brown Olufunke, MBBS²

¹Department of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, ²Division of Pulmonary Medicine, Children's Hospital of Philadelphia, Philadelphia, Pennsylvania

Introduction: Kleine-Levin syndrome (KLS) is a central disorder of hypersomnolence characterized by recurrent episodes of excessive sleepiness and sleep duration, along with varying degrees of cognitive dysfunction, altered perception, disinhibited behaviors, and eating disorders. Individuals with KLS are reported to be asymptomatic between episodes. KLS has a male preponderance with symptom onset typically during adolescence. A genetic basis has been suggested due to clustering of cases in certain ethnic populations and families. Purported triggers include viral infections, physical exertion, head trauma, and sleep deprivation.

Report of Case: A 15-year old male developed sudden-onset hypersomnolence with sleep duration of 20-23 hours per day. While awake, he had altered mentation and personality changes; parents reported aggression, hypersexual behaviors, hyperphagia, and derealization. During this 2-week episode, he underwent an extensive, yet unrevealing work-up and eventually experienced symptom resolution. Over 16 months, he has had 5 discrete episodes of hypersomnolence with abnormal wake behaviors, all preceded by viral illnesses and/or significant physical exertion and felt to be

consistent with KLS. He has reported ongoing, albeit less severe, excessive daytime sleepiness between episodes. Polysomnography and multiple sleep latency testing, both performed while asymptomatic, were largely unremarkable. As episodes have decreased in duration and intensity over time, trials of lithium and stimulant therapy have been deferred per family preference, though the patient was started on a short course of azithromycin during his last episode for anti-inflammatory properties. Notably, the patient has developed significant anxiety due to fear of recurrence and is now engaged in therapy.

Conclusion: KLS is a rare disorder that tends to have shorter and less severe manifestations over time. To date, very few systematic studies have been done to evaluate potential treatments, such as lithium and macrolide therapy. Avoidance of triggers and adequate sleep are encouraged, and mental health support should also be considered.

1252

RAPID-ONSET OBESITY WITH HYPOTHALAMIC DYSFUNCTION, HYPOVENTILATION, AND AUTONOMIC DYSREGULATION (ROHHAD) SYNDROME IN A PATIENT ON STIMULANTS AND NORMAL BMI AT THE TIME OF DIAGNOSIS: A CASE REPORT AND LITERATURE REVIEW.

McGhee Vera, MD^{1,2}, *Viteri Ernesto, MD*^{1,2}, *Schoumacher Robert, MD*², *Relia Sachin, MD*², *Harris Atia, MD*²

¹University of Tennessee Health Science Center, ²LeBonheur Children's Hospital

Introduction: ROHHAD is a rare and complex pediatric disorder presenting in early childhood with rapid onset obesity, followed by central hypoventilation, hypothalamic dysfunction, endocrine disorders, and neurobehavioral disorders.

Report of Case: We present a unique case of a 6-year-old girl diagnosed with ROHHAD in the setting of normal Body Mass Index (BMI). Patient had a history of food-seeking behavior, hyperphagia, and rapid onset obesity at the age of 4 years old with subsequent development of autonomic dysregulation, hypoventilation, and neurobehavioral disorders. At the time of evaluation in Sleep Medicine Clinic for snoring and disrupted sleep the patient had been treated for hyperactivity with several appetite-suppressing stimulant medications leading to weight loss and causing her to have a normal BMI. The original polysomnogram demonstrated severe Obstructive Sleep Apnea (OSA) with obstructive Apnea-Hypopnea Index (AHI) of 11.2/hour and was concerning for hypoventilation based on the end-tidal CO2 values and morning blood gas. Despite improvement in OSA with CPAP therapy, the patient's hypercapnia persisted. The overall clinical presentation resulted in extensive multispecialty workup, which ultimately led to the diagnosis of ROHHAD syndrome despite the patient's normal BMI.

Conclusion: ROHHAD syndrome is a rare and complex pediatric disorder requiring multi-disciplinary approach, with early diagnosis and intervention being essential for management of the condition. Hypothalamic obesity is one of the diagnostic hallmarks of ROHHAD. However, about half of patients with ROHHAD develop neurobehavioral disorders, which might require treatment with stimulant medications. Literature review revealed several case reports addressing the effect of stimulant medications on hypothalamic obesity, however none focusing on patients with ROHHAD syndrome. This case raises questions about the role of appetite-suppressing medications in the management of obesity in patients with ROHHAD. It also suggests possible need for further specific

workup in patients on stimulants with hypoventilation in the absence of obesity.

1253

MULTIPLE SLEEP ONSET REM EPISODES IN MIDDLE AGE WOMAN WITH EXCESSIVE DAYTIME SLEEPINESS – IS THIS AUTOMATICALLY ASSUMED NARCOLEPSY?

Patel Kamal, MD and Lang Bianca J., MD

Dartmouth-Hitchcock Medical Center, Department of Sleep Medicine

Introduction: Presence of sleep onset REM episodes often raises concerns of narcolepsy. However other conditions have shown to have presence of sleep on REM episodes which include but not limited to obstructive sleep apnea, sleep wake schedule disturbance, alcoholism, neurodegenerative disorders, depression and anxiety

Report of Case: Here we present a case of 30 year old female with history of asthma, patent foraman ovale, migraine headache, and anxiety who presented with daytime sleepiness, falling asleep while at work, occasional scheduled naps, non-restorative sleep, sleep paralysis, and hypnopompic hallucination. Pertinent physical exam included; mallampati score of 4/4, retrognathia, high arched hard palate, crowded posterior oropharynx. She had a score of 16 on Epworth sleepiness scale. Patient previously had multiple sleep latency test at outside facility which revealed 4/5 SOREM, with mean sleep onset latency of 11.5 minutes. She however was diagnosed with narcolepsy and tried on modafinil which she failed to tolerate. She was tried on sertraline as well which was discontinued due to lack of benefit. She had repeat multiple sleep latency test work up which revealed 2/5 SOREM, with mean sleep onset latency was 13.1 minutes. Her overnight polysomnogram prior to repeat MSLT showed SOREM with sleep onset latency of 10 minutes. Actigraphy showed consistent sleep pattern overall with sufficient sleep time but was taking hydroxyzine and herbal medication. Patient did not meet criteria for hypersomnolence disorder and sleep disordered breathing.

Conclusion: There is possibility her medication may have played pivotal role with her daytime symptoms. We also emphasize SOREMs can be present in other disorders such as anxiety in this case and not solely in narcolepsy

1254

SLEEP RELATED HALLUCINATIONS AS PRESENTING SYMPTOM OF DEMENTIA WITH LEWY BODIES: A CASE REPORT

Mallapareddi Arun Nag Santhosh, MD, and Judd Brooke, MD Department of Sleep Medicine, Dartmouth-Hitchcock Medical Center

Introduction: Visual hallucinations are one of the prominent symptoms of Dementia with Lewy Bodies (DLB). These hallucinations are often vivid and bothersome to patients. There is limited literature regarding patients with DLB presenting with hypnopompic/ hypnagogic hallucinations as the initial chief complaint. We report a case of a 77 year old female patient presenting to sleep medicine clinic with history of newer onset sleep-related hallucinations as well as concerns for sleep-disordered breathing, but was subsequently diagnosed with DLB.

Report of Case: 77 year old female patient initially presented to PCP with a one year history of disturbing hallucinations while falling asleep. Additionally, patient had excessive daytime sleepiness along with family history significant for obstructive sleep apnea (OSA).

Given the concerns of parasomnias and OSA, patient was referred to Sleep Medicine clinic. On presentation, patient continued to have hypnogogic/hypnopompic hallucinations along with symptoms concerning for OSA. An in-lab polysomnogram (PSG) was performed, which demonstrated evidence of a moderate to severe degree of OSA. Patient reported hypnopompic hallucination on the night of PSG. CPAP therapy was initiated. Despite treatment, her sleep-related hallucinations continued. She also began to have daytime hallucinations as well as cognitive impairment. Due to progression of symptoms despite treatment of OSA, neurology consultation was recommended. Per neurology evaluation, patient received PET CT metabolites which demonstrated changes consistent with DLB.

Conclusion: DLB should be considered as one of the differential diagnosis in patients presenting with hypnagogic/hypnopompic hallucinations especially if the patient has other signs of dementia.

1255

PREVIOUSLY UNKNOWN VENTRICULAR ARRHYTHMIA IN A GLOBALLY DELAYED PEDIATRIC PATIENT WITH TUBEROUS SCLEROSIS

Madhavarapu Sumith, DO, Casturi Lata, MS, CCSH, RPSGT, RST, Malhotra Sonal, MD, MPH, FAAP

Baylor College of Medicine, Texas Children's Hospital, Houston, Texas

Introduction: We present a case of previously unknown ventricular ectopy in a pediatric patient who is globally delayed. As the patient is non-verbal, it is possible the patient may have ultimately experienced heart failure or a lethal arrhythmia were it not for the polysomnogram (PSG).

Report of Case: A four year old female with Tuberous Sclerosis (TS) was referred to our pediatric sleep center for snoring. Her past medical history includes intracardiac tumors, daily seizures, and global developmental delay. Initial PSG showed moderate obstructive sleep apnea defined by an obstructive Apnea-Hypopnea Index of 6.89 and no cardiac arrhythmias. Positive airway pressure titration study was performed one vear later. Premature ventricular contractions were noticed during the setup while patient was awake. Intermittent couplets and triplets occurred during sleep as well as a prolonged run of bigeminy that initiated during a period of wake after sleep onset and persisted into non-rapid eye movement sleep. She was escorted to the emergency room where a 12-lead electrocardiogram (EKG) showed sinus rhythm with non-specific interventricular conduction delay and right ventricular hypertrophy. She was discharged with a Holter monitor and subsequent analyses was concerning for frequent ventricular couplets and non-sustained runs of ventricular tachycardia. She required admission to initiate anti-arrhythmic therapy. Imaging revealed stable intracardiac tumors, but revealed scarring within sites of intramyocardial lesions. She failed different anti-arrhythmic agents before settling with amiodarone.

Conclusion: Sleep disordered breathing (SDB) can cause or worsen cardiac arrhythmias. This case highlights the importance of routine surveillance in patients with both known cardiac disease who have or are at risk of having significant cardiac arrhythmias and suspected SDB. Additionally, current guidelines for TS patients recommend EKG once every 3 to 5 years. This case may also highlight the importance of increased cardiac surveillance in this population group.

1256

NARCOLEPSY TYPE 2 IN A PEDIATRIC PATIENT WITH AUTO-IMMUNE DISEASE

Madhavarapu Sumith, DO, Patel Amee, DO

Baylor College of Medicine, Texas Children's Hospital, Houston, Texas

Introduction: We present a case of Narcolepsy Type 2 in the setting of concomitant auto-immune disease.

Report of Case: At 9 years of age, an African-American female was referred to a sleep center for fatigue, excessive daytime sleepiness (EDS), and snoring. Polysomnogram (PSG) at an outside facility recorded a total sleep time (TST) of 408 minutes, 92% sleep efficiency (SE), sleep onset latency (SOL) 22 minutes, REM latency 70 minutes, and apnea-hypopnea index (AHI) of 0.3. Multiple Sleep Latency Test (MSLT) was not done at that time. Due to her daytime sleepiness, methylphenidate was initiated empirically twice daily with improvement in daytime sleepiness and sleep maintenance. Over time, methylphenidate was steadily increased due to increasing daytime sleepiness. Between 14 to 15 years of age, she developed increased fatigue, skin changes, arthralgias, myalgias, and presented to our hospital in respiratory distress secondary to severe pulmonary hypertension. Clinical exam and workup indicated scleroderma. Methylphenidate was discontinued due to pulmonary hypertension. At 17 years of age (after receiving treatment for scleroderma and pulmonary hypertension), she had an overnight PSG followed by MSLT. PSG recorded TST 309 minutes, 67% SE, SOL 1.5 minutes, REM latency 48 minutes, AHI 0.2. The MSLT recorded a mean SOL of 2.4 minutes with 2 out of 4 sleep onset REM periods - diagnostic of narcolepsy. She does not endorse cataplexy, hallucinations or sleep paralysis.

Conclusion: While the link between Narcolepsy Type 1 and an auto-mediated process is more supported given consistent human leukocyte antigen findings, the link between Narcolepsy Type 2 and auto-immune mediated process is less clear. Patients with auto-immune disease may have symptoms of narcolepsy; therefore, they may benefit from screening for EDS.

1257

LOW NOCTURNAL SPO2 AND DAYTIME SLEEPINESS IN A CHILD

Pi MY, Han F

Department of Pulmonary Medicine, Peking University People's Hospital, Beijing, 100044. China.

Introduction: Patients with nocturnal oximetry shows SpO2 during sleep of $\leq 90\%$ in children for ≥ 5 minutes and sleep related hypoventilation is not documented could be diagnosed as sleep related hypoxemia (ICSD-3). However, known physiological causes should be indicated.

Report of Case: A 5-year-old Chinese boy was referred to sleep lab for daytime sleepiness for three months. Type I narcolepsy was confirmed.

Nocturnal PSG indicated a continuous low SpO2 baseline around 82%, with a nocturnal mean SpO2 of 83%, but no respiratory events. Voluntary hyperventilation made SpO2 increase from 83% to 95%, and hypoventilation syndrome was considered. PtcCO2 during PSG maintained 40mmHg, and no sleep related elevation observed. BPAP treatment had no effect on both SpO2. No dyspnea was complained. When he was 12 years old, follow-up PSG and MSLT indicated narcolepsy remained. During the past 7 years, no comorbidities occurred. Hb 104g/L, RBC 3.53×1012/L,

C. Case Reports

Hct 31%. ABG showed SaO2 of 97%, PaO2 98mmHg and PaCO2 40mmHg. Inconsistency between SpO2 and SaO2 reminds the existence of abnormal hemoglobin.

A positive isopropanol precipitation test and further hemoglobin electrophoresis showed abnormal Hb and HbA were 37.6% (normal 0%) and 58.4% (normal 94.3–98.5%), respectively, but HPLC did not identify. Spectrophotometric analysis indicated that the absorption spectra were significantly different at 450nm~540nm and 600nm~1000nm. Hemoglobin structure analysis found the abnormal site. Gene sequencing identified a novel HBB: c.212 C>A mutation, resulting in amino acid 71 to change from alanine(Ala) to aspartic(Asp) acid as a de novo mutation. A diagnosis of Hemoglobin Seattle [β 71:Ala \rightarrow Asp ; HBB:c.212C>A] was confirmed.

Conclusion: Pediatric sleep medicine has a rapid development. Before a diagnosis of sleep related hypoxemia is made, in rare situation, abnormal hemoglobin should be considered.

1258

NO LAUGHING MATTER - LAUGHTER INDUCED SYNCOPE (GELASTIC SYNCOPE) MIMICKING CATAPLEXY

Belani Dinesh, MD¹, Simon Edwin, MD²

¹Pulmonary Medicine Fellow, Rosalind Franklin University of Medicine and Science, North Chicago, IL, ²Sleep Medicine Attending, Rosalind Franklin University of Medicine and Science, North Chicago, IL

Introduction: Laughter is a common emotion and may rarely be a manifestation of neurological illnesses. It has been associated with cataplexy as well. Cataplexy is usually triggered by strong emotions. Gelastic syncope is an uncommon phenomenon which may be mistaken for cataplexy. We summarize 3 cases referred to the Sleep Medicine clinic for evaluation for Narcolepsy.

Report of Case: 55 yo male comes with 2 episodes of blacking out and falling down relating to episodes of laughter in 3 months. Patient describes loss of consciousness and no episodes of freezing. Reported 15 years of snoring and witnessed apneas along with grinding his teeth while sleeping. Polysomnogram revealed Obstructive Sleep Apnea (OSA) with an AHI of 20.

60 yo male comes with episodes of loss of consciousness over the past 6 months, including sitting in a chair, laughing, urinating, washing dishes while standing, expressing strong emotions (father's funeral), etc. Also reports bugs crawling over his legs when trying to sleep, loud snoring and waking up choking while sleeping. Polysomnogram revealed OSA with an AHI of 20.

43 yo male comes 3 episodes of loss of consciousness, 2 of them related to laughing and the last one related to stretching his arms out. He passes out for 5-10 seconds at a time and a period of 20-30 seconds before passing out where he feels dizzy when he is unable to respond at this time, no post episode confusion. Positive on the Cataplexy Emotional Trigger Questionairre. Reported witnessed apneas, snoring and sleep talking. Polysomnogram revealed OSA, hence the Multiple Sleep Latency Testing ordered was not completed.

Conclusion: While the first two episodes point towards Gelastic Syncope based on symptoms, the third did warrant MSLT if there was no OSA on PSG. It is important to recognize gelastic syncope as an entity and differentiate it from cataplexy.

1259

PERIODIC NECK MYOCLONUS DURING SLEEP (PNMS) IS ASSOCIATED WITH UPPER AIRWAY RESISTANT SYNDROME, BUT RESOLVES WITH CPAP Alfi Majed A., MD¹, Avidan Alon Y., MD, MPH²

¹University of California, Los Angeles, Department of Sleep Medicine, Los Angeles, CA, ²University of California, Los Angeles, Department of Neurology, Los Angeles, CA

Introduction: Periodic neck myoclonus during Sleep (PNMS) is a movement disorder of sleep characterized by sudden myoclonic flexion or version of the head that manifest during REM and NREM sleep. While its finding has been attributed to a normal physiologic phenomenon, to the best of our knowledge, our case represents the first report of with PNMS attributed to sleep disordered breathing with resolution using CPAP

Report of Case: A 22 y/o male with no significant clinical history was referred for evaluation of snoring and excessive sleepiness. Nocturnal polysomnogram coupled with expanded EMG montage demonstrates evidence of upper airway resistance syndrome (UARS), characterized by frequents respiratory effort-related arousals (RERAS), primarily during REM sleep associated with arousals. The majority of these events resulted in sudden myoclonic movements of the neck and head that were associated with arousals and sleep fragmentation. PNMS manifested in the PSG as a flexion myoclonic motor artifact lasting 200-800 ms during REM sleep with an associated EEG arousal. The overall Respiratory Disturbance Index (RDI) was12/hr. The subsequent application of CPAP at a setting of 5-6 cm resolved these movements supporting this origin as a phenomenon of sleep-state instability.

Conclusion: While previous investigators have explained PNMS as an incidental finding or one common in patients with RBD, our case highlights a potential new mechanism for their appearance. This case helps shed more light on the origin of PNMS as a secondary phenomenon related to sleep state instability due to sleep disordered breathing given the temporal association with RERAS and dramatic resolution with CPAP therapy.

1260

HIGH TIDAL VOLUME NON-INVASIVE VENTILATION REQUIRED TO TREAT OBESITY HYPOVENTILATION

*Rezayat Talayeh DO, MPH*¹, *Beggs Abigail MD*¹, *Avidan Alon Y., MD, MPH*² & *Javaheri Shahrokh, MD*³

¹University of California, Los Angeles, Department of Pulmonary Critical Care and Sleep Medicine, Los Angeles, CA, ²UCLA Sleep Disorders Center, Department of Neurology, University of California, Los Angeles, Department of Neurology, Los Angeles, CA, ³Division of Pulmonary and Sleep Medicine, Bethesda North Hospital, Cincinnati, OH

Introduction: Current guidelines recommend CPAP or non-invasive ventilation with tidal volume (VT) <10ml/kg of ideal body weight (IBW) for the treatment of obesity hypoventilation. However, in select patients with significant obesity hypoventilation, this recommendation may not be sufficient to resolve nocturnal hypoventilation.

Report of Case: A 35 y/o male with hypertension and class III obesity (BMI 58 kg/m2) was referred for evaluation of acute respiratory failure with hypoxia and hypercapnia. ABG demonstrated daytime PCO2 of 71 mmHg. Patient reported sleep fragmentation, snoring, choke awakenings, poor concentration,

depression and sleep attacks. PSG demonstrated severe OSA, with an AHI of 154 events/hour, persistent hypoxia and hypercapnia with a SpO2 nadir of 50% and ET-CO2 of 83 mmHg during REM sleep. Respiratory events persisted with CPAP and bilevel, up to a setting of 25/16. Average volume assured pressure support (AVAPS) S/T titration study was performed and resolved sleep apnea at settings of IPAP 24-30, EPAP 4-15, VT 790 (10 mL/kg IBW), 0.5 LPM O2, rate 16. The patient reported having had the best sleep of his life at the end of this study and has since been started on treatment.

Conclusion: Treatment of OHS should be individualized and may require use of tidal volumes above 10ml/kg for effective treatment. We suggest that in super morbidly obese patients, with extremely noncompliant respiratory system, larger than recommended tidal volume is necessary to ventilate the patient and improve gas exchange. The sustained higher pressures achieved by AVAPS to impose the augmented tidal volume more effectively ameliorate OSA, by keeping the upper airway open. Higher pressures achieved also could elevate FRC, not only increasing oxygen stores, but also contributing to maintenance of open upper airway through its tethering effect. Further physiological studies are needed in super morbidly obese patients comparing low and high tidal volumes.

1261

THE LONG TERM EXHAUSTION OF A VIRAL INFECTION *Mulhern Kellen, DO*

George Washington University

Introduction: Narcolepsy is a rare condition affecting only 0.02% to 0.18% of United States and Euro-pean populations. Throughout the years, hypersomnolence in the form of narcolepsy or idiopathic hypersomnia has been identified as a post infectious syndrome in rare instances. In this case, we observe a child with excessive day-time sleepiness after an infectious rash.

Report of Case: A 4 year old male presented to clinic for evaluation of excessive daytime sleepiness for the past 10 months. At that time, he developed a full body rash that lasted for about 1 week, and was treated with diphenhydramine and prednisone. No imminent cause was identified. For the preced-ing weeks, he was taking frequent and longer naps, and complained of difficulty with keeping his head up and his eyes open.

He was evaluated for possible seizures via EEG with no seizure activity seen. Blood work was unremarkable, and a lumbar puncture showed highly elevated IgG for EBV, and low IgM for EBV, indicating a prior infection.

His excessive somnolence persistent, and he was referred to undergo actigraphy, poly-somnography (PSG), and a multiple sleep latency test (MSLT). Actigraphy demonstrated disrupt-ed nocturnal sleep, PSG showed an elevated periodic leg movement index of 8.6/hr, and MSLT showed reduced sleep onset latency and sleep onset REM on 2 of 4 naps.

During his physical exam, he had drooping eyelids and tongue protrusion, concerning for cataplectic facies, highly suspicious for Narcolepsy Type 1. Further confirmatory testing is pend-ing, including HLA subtype testing and hypocretin levels in CSF.

Conclusion: This case shows a strong association with a subacute EBV infection and the development of nar-colepsy. There are multiple cases involving the development of hypersomnolence conditions as a consequence of viral infections. Further cases would be beneficial to form additional associations.

1262

CASE SERIES ON THE USE OF VOLUME ASSURED PRESSURE SUPPORT (VAPS) IN PATIENTS WITH INTERSTITIAL LUNG DISEASE AND PROGRESSIVE HYPERCAPNIA

LeMaster William, MD, Jun Dale, MD, De Cruz Sharon, MD, Zeidler Michelle, MD, Saggar Rajan, MD UCLA School of Medicine, Los Angeles, CA, United States

Introduction: Many patients with interstitial lung disease (ILD) experience progressive respiratory failure. While various therapies are implemented for acute hypercapnic respiratory failure during inpatient ILD flares, there is little data regarding the management of chronic hypercapnia in ILD with nocturnal Volume Assured Pressure Support (VAPS). We present three patients who were prescribed nocturnal VAPS for their progressive hypercapnia as a bridge to lung transplantation.

Report of Case: Patient 1 is a 45-year-old woman with rheumatoid arthritis related ILD and progressive hypercapnia. Despite optimal therapy, her ILD resulted in an admission for hypercapnic and hypoxemic respiratory failure requiring treatment with BPAP, then transition to nocturnal VAPS on discharge. Dyspnea and pCO2 improved as an outpatient (Fig. 1). Patient 2 is a 70-year-old female with history of scleroderma associated ILD with severe PH and hypercapnia. Initiation of VAPS improved her pCO2 levels although she was readmitted after a few months of treatment for an ILD flare. Patient 3 is a 60-year-old patient with connective tissue disease related ILD who was admitted for respiratory failure due to pneumonia and was transitioned to BPAP for hypercapnic respiratory failure. Due to insurance issues she has been unable to obtain a home VAPS and her pCO2 remains elevated. A plot of each patient's pCO2 over time is in Figure 1.

Conclusion: In patients with severe lung disease, the normal decrease in tidal volumes that occurs with sleep can result in CO2 retention. Non-invasive ventilation (NIV) is well-studied in both stable obstructive lung disease and exacerbations but there is little data examining the utility of NIV to treat the chronic hypercapnia of ILD. In this case series, nocturnal VAPS stabilized or reduced PCO2 in patients with ILD and hypercapnia. Additional studies are needed to assess long term effects of VAPS in these patients.

1263

MANDIBULAR ADVANCEMENT DEVICE REFERRALS AND COMPLIANCE AT THE VA

Hsieh Caleb, MD MS^{1,2}, Ryden Armand, MD², Zeidler Michelle, MD^{1,2}

¹UCLA Department of Medicine, Department of Sleep Medicine. Los Angeles, CA, ²West Los Angeles VA Medical Center, Department of Sleep Medicine. Los Angeles, CA

Introduction: For patients with obstructive sleep apnea (OSA) who are either intolerant or otherwise non-compliant with positive airway pressure (PAP) therapy, the use of a mandibular advancement device (MAD) is a viable and minimally invasive alternative for select individuals. Many device variations exist, but the common mechanism of action involves airway augmentation via traction on the lower jaw provided by a dental appliance. Prior studies have identified factors associated with treatment success, however, actual clinical experiences fluctuate widely. To identify patients within the VA that might benefit most from MAD, we conducted a preliminary review of MAD referrals within a one-year period at the West Los Angeles VA (WLA VA).

C. Case Reports

Report of Case: This retrospective database review was performed to identify areas for improvement in MAD referrals within the West Los Angeles VA network. The need for informed consent was waived as part of reviews preparatory to research as outlined in paragraph 23c of the VHA Directive 1200.05. Our database was queried for all MAD dental referrals between January 1, 2017 and Dec 31, 2017. All patients that completed at least one dental visit were included, those that did not complete referrals were excluded. Patient charts were reviewed for demographic data, comorbidities, dentition quality, pre- and post- MAD sleep studies, prior failed therapies, duration of MAD usage, reasons for discontinuation, and final OSA treatment modality. Between January 1, 2017 and December 31, 2017, 246 patients were completed, 55 were discontinued for various reasons, and 7 were cancelled.

*** Preliminary data for first 84 patients***

Of 84 patients seen in dental clinic, 33 (39%) were deemed poor candidates either because of insufficient dentition or because of existing temporal-mandibular joint discomfort, 4 patients declined any intervention. Of 47 patients that received an MAD, 6 patients continued to use MAD alone, 20 switched to PAP or other modalities, and 21 were lost to follow up.

Conclusion: Pending more in depth analysis

1264

INNOVATION AND INGENUITY TO IMPROVE TREATMENT EFFICACY OF NON-INVASIVE POSITIVE PRESSURE VENTILATION

Nuzhny V.1 Kleerup E.C.2, Zeidler M. R.2

¹Eisenhower Health, Family Medicine Residency Program, Rancho Mirage, CA, United States ²David Geffen UCLA School of Medicine, Los Angeles, CA, United States

Introduction: Use of chronic non-invasive-positive-pressure ventilation (NIPPV) in individuals with neuromuscular disease allows for increased independence and mobility in this population. Optimal mask fit is imperative due to the chronic and extensive device use. Commonly cited side effects of improper mask fit include air leak, dermatitis, skin breakdown, nasal discomfort, and claustrophobia. Report of Case: This is a case report of a 46-year-old female with rigid spine muscular dystrophy leading to chronic hypercarbic respiratory failure secondary to neuromuscular weakness and thoracic cage abnormalities. The patient is dependent on continuous use of NIPPV with a PLV 100 device (Philips Respironics; discontinued model). The patient prefers this model because of its unique ventilation delivery mode, which allows her to pause breaths to speak. The patient developed a significant air leak with her nasal mask and was unable to be fitted properly with commercially available nasal masks. In order to minimize her air leak her husband used an innovative approach using 3D printing technology. He created a 3D print of her facial and nose features and then used this to print a 3D mask. Additionally he 3D printed silicone nose clips, that reinforced the seal on the outside of her nose. The patient had a significant decrease in her air leak and subjectively reported improved comfort with use of the 3D printed mask.

Conclusion: This represents a case where application of ingenuity and innovative technology improved treatment efficacy and compliance with NIPPV. The combination of 3D custom fit mask (currently available from a limited number of vendors), with custom fit nasal clips may possibly be applied to a wider category of patients with similar complaints of nasal discomfort and frequent air leaks.

1265

SEVERE CENTRAL AND OBSTRUCTIVE SLEEP APNEA IN TEENAGER WITH SEVERE OBESITY, TONSILLAR HYPER-TROPHY, AND MAXILLARY CONSTRUCTION Jaeger Marv-Alice MD

University of Oklahoma Health Sciences Center

Introduction: Obesity in children escalated in the past 50 years. For American children 2-19 years old, Obesi-ty(BMI ≥ 95 th%) increased from 5% in 1971-4 to 19%(13.7 million children)2015-16. Severe Obesity(BMI ≥ 120 th% or ≥ 35) is less common with prevalences of 1% 1971-4 to 6% in 2015-16(1). Obesity increases risk for physical and mental illness.

Sleep apnea risk factors include obesity, maxillary restriction(3), and adenotonsillar hypertro-phy(4).

Report of Case: 16 yo boy with snoring, gasping during sleep, witnessed apneas, mouth breathing, morning head-aches, EDS, and learning disability requiring an IEP. Past medical history of neonatal snoring, apneas, and reflux.

Physical exam revealed severe obesity(BMI 45.3), high arched/ narrow palate, Class II bite, large tongue, Mallampati IV, Grade 3-4 tonsils, CricoMental Space +1cm. Inattentive with mildly de-pressed affect. No cardiovascular, pulmonary or neurologic findings.

PSG: CAI 31.2, OAHI 23.8. Average O2 sat 97% with 11 minutes<88%. End-tidal CO2 average 50 during sleep and wake. 51% of total sleep time with ETCO2>50 mmHg.

CPAP titration: CAI 1.8, OAHI 10.4. Average O2 sat 96% with <1 minute<88%. Events improved with CPAP 14 cm H20 to OAHI 3.5 with >30 minutes of supine REM.

Conclusion: Severe Central Sleep Apnea with significant obstructive component associated with hypoxia and hypoventilation. With the diurnal hypoventilation, the likely etiology for central apneas is Obesity Hypoventilation Syndrome(5). The central apnea improved with CPAP. His management included CPAP therapy, ENT referral for adenotonsillectomy(5), bariatric referral, and further evaluation for learning/behavior concerns.

In retrospect, earlier diagnosis/intervention on behalf of this teenage boy with a history of neonatal snoring presenting now with Severe Obesity, tonsillar hypertrophy and maxillary constriction may have made a significant difference for his cognitive/ mental/physical health outcomes.

1266

PRIMARY CENTRAL APNEA AND LOSS OF MUSCLE ATONIA DURING REM

Hernandez M. Elizabeth C., M.D., Velamuri Kanta, M.D. Baylor College of Medicine, Houston, Texas, Michael E. DeBakey VA Medical Center, Houston, Texas.

Introduction: Central sleep apnea (CSA) syndrome is defined when five or more central apneas and/or hypopneas are present per hour of sleep, more than 50% of all respiratory events. CSA usually occur during NREM stage and rarely during REM. CSA is important to recognize because of complications ranging from frequent nighttime awakenings, sleepiness to adverse cardiovascular outcomes. We present a 40 year old female patient with rare CSA during REM sleep and dream enactment.

Report of Case: 40yo African American female with history of loud snoring, witnessed sleep apnea, and daytime fatigue. She reported nightmares, sleep talking, and acting out her dreams without injury. Epworth sleepiness score was 5 /24. Her past medical history

Case Reports from Clinical Trainees

is significant for depression and anxiety. She has no history of head trauma, no neurologic or cardiovascular disorders. Her medications include fluoxetine and,quetiapine. She denied substance use, narcotic use, or alcohol use.

Her level 1 sleep study showed predominantly REM-associated central sleep apneas which is rare. She also was observed to have loss of REM sleep muscle atonia suggestive of REM Behavior disorder.

Her sleep architecture was atypical with decreased N3 sleep stage. REM sleep duration was adequate. She was noted to have loss of REM muscle atonia based on AASM guidelins elevated chin EMG, excessive transient muscle activity, and witnessed movement during REM stage via video monitoring.

During the study, she had an apnea/hypopnea index (AHI) of 13.1 per hour of sleep, Central apneas were predominantly noted during REM stage, 10 per hour, comprised of 50% of her respiratory events. The minimum SpO2 value with CSA was 94%. She had normal sinus rhythm.

Her sleep was fragmented. A total arousals were 28.4/hour,and 7.9/hour were respiratory arousals, and the rest were spontaneous arousals.

An echocardiogram showed normal left ventricular ejection fraction of 55 to 60 %. Her room air arterial blood gas was normal with PaC02 of 37 mmHg. MRI of the brain/brainstem was ordered given her atypical REM sleep. She had no acute intracranial abnormalities. There is a non specific finding of a low lying cerebellar tonsils without evidence of Chiari I malformation.

Conclusion: Our patient has rare idiopathic central apnea in REM stage and is third case reported. She also has loss of muscle atonia during REM with dream enactment which is also rare in her age group. Injury precaution advised.

1267

SEVERE OBSTRUCTIVE SLEEP APNEA IN PATIENT WITH LARGE MULTINODULAR GOITER

Macias Maria, MD, Agrawal Ritwick, MD

Baylor College of Medicine, Michael E. DeBakey Veteran Affairs Medical Center – Houston, TX

Introduction: Obstructive sleep apnea (OSA) is characterized by upper airway narrowing or closure during sleep. Age and obesity are common contributors, but large thyroid goiters have also been shown to contribute to OSA.

Report of Case: We report a case of a 65 year-old-man (BMI 47 kg/ m2) who presents with dyspnea and intolerance to PAP therapy. He had a slowly progressive goiter which was first noticed at age 45. He declined thyroidectomy due to concern of complications. A recent CT reported markedly enlarged thyroid (right thyroid lobe 10.9 x 8.5 cm, left thyroid lobe 7.0 x 6.5 cm and thyroid isthmus 4.0 cm). It had extension into the superior mediastinum and circumferential encasement of the subglottic trachea with effacement involving the lateral walls. In past, he was non-tolerant with BPAP therapy due to high pressure settings (24/14 cm water). Multiple attempts to desensitize were not successful.

Most recent diagnostic polysomnography reported an apnea hypopnea index (AHI) of 35.2/ hour, oxygen nadir of 77%. Supplemental oxygen was titrated upward to 5 LPM due to persistent oxygen desaturations in the absence of obstructive events. In the PAP titration study, despite multiple efforts and patient's poor tolerance, the titration study was suboptimal. He was titrated to BPAP 15/11 and still had a residual AHI of 28.4/hour. Considering these findings thyroidectomy was again discussed which could potentially reduce OSA severity significantly. After long discussion, unfortunately the patient declined this recommendation. Other surgical options such as hypoglossal nerve stimulation was not technically feasible due to large goiter. Ultimately, he decided to remain on nightly supplemental oxygen.

Conclusion: Large multinodular goiters with retropharyngeal extension can worsen obstructive sleep apnea and pose unique diagnostic and therapeutic challenges. In this case, thyroidectomy may have led to improvement of degree of sleep disordered breathing.

1268

WHEN SCARS DON'T LET YOU SLEEP- UNUSUAL CASE OF RESTLESS LEGS SYNDROME SECONDARY TO KELOIDS

Agarwal Abhishek, Chopra Nikki, Rives-Sanchez Marisela, Bae Charles

Hospital of University of Pennsylvania

Introduction: Restless leg syndrome(RLS) is a common disorder which has been estimated to be present in 5 - 15% of the general population with 2.5 % requiring intervention. RLS can be primary or secondary. Secondary RLS usually develops later in life and is associated with another disorder like chronic kidney disease, iron deficiency, spinal cord disease, or varicose veins. Keloids have never been reported to be the cause of RLS.

Report of Case: 50 y.o female with hypertension and diabetes presented for evaluation of loud snoring and witnessed apneas. Prior to the visit she underwent a sleep study that showed she had moderate sleep apnea (AHI 15). She also reported having a painful sensation in her legs mainly at night affecting the skin on her calves. The painful sensation gave her an urge to move her legs. Moving or rubbing her legs helped relieve the painful sensation partially. She did not have these symptoms during the day. The symptoms started in the summer of 2018 after she started having keloids on her calves. Keloids followed the course of her varicose veins and the keloids progressed over the last year. Her RLS symptoms were less bothersome when the keloids were smaller. When she has taken tramadol and oxycodone in the past for other pains, she noticed that her painful RLS symptoms were completely eliminated. She was taking gabapentin 600 mg at bedtime for neuropathic pain which hasn't helped her RLS symptoms. On physical exam, major keloids were seen bilaterally on her calves. Her OSA with treated with CPAP and she was also asked to increase her gabapentin from 600 mg to 1200 mg for her restless legs.

Conclusion: This is one of the first reported cases of RLS secondary to keloids. Whether treatment of keloids would help relieve RLS symptoms is unknown.

1269

OFF-LABEL USE OF SOLRIAMFETOL IN IDIOPATHIC HYPERSOMNIA

Wong Tammy, MD, Memon Talha, MD¹

¹Riverside Community Hospital-University of California, Riverside School of Medicine

Introduction: Solriamfetol is a new daily dopamine and norepinephrine reuptake inhibitor indicated for improving daytime wakefulness in adults with obstructive sleep apnea (OSA) and narcolepsy. It was FDA-approved on 3/20/2019. In this report, we present a

C. Case Reports

patient with refractory idiopathic hypersomnia (IHS) who benefitted from off-label use of solriamfetol at a private sleep center.

Report of Case: A 37-year-old non-obese female previously diagnosed with IHS vs narcolepsy, presented in 2015 with excessive daytime sleepiness since her mid-teens. She also had a history of periodic limb movement (PLM) and anxiety and trialed clonazepam, which caused daytime hangover effects. At her previous office, she began sodium oxybate but had psychiatric side effects, and was thus started on methylphenidate. Modafinil was added due to build-up of tolerance. Three separate polysomnographies (PSG) over 9 years revealed similar findings of mild snoring without OSA, PLM with variable arousals. Multiple sleep latency testing (MSLT) showed very rapid sleep onset <4 min and no sleep-onset REM periods. The patient never had cataplexy but occasional hypnogogic hallucinations and sleep paralysis. All studies to-date suggested IHS, less likely narcolepsy without cataplexy, and at her initial visit, the patient trialed armodafinil instead of modafinil, and methylphenidate was weaned down. When seen again in 2017, the patient reported persistent daytime sleepiness on methylphenidate and modafinil (PSG-MSLT showed sleep latencies ~3.5 min on 5/5 nap opportunities), without cataplexy. When seen 9/2019, patient continued to have daytime sleepiness and fatigue. She was then weaned off methylphenidate and modafinil, and started on solriamfetol. Since then, she has been doing well solely on solriamfetol 75mg BID.

Conclusion: Off-label use of solriamfetol for IHS has been demonstrated to be effective in a treatment-resistant patient at a private sleep center. More data and further discussion in support of its off-label use are warranted for this patient population.

1270

WOBBLY KNEES: CATAPLEXY OR SEIZURE? A CASE REPORT.

Nadhim, A.¹, wong, J.¹, Gupta, D.

¹JFK Neuroscience institute, Edison, NJ, ²JFK Neuroscience institute, Edison, NJ

Introduction: Cataplexy (associated with narcolepsy) is difficult to differentiate from conditions such as seizure, syncope or TIA, but using validated clinical tools can help. We report a case that was mistakenly diagnosed as cataplexy, delaying diagnosis and treatment of his underlying seizures.

Report of Case: A 42 years old male presented with "freezing spells" described as spontaneous episodes of weakness in his knees. He may stagger and drop objects but had never fallen. He denies loss of consciousness. Post-ictally, he takes a moment to readjust his body and returns to baseline. There was no warning or aura before the episodes. These spells initially occurred 1-2 times/year in 2002, then progressed to 1/month by 2006 and then increased to 15 times/ day in Jan 2019, lasting 5-10 seconds at a time. He became hesitant to drive. He was evaluated by a pulmonologist/sleep specialist. PSG showed Mild OSA but MSLT wasn't suggestive of Narcolepsy. He was prescribed CPAP but stopped after 6 months since it didn't improve his symptoms. He was prescribed Venlafaxine to treat presumptive Cataplexy, without any benefit. Eventually, he was referred to neurology/sleep clinic. Cataplexy questionnaire was administered and was negative: specifically, there were no emotional triggers of his episodes, such as hearing a joke, laughing or crying. Due to the stereotyped nature of his spells, he was referred to epilepsy specialist. He underwent 72 hours video EEG monitoring which showed that his clinical episodes were associated with EEG abnormality, suggestive of frontal lobe epilepsy. He was placed on Keppra and Oxcarbazepine. On follow up visit, he reported improvement in seizure frequency from 10-20/day to 1-2/day.

Conclusion: A validated1 clinical Tool such as Stanford Cataplexy questionnaire helps in differentiation of Cataplexy from other forms of transient muscle weakness. It can prevent 15 years delay in diagnosis and treatment of patients, or use of unnecessary medication.

1271

HIGH-DENSITY EEG CORRELATES DURING CONFUSIONAL AROUSALS IN A CHILD WITH A LONG-LASTING HISTORY OF SLEEPWALKING

Castelnovo, A.^{1,2}, *Amato, N.*², *Riccardi, S.*², *Pereno, M.*², *Miano, S.*², *Manconi, M.*²

¹Sleep Center, Neurocenter of the Southern Switzerland, Regional Hospital (EOC) of Lugano, Lugano, SWITZERLAND, ²Sleep Center, Neurocenter of the Southern Switzerland, Regional Hospital (EOC) of Lugano, Lugano, Switzerland, Lugano, SWITZERLAND

Introduction: Sleepwalking belongs to a family of disorders (Disorders of Arousal - DOA) that are thought to derive from incomplete arousals out of Non Rapid Eye Movement (NREM) sleep. At yet, our knowledge about the specific neural dynamics occurring during clinical episodes is limited and relies on one SPECT case study, four stereo-EEG case reports/ series and one single high-density electro-encephalography (hdEEG) case report. We herein describe a single case captured by hdEEG.

Report of Case: We collected two consecutive sleep recordings (using a 256-channel hdEEG coupled with standard videopolysomnography) of a non-medicated, otherwise healthy, 13-year-old male, with a history of recurrent daily sleepwalking episodes. We visually identified 17 behavioral events during sleep stage 3 and divided them into two groups: clear clinical episodes (n = 7)and simple movements associated with burst of delta waves (n = 10). Source power topography in the delta range (1-4 Hz) was computed using LORETA. Source images during selected episodes were compared to 30 second-windows of baseline stage 3 sleep. Comparisons were performed using statistical non-parametric mapping with supra-threshold cluster tests. Events were associated with an increase of delta power over the right frontopolar prefrontal cortex (rPFC) / Broadman area 10 (BA10) at their onset. This finding was clearly observable even when considering only clear-cut events, followed by the involvement of the right dorsolateral and medial prefrontal cortex / BA9 and of the left superior temporal gyrus/ BA 22. Conclusion: We were able to replicate a recently published case report by our group, where we highlighted the putative role of rPFC and PFC and prefronto-temporal circuit in DOA episodes. Intriguingly, we observed a lateralization of this effect, with a prominent right frontal involvement. Novel research has shown a physiological asymmetry in the generation of large slow waves between the two hemispheres. An increased right-left unbalance might prime behavioral episodes in DOA patients.

1272

CONTROL OF SLEEP-ASSOCIATED RESPIRATORY DISTRESS WITH A MANDIBULAR ADVANCEMENT ORAL APPLIANCE IN AN UNUSUAL CASE OF A PATIENT WITH A MILD RATHER PERSISTENT NOCTURNAL BASELINE HYPOVENTILATION/HYPOXIA

Manetta, I.¹, Almeida, A.², Schwartz, D.³, Meira e Cruz, M.⁴ ¹Pontifical Catholic University of Campinas, Campinas, BRAZIL, ²School of Medical Science, Campinas State University, Campinas, BRAZIL, ³Center for Sleep Medicine, Chicago, IL, ⁴Sleep Unit, Cardiovascular Center of University of Lisbon, Lisbon School of Medicine, Lisbon, PORTUGAL **Introduction:** Normal respiratory function is crucial for adequate sleep. Sleep Related Breathing Disorders, namely Obstructive Sleep Apnea (OSA) are frequent conditions among a large spectrum of obstructive events in the upper airway which are often linked to significant sleep related oxyhemoglobin dessaturation levels compared to those observed in baseline PSG-oximetry recorded. On the other hand, low baseline oxygen saturation (LBOS) is commonly linked to symptomatic cardiorespiratory disturbances which may adversely impact respiratory outcomes either awake or during sleep. Therefore it is relevant to be aware of the baseline ventilatory status in order to optimize the therapeutic care.

Report of Case: We present an unusual case of a patient with moderate OSA which was successfully controlled with a Mandibular Advancement Oral Appliance (OAm). Despite the control with the OAm, the patient still maintained a nocturnal pattern of hypoventilation/hypoxia. The 53 yo female patient with normal weight/ height ratio (BMI=25,2) complaining of non restorative sleep, tiredness, impaired memory, excessive diurnal sleepiness (Epworth Sleepiness Scale - ESS=10 and bruxism with a PSG diagnosis of moderate OSA (BaselineO2Sat=94%; IAH=17,4 ev/h; ODI=32,9 ev/h; T90=34,7% of TST) was referred for treatment with a OAm. A PM Type 1 positioner was inserted and titrated until 12 mm of advancement (80% of maximal measured protrusion). Within 6 months follow up and after clinical titration, there was a clear symptomatic and objective improvement with resolution of all major complaints including sleepiness (ESS=5). Titration PSG showed a normalization in all parameters (IAH=1,4 ev/h;ODI=4 ev/h;T90=0,1% of TST). Baseline O2 Sat however remained low (93%) in the titration PSG even though no symptoms or signs of a disorder existed even after discounting for Ph related metabolic changes (normal levels of HCO3)

Conclusion: This is an interesting case of an unusual patient who presented with a LBOS level in the diagnostic PSG. Despite the

therapeutic success of the OAm in controlling the sleep related respiratory condition, measured by all otherwise normalized PSG based sleep and respiratory parameters, maintained a LBOS in the titration PSG without any signs or symptoms of disease.

1273

DOES A MORE PERSONALIZED APPROACH TO DESENSITIZATION AND DAYTIME TITRATIONS IMPROVE PAP COMPLIANCE, PAP THERAPY SATISFACTION/EFFICACY, AND PERHAPS EVEN SAVE RESOURCES?

Yeager K.K., MD, MPH¹, Kern J.D., MD², Cutrufello N.J., MD², Begay M.N., MD², Glasser, RPSGT J.²

¹ University of New Mexico Division of Pulmonary Critical Care and Sleep Medicine, Albuquerque, New Mexico, ²Raymond G. Murphy VA Medical Center Sleep Disorders Center, Albuquerque, New Mexico

Introduction: Seventy three patients at the Raymond G Sleep Center within the New Mexico VA Healthcare System were identified as patients who were at high risk for PAP therapy non-compliance, were invited to participate in a new patient service program.

Report of Case: This program included an individualized daytime desensitization process coupled with a potential daytime titration. The final analysis of these data revealed co-morbid conditions that may serve as barriers to PAP usage as well as medical conditions which are known to benefit from compliance on PAP therapy. Special attention was paid to co-morbid psychiatric conditions.

Conclusion: This process resulted in a roughly 25% increase in PAP compliance, a 30% change in PAP modality and improved access to in-lab titrations. This study elucidated factors that may help predict success or failure regarding PAP therapy.

Author Index

A

Abbasi, A. A
Abbott, S. M
Abdi, H
Abedi, A
Abel, T
Aboussouan, L. S
Abraham, R
Abramson, M
Abreu, A. R
Abu Awad, Y
Abu-Halimeh, N
Acaster, S
Acenowr, C
Actanta, P
Acharya, S
Ackley, S
Adachi, T
Adams, I
Adams, J
Adams, R. J
Adeleye, A
Adkins, E0754
Adlou, B
Adornetti, J. P
Aeschbach, D
Aesmani, D
Afolabi-Brown, O
Agam, N
Agrawal, R
Agrawal, S
Agrawal, S
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0907 Ajisebutu, A. .0906
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Ajjala, D. .0760
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Ajijala, D. .0760
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Ajijala, D. .0760
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0361 Aird, C. .1189 Ajayi, A. O. .0906 Ajjala, D. .0760 Akerstedt, T. .0460, 0460 Akey, M. A. .0556
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0907 Ajisebutu, A. .09006 Akerstedt, T. .0460, 0460 Akey, M. A. .0556
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0907 Ajisebutu, A. .0906 Ajjala, D. .0760 Akerstedt, T. .0460, 0460 Akey, M. A. .0556 Akram, U. .0556
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .09077 Ajisebutu, A. .0906 Ajjala, D. .0760 Akerstedt, T. .0460, 0460 Akey, M. A. .0556 Akram, U. .0556 Al-Abri, M. A. .0396
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Akerstedt, T. .0460, 0460 Akey, M. A. .0556 Akram, U. .0556 Akram, U. .0556 Al-Abri, M. A. .0396 Alaca, A. N. .1028 Al-Azzawi, S. .0560
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Ajjala, D. .0760 Akerstedt, T. .0460, 0460 Akey, M. A. .0556 Akram, U. .0556 Al-Abri, M. A. .0396 Alaca, A. N. .028 Al-Azzawi, S. .0560
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Ajjala, D. .0760 Akerstedt, T. .0460, 0460 Akey, M. A. .0556 Akram, U. .0549 Al-Abri, M. A. .0396 Alaca, A. N. .028 Al-Azzawi, S. .0560 Albrecht, E. .0726
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Ajjala, D. .0760 Akerstedt, T. .0460, 0460 Akey, M. A. .0556 Alram, U. .0549 Al-Abri, M. A. .0396 Alaca, A. N. .1028 Al-Azzawi, S. .0560 Albrecht, E. .0726 Albrecht, J. .1178
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Ajiala, D. .0760 Akerstedt, T. .0460, 0460 Akey, M. A. .0556 Alram, U. .0549 Al-Abri, M. A. .0556 Allaca, A. N. .0286 Al-Azzawi, S. .0560 Albrecht, E. .0726 Albrecht, J. .1178 Albrecht, J. S. .0538, 1183
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Aijala, D. .0760 Akerstedt, T. .0460, 0460 Akey, M. A. .0556 Alram, U. .0549 Al-Abri, M. A. .0556 Alaca, A. N. .028 Al-Azzawi, S. .0560 Albrecht, E. .0726 Albrecht, J. .1178 Albrecht, J. .1178 Albrecht, J. .1097
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Aijala, D. .0760 Akerstedt, T. .0460, 0460 Akey, M. A. .0556 Alram, U. .0549 Al-Abri, M. A. .0396 Alaca, A. N. .0028 Al-Azzawi, S. .0560 Albrecht, E. .0726 Albrecht, J. S. .0538, 1183 Alcantara, C. .1097 Alenazi, A. .1059
Agrawal, S. .0707 Agudelo, C. .1144 Agudelo, C. W. .0822, 0823, 1012 Aguila, A. P. .0556 Ágústsson, J. S. .0447, 0448 Ahmad, M. .0477 Ahmad, M. .0477 Ahmad, M. .0162 Ahmed, N. .0795 Aiello, A. .0361 Aird, C. .1189 Ajayi, A. O. .0977 Ajisebutu, A. .0906 Ajjala, D. .0760 Akerstedt, T. .0460, 0460 Akey, M. A. .0556 Akram, U. .0549 Al-Abri, M. A. .0396 Alaca, A. N. .1028 Al-Azzawi, S. .0560 Albrecht, E. .0726 Albrecht, J. .1132 Albrecht, J. .1178 Albrecht, J. .0538, 1183 Alcantara, C. .0097 Alessi, C. .0467, 0537, 1017
Agrawal, S.
Agrawal, S. 0707 Agudelo, C. 1144 Agudelo, C. W. 0822, 0823, 1012 Aguila, A. P. 0556 Ágústsson, J. S. 0447, 0448 Ahmad, M. 0477 Ahmad, M. 0162 Ahmed, N. 0162 Ahmed, N. 0795 Aiello, A. 0361 Aird, C. 1189 Ajayi, A. O. 0977 Ajisebutu, A. 0906 Ajjala, D. 0760 Akerstedt, T. 0460, 0460 Akey, M. A. 0556 Akram, U. 0549 Al-Abri, M. A. 0396 Alaca, A. N. 1028 Al-Azzawi, S. 0560 Albrecht, J. 1178 Albrecht, J. 1178 Albrecht, J. 1097 Aleazi, A. N. 0538, 1183 Alcantara, C. 0467, 0537, 1017 Alessi, C. A. 0475, 0581 Alfano, C. A. 0273, 0273, 02939, 1090
Agrawal, S.

Alhejaili, F	
Al Ikhwan, M	
Alio, C	
Alioa, M. S	
Alipio Jocson, V	
Aljarod, T	
Alkathiry, A	
Al-Kindi, T.	
Alkire, C	
Alkire, C	
Alkozei, A 0014, 0038, 0070, 0079,	
	1077, 1083, 1158, 1158, 1160
Allard, T	
Allard, T. L	
Allen, R	
Allen, R. P	
Allison, T. G.	
Alloy, L. B	
Almaghasilah, A	
Almasy, L	
Almeida, E	.0608, 1136, 1136, 1177, 1177
Almeida, F. R.	
Almeida, L. A.	
Almklov, E. A.	
Almojaddidi, H	
Al-Mughales, J.	
Aloia, M. S	
Alothman, S.	
Alperin, N.	
Al Saleh, Q	
Alsallum, F.	
Alsameen, M	
Alschuler, V	
Alschuler, V	
Alschuler, V	
Alschuler, V.Al-Shawwa, B.Alshehri, M.Altman, Y.Altmann, E. M.Alton, D.Amade, S.Amalean, A.Amaral, F. G.Amatrudo, G.Amatrudo, G.Ambati, A.	
Alschuler, V	$\begin{array}{c}$
Alschuler, V	$\begin{array}{c}$
Alschuler, V	$\begin{array}{c}$
Alschuler, V.Al-Shawwa, B.Alshehri, M.Altman, Y.Altmann, E. M.Alton, D.Amade, S.Amalean, A.Amaral, F. G.Amatrudo, G.Amatrudo, G.Ambati, A.Ambati, A.Ambosone, C.Amdur, R.Amdur, R.Amman, A.Amorim, J.Andri, R.Anorim, J.Andri, R.Anorim, J.Andur, R.Amorim, J.Annafi, R.Anafi, R.Ancoli-Israel, S.	$\begin{array}{c}$
Alschuler, V.Al-Shawwa, B.Alshehri, M.Altman, Y.Altmann, E. M.Alton, D.Amade, S.Amalean, A.Amaral, F. G.Amatrudo, G.Amatrudo, G.Ambati, A.Ambati, A.Ambati, A.Amdur, A.Amdur, R.Aminian, A.Amorim, J.Andru, R.Amorim, J.Andri, R.Amari, R.Amari, R.Amari, R.Amari, R.Annorim, J.Ann, H.Anafi, R.	$\begin{array}{c}$
Alschuler, V.Al-Shawwa, B.Alshehri, M.Altman, Y.Altmann, E. M.Alton, D.Amade, S.Amalean, A.Amaral, F. G.Amatrudo, G.Amatrudo, G.Ambati, A.Ambati, A.Ambour, A.Amdur, A.Amdur, R.Amman, A.Amorim, J.Annorim, J.Annafi, R.Anafi, R.Anderer, P.Anders, T. F.	$\begin{array}{c}$
Alschuler, V.Al-Shawwa, B.Alshehri, M.Altman, Y.Altmann, E. M.Alton, D.Amade, S.Amalean, A.Amaral, F. G.Amatrudo, G.Amatrudo, G.Ambati, A.Ambati, A.Amboxone, C.Amdur, A.Amdur, R.Amorim, J.Annorim, J.Annafi, R.Anafi, R.Anderer, P.Andersen, M.	$\begin{array}{c}$
Alschuler, V.Al-Shawwa, B.Alshehri, M.Altman, Y.Altmann, E. M.Alton, D.Amade, S.Amalean, A.Amaral, F. G.Amatrudo, G.Amatrudo, G.Ambati, A.Ambati, A.Ambour, A.Amdur, A.Amdur, R.Amman, A.Amorim, J.Annorim, J.Annafi, R.Anafi, R.Anderer, P.Anders, T. F.	$\begin{array}{c}$
Alschuler, V.Al-Shawwa, B.Alshehri, M.Altman, Y.Altmann, E. M.Alton, D.Amade, S.Amalean, A.Amaral, F. G.Amatrudo, G.Amatrudo, G.Ambati, A.Ambati, A.Ambour, A.Amdur, R.Aminian, A.Amorim, J.Andri, R.Anafi, R.Anafi, R.Anderer, P.Andersen, M.Andersen, M. L.0022, 0272, 0493,	$\begin{array}{c}$
Alschuler, V.Al-Shawwa, B.Alshehri, M.Altman, Y.Altmann, E. M.Alton, D.Amade, S.Amalean, A.Amaral, F. G.Amatrudo, G.Ambati, A.Ambati, A.Ambosone, C.Amdur, A.Amdur, R.Amari, A.Amdur, R.Annafi, R.Anorim, J.Anderer, P.Andersen, M.Anderson, C.Anderson, C.	$\begin{array}{c}$
Alschuler, V. Al-Shawwa, B. Alshehri, M. Altman, Y. Altmann, E. M. Alton, D. Alton, D. Amade, S. Amalean, A. Amaral, F. G. Amatrudo, G. Ambati, A. Ambati, A. Amboxi, A. Ambati, A. Ambrosone, C. Amdur, A. Amdur, R. Amorim, J. Ann, H. Anafi, R. Anderer, P. Andersen, M. Andersen, M. Anderson, C. Anderson, S. E.	$\begin{array}{c}$
Alschuler, V.Al-Shawwa, B.Alshehri, M.Altman, Y.Altmann, E. M.Alton, D.Amade, S.Amalean, A.Amaral, F. G.Amatrudo, G.Ambati, A.Ambati, A.Ambosone, C.Amdur, A.Amdur, R.Amari, A.Amdur, R.Annafi, R.Anorim, J.Anderer, P.Andersen, M.Anderson, C.Anderson, C.	$\begin{array}{c}$

Anderson, W. M
Anderson, W
Anderson, W. M
Andre, C. J
Andrefsky, J. C
Andrew, M
Andrew, R
Andrews, A
ANDRILLON, T
Angadi, S. S
Ankita, A
Anlap, I
Antelmi, E
Antila, H
Antonescu, C
Antony, K
Aparicio, H. J
Apolinar, G
Appleby, G
Appleton, S. L
Apseloff, G
Aquino, A
Araujo, L. G
Araujo, P
Arbaijo, 1
Arobul, C
Areekui, w
Arenano, A. K
Armoni-Domany, K
Armstrong, B
Arnal, P
Arnal, P. J
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arp, M. .0934
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arp, M. .0934 Arputhan, A. .0896
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Arora, M. J. .0108, 0108 Arp, M. .0934 Arputhan, A. .0896 Arra, N. .0916, 0916
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Arora, M. J. .0108, 0108 Arp, M. .0934 Arputhan, A. .0896 Arra, N. .0916, 0916 Arrigoni, E. .0156, 0156
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arraigoni, E. .0156, 0156 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Array, N. .0916, 0916 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .0099
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .0099 Arsintescu, L. .0310
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .0030 Areite, M. .00310 Arreating, L. .0310
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsintescu, L. .0310 Artemis, L. .0310 Artemis, L. .0352
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arragoni, E. .0156, 0156 Arshad, O. A. .0025 Arsintescu, L. .0310 Artemis, L. .0310 Artemis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arragoni, E. .0156, 0156 Arshad, O. A. .0025 Arsintescu, L. .0310 Artermis, L. .0310 Artermis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arruin, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .0099 Arsintescu, L. .0310 Artemis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849 Asante, K. .0966 Asarnow, L. .0499
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arp, M. .0934 Arputhan, A. .0896 Arra, N. .0916, 0916 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .0039 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849 Asante, K. .0966 Asarnow, L. .0478
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arrugoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .0099 Arstick, M. .00310 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849 Asante, K. .0966 Asarnow, L. .0178 Asch, D. A. .0196, 0261
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Arora, M. J. .0108, 0108 Arp, M. .0934 Arputhan, A. .0896 Arrar, N. .0916, 0916 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .0310 Artemis, L. .0310 Artemis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849 Asarnow, L. .04597, 0644 Asarnow, L. .04597, 0644 Ashe, D. A. .0178 Asch, D. A. .0196, 0261 Ash, T. .0624
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Arora, M. J. .0108, 0108 Arp, M. .0108, 0108 Arran, N. .0934 Arputhan, A. .0896 Arra, N. .0916, 0916 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .00310 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849 Asarnow, L. .04597, 0644 Asarnow, L. .0499 Asbee, J. .0178 Asch, D. A. .0196, 0261 Ash, T. .0624 Asin, J. .0673, 0673
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arputhan, A. .0996 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .00310 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849 Asarnow, L. .04597, 0644 Asarnow, L. .04597, 0644 Asarnow, L. .0499 Asbee, J. .0178 Asch, D. A. .0196, 0261 Ash, T. .0624 Asin, J. .0673, 0673 Assadi, M. .0128
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arputhan, A. .0996 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .00310 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849 Asarnow, L. .0499 Asbee, J. .0178 Asch, D. A. .0196, 0261 Ash, T. .0673, 0673 Assadi, M. .0128 Athey, A. .0226, 0236, 0240
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arp, M. .0934 Arputhan, A. .0994 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .00310 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849 Asante, K. .0966 Asarnow, L. .0499 Asbee, J. .0178 Asch, D. A. .0196, 0261 Ash, T. .0673, 0673 Assadi, M. .0128 Athey, A. .0226, 0236, 0240 Atkins, Jr., N. .0485
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arp, M. .0934 Arputhan, A. .0936 Arra, N. .0916, 0916 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .00310 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0352 Asante, K. .0597, 0644 ASAKA, y. .0848, 0849 Asante, K. .0966 Asarnow, L. .0178 Asch, D. A. .0196, 0261 Ash, T. .0624 Asin, J. .0673, 0673 Assadi, M. .0128 Athey, A. .0226, 0236, 0240 Atkins, Jr., N. .0485
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arp, M. .0934 Arputhan, A. .0994 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .0025 Arsic, M. .00310 Artemis, L. .0310 Artemis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849 Asarnow, L. .0456 Assante, K. .0966 Asarnow, L. .0178 Asch, D. A. .0196, 0261 Ash, T. .0624 Asin, J. .0673, 0673 Assadi, M. .0128 Athey, A. .0226, 0236, 0240 Atkins, Jr., N. .0485 Atochin, D. N. .0133
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arputhan, A. .0934 Arputhan, A. .0934 Arrang, N. .0108, 0108 Arra, N. .0916, 0916 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .0099 Artemis, L. .0310 Artemis, L. .0310 Artemis, L. .0597, 0644 ASAKA, y. .0848, 0849 Asarnow, L. .0499 Asbee, J. .0178 Asch, D. A. .0196, 0261 Ash, T. .0624 Asin, J. .0673, 0673 Assadi, M. .0128 Athey, A. .0226, 0236, 0240 Atkins, Jr., N. .0485 Atochin, D. N. .0133 Attali, V. .0665
Arnal, P. J. .0352, 0546, 0616, 1211, 1211 Arnedt, J. .0039, 0513, 0532 Arnett, L. .0465 Arngrimsson, S. A. .0260 Arnulf, I. .0026, 0798, 0798 Aronis, J. .1146 Arora, M. J. .0108, 0108 Arp, M. .0934 Arputhan, A. .0994 Arrigoni, E. .0156, 0156 Arshad, O. A. .0025 Arsic, M. .0025 Arsic, M. .00310 Artemis, L. .0310 Artemis, L. .0352 Arvai, k. .0597, 0644 ASAKA, y. .0848, 0849 Asarnow, L. .0456 Assante, K. .0966 Asarnow, L. .0178 Asch, D. A. .0196, 0261 Ash, T. .0624 Asin, J. .0673, 0673 Assadi, M. .0128 Athey, A. .0226, 0236, 0240 Atkins, Jr., N. .0485 Atochin, D. N. .0133

Atwood, C. W
Atwood, M. E
Au, C. T
Au, D. H
Auclair, J
Auerbach, A
Auerbach, S. L
Aufricht, J
August, J
Auhasira, R
Aung, A. T
Auricchio, L
Avirappattu, G
Ayad, M. W
Ayappa, I
Aylor, J
Ayoub, S
•
Aysola, R
Azarbarzin, A
Azizi, S. A
Azzarto, E

B

SLEEP, Volume 43, Abstract Supplement, 2020

Banks, S
Banno, M
Bansal, K
BANZOLI, C. V
Barak, S
Barakat, L
Barateau, L
Bare, L. A
Barger, L. K
Baril, AA
Baril, AA
Barillas-Lara, M
Barinas-Mitchell, E
Barinas-Mitchell, E. J
Barker, D. H
Barker, M
Barnes, A
Barnes, F
Barnes, M
Barnet, J. H
Barngrover, S
Barranca, M
Barrett, M
Bartet, P
Bartle, A
Bartlett, D. J
Baruch, M
Barwick, F
Basheer, R
Bashore, L
Basiarz, E
Basishvili, T
Dusisiii, i
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292,
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A 0891, 0891 Bassetti, C
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A 0891, 0891 Bassetti, C
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A 0891, 0891 Bassetti, C
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A 0391, 0891 Bassetti, C
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A. . 0891, 0891 Bassetti, C. . 0521 Basta, M. . 0458, 0506, 0585, 1035, 1120, 1130 Bastien, C. . 0553, 1108 Bastien, L. . 0954, 0962 Bastin, G. . 0632 Bathurst, N. G. . 0632 Batterham, P. J. . 0520 Batterham, P. J. . 0524 Bauer, E. D. . 0766 Baughn, J. M. . 0895 Baurr, J. A. . 0392, 0393, 0818, 0943 Baur, J. A. . 0346 Baxter, B. . 0113 Bayon, V. . 0726 Bazalakova, M. . 0808, 0808, 0870 Bean, C. . 1084 Bean, C. A. . 00458 Beard, B. . 0286 Beaugris, L. . 0286 Beaugris, L. . 0286 Beaulieu-Bonneau, S. . 0511
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0292, 0304, 1100, 1100 Bassam, A. . 0891, 0891 Bassam, A. . 0521 Basten, C. . 0553, 1103 Bastien, C. . 0553, 1108 Bastien, L. . 0954, 0962 Bastin, G. . 0632 Bathurst, N. G. . 0632 Batterham, P. J. . 0524 Bauer, E. D. . 0766 Baughn, J. M. . 0392, 0393, 0818, 0943 Baur, J. A. . 0346 Baxter, B. . 0113 Bayon, V. . 0726 Bazalakova, M. . 0808, 0808, 0870 Bean, C. . 00342 Bearty, C. . 0286 Beaugris, L. . 0359 Bearty, C. . 0286 Beaugris, L. . 0511 Beck, A. J. . 0917 Beck, S. E. . 0885, 0885
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0304, 1100, 1100 Bassam, A
Basner, M 0105, 0105, 0187, 0196, 0261, 0282, 0291, 0292, 0304, 1100, 1100 Bassam, A

	0057, 0057, 0463, 0463, 0583, 0583,	
· · · · · · · · · · · · · · · · · · ·		· · · · ·
,		
	0091, 0325, 0329, 0335, 0422, 0425,	
· · · · · · · · · · · · · · · · · · ·		· · · · ·
	· · · · · · · · · · · · · · · · · · ·	
Benoit, K. M		0944
Bergman, B. C		0295, 0295
Berkley, A. S		0839
• •		
· · · · · · · · · · · · · · · · · · ·		
•		
•		
·		
· · · · · · · · · · · · · · · · · · ·		
U .		

Bhavsar, R
Bhyrapuneni, G
Bialasiewicz, P
Bichuetti, D. B
Biddle, J
Biggs, M
Billings, M. E
Birks, B. R
Birse, C. E
Bisesi, P. J
Bishop, T
1
Bishop, T. J
Bishop, T. M
Bishop-Gilyard, C. T
Bisson, J. B
Bittencourt, L
Bixle, E
Bixler, E. O 0319, 0457, 0458, 0585, 0878, 0890, 0919,
0920, 0936, 1107
Bizhanova, Z
Black, J
Black, M. M
Blackwell, T
Blair, E. E
Blais, H
Blanc, J 0373, 0377, 0863, 0864, 0964, 1064, 1082, 1089, 1189
Blank, E
Blase, A
Blattner, M
Blattner, M. S
Blennow, K
Bliwise, D
Bliwise, D. L
Bocanegra, Y
Boehm, J
Boeve, B
Boeve, B. F
Bogan, R
Bogan, R. K
Bogner, J
Boh, M
Bojja, K
Boland, E. M
Boland, E. M
D 111 1 1 D 0055
Bolkhovsky, J. B
Bolkhovsky, J. B
Bollu, P. C
Bollu, P. C
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonner, H. .0622, 1052
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonner, H. .0622, 1052 Bonthapally, V. .0535
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonner, H. .0622, 1052 Bonthapally, V. .0535 Bonuck, K. .1179, 1181
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonner, H. .0622, 1052 Bonthapally, V. .0535
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonner, H. .0622, 1052 Bonthapally, V. .0535 Bonuck, K. .1179, 1181
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonner, H. .0622, 1052 Bonthapally, V. .0535 Bonuck, K. .1179, 1181 Book, S. W. .1110 Boon, M. .0637
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonner, H. .0622, 1052 Bonthapally, V. .0535 Bonuck, K. .1179, 1181 Book, S. W. .1110 Boon, M. .0637 Borah, E. .0483, 0483
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonner, H. .0622, 1052 Bonthapally, V. .0535 Bonuck, K. .1179, 1181 Book, S. W. .1110 Boon, M. .0637 Borah, E. .0483, 0483 Borcsok, R. .0159
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonner, H. .0622, 1052 Bonthapally, V. .0535 Bonuck, K. .1179, 1181 Book, S. W. .1110 Boon, M. .0637 Borah, E. .0483, 0483 Borcsok, R. .0159 Borker, P. V. .06677
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonthapally, V. .0535 Bonuck, K. .1179, 1181 Book, S. W. .1110 Boon, M. .0637 Borah, E. .0483, 0483 Borcsok, R. .0159 Borker, P. V. .0677 Borshenko, V. .0012
Bollu, P. C. .0692 Bollu, P. .0619, 0802 Boly, M. .0808, 0808 Bombarda, A. .0235 Bon, J. M. .0547 Bonikowske, A. R. .0719, 0719 Bonilla-Santiago, G. .0925 Bonnar, D. .0192 Bonner, H. .0622, 1052 Bonthapally, V. .0535 Bonuck, K. .1179, 1181 Book, S. W. .1110 Boon, M. .0637 Borah, E. .0483, 0483 Borcsok, R. .0159 Borker, P. V. .06677

Botbyl, J	
Bottary, R. M	
BOUCHEQUET, P	
Boudreau, E	
Boudreaux-Kelly, M. Y	7
Bourchetin, E	
Bourchtein, E	0
Bourgon, V	8
Boursier, J	9
Boustani, M	5
Boutin, I	
Boutros, C	
Bower, J	
Bower, J. L	
Bowles, N. P	
Bowman, M1088	
Bowman, M. A	
Bowman, M. A	
Boyle, J	
Boyle, J. T	
Boyle, L	
Bradley, B. F	
Bradshaw, C	
Brager, A. J	
Bragg, S	
Brake, L	
Braley, T	
Braley, T. J	
Branas, C0013, 0241, 0243, 0376, 0406, 0544, 0865, 1095, 1108	
Brandt, K	
Braun, I. M	
Bremer, V	
Brennan, E. M	
Brennan, E. M	2
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0126	2 6
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0126 Brim, W. .1186	2 6 6
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1088	2 6 6 8
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0126 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0115	2 6 8 5
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0126 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0115 Brock, M. .0866	2 6 6 8 5 0
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0860 Brock, M. S. .0399, 0803	2 6 8 5 0 3
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0126 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0866 Brock, M. S. .0399, 0802 Brodkin, E. S. .0974	2 6 8 5 0 3 4
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0126 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0866 Brock, M. S. .0399, 0802 Brodkin, E. S. .0972 Brodner, D. .0471	2 6 8 5 0 3 4 1
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1083 Brinkman, T. M. .0112 Brock, M. .0860 Brock, M. S. .0399, 0803 Brodkin, E. S. .0974 Brodkin, A. .0420	2 6 8 5 0 3 4 1 0
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1083 Brinkman, T. M. .0112 Brock, M. .0860 Brock, M. S. .0399, 0803 Brodkin, E. S. .0974 Brodkin, E. S. .0471 Brodtmann, A. .0420 Bronas, U. .0869	2 6 8 5 0 3 4 1 0 9
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0866 Brock, M. S. .0399, 0803 Brodkin, E. S. .0974 Brodkin, E. S. .0974 Brodtmann, A. .0420 Bronas, U. .0866 Brookfield, J. S. .0097	2 6 8 5 0 3 4 1 0 9 7
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0860 Brock, M. S. .0399, 0803 Brodkin, E. S. .0974 Brodkin, E. S. .0974 Brodtmann, A. .0420 Bronas, U. .0866 Brookfield, J. S. .0097 Brooks, J. .1004, 116	2 6 6 8 5 0 3 4 1 0 9 7 1
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0860 Brock, M. S. .0399, 0803 Brodkin, E. S. .0974 Brodkin, E. S. .0974 Brodkin, E. S. .0974 Brodkin, J. S. .0097 Brooks, J. .1004, 1161 Brooks, M. M. .0176	2 6 6 8 5 0 3 4 1 9 7 1 6
Brennan, E. M. 0729 Brenowitz, W. 1152 Brier, L. M. 0120 Brim, W. 1180 Brindle, R. C. 0162, 1088 Brinkman, T. M. 1012 Brock, M. 0860 Brock, M. 0860 Brodkin, E. S. 0974 Brodner, D. 0471 Brodner, D. 0471 Brodkin, E. S. 0974 Brodkin, J. S. 0974 Broks, J. 0420 Brooks, J. 0104, 1161 Brooks, M. M. 0170 Brothmann, L. 0964, 0995	$2 \\ 6 \\ 8 \\ 5 \\ 0 \\ 3 \\ 4 \\ 1 \\ 0 \\ 9 \\ 7 \\ 1 \\ 6 \\ 5 \\ 5 \\ 1 \\ 6 \\ 5 \\ 5 \\ 1 \\ 6 \\ 5 \\ 1 \\ 1 \\ 1 \\ 1 \\ 1 \\ 1 \\ 1 \\ 1 \\ 1$
Brennan, E. M. 0729 Brenowitz, W. 1152 Brier, L. M. 0120 Brim, W. 1180 Brindle, R. C. 0162, 1088 Brinkman, T. M. 1012 Brock, M. 0860 Brock, M. 0860 Brodkin, E. S. 0974 Brodner, D. 0471 Brodtmann, A. 0422 Brons, U. 0869 Brooks, J. 1004, 1161 Brooks, M. M. 0176 Brotman, L. 0964, 0992 Broussard, J. L. 0295, 0295	2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0162, 1088 Brok, M. .0162, 1088 Brok, M. .0162, 1088 Brokkin, E. S. .0162, 1089 Brokkin, E. S. .0399, 0802 Brodkin, E. S. .0974 Brodkin, E. S. .0974 Brodtmann, A. .0420 Bronas, U. .0471 Brodtmann, A. .0420 Bronkfield, J. S. .0097 Brooks, J. .1004, 1161 Brooks, M. M. .0176 Brotman, L. .0964, 0992 Broussard, J. L. .0295, 0292 Brown, F. M. .1101	2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0162, 1088 Brok, M. .0112 Brok, M. S. .0399, 0802 Brodkin, E. S. .0974 Brodtmann, A. .0420 Bronas, U. .0471 Brooks, J. .0097 Brooks, J. .0097 Brooks, M. M. .0176 Broussard, J. L. .0295 Brown, F. M. .1101 Brown, G. J. .0572	2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0162, 1088 Brock, M. .0162, 1088 Brok, M. S. .0162, 1088 Brok, M. S. .0162, 1088 Brok, M. .0162 Brok, M. S. .0399, 0802 Brodkin, E. S. .0972 Brodtmann, A. .0420 Bronas, U. .0471 Brooks, J. .0097 Brooks, J. .0097 Brooks, J. .0097 Brows, M. M. .0176 Brows, F. M. .0107 Brown, K. L. .0523, 0541, 0542 <td>2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3 2</td>	2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3 2
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0162, 1088 Brock, M. .0162, 1088 Brok, M. .0162, 1088 Brok, M. .0162, 1088 Brok, M. S. .0162, 1088 Brock, M. S. .0162, 1088 Brokk, M. .0162 Brok, M. S. .0399, 0802 Brodkin, E. S. .0399, 0802 Brodkin, E. S. .0399, 0802 Brodkin, E. S. .0974 Brodkin, E. S. .0399, 0802 Brodkin, E. S. .0974 Brodkin, E. S. .0399, 0802 Brodkin, E. S. .0974 Brodtmann, A. .0471 Brodtmann, A. .0420 Brons, U. .0866 Brooks, J. .0097 Broks, J. .0097 Broks, M. M. .0176 Brown, R. M. .0176 Brown, R. E. .0523, 0541, 0542<	2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3 2 4
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0162, 1088 Brock, M. .0162, 1088 Brok, M. S. .0162, 1088 Brok, M. S. .0399, 0802 Brokk, M. S. .0399, 0802 Brodkin, E. S. .0399, 0802 Brodkin, E. S. .0397 Brodkin, E. S. .0397 Brodtmann, A. .0420 Bronas, U. .0471 Brodtmann, A. .0420 Brons, U. .0869 Brookfield, J. S. .0097 Broks, J. .1004, 116 Brows, M. M. .0176 Brows, M. M. .0295, 0295 Brown, F. M. .101 Brown, G. J. .0523, 0541, 0542 Brown, K. L. .0523, 054	266850341097165513248
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0162, 1088 Brock, M. .0162, 1088 Brok, M. S. .0162, 1088 Brock, M. S. .0399, 0803 Brokkin, E. S. .0399, 0803 Brodkin, E. S. .0399, 0803 Brodkin, E. S. .0397 Brodkin, E. S. .0397 Brodkin, E. S. .0399, 0803 Brodkin, E. S. .0077 Brodtmann, A. .0420 Bronas, U. .0471 Broks, J. .0097 Brooks, M. M. .0176 Brows, M. M. .0176 Brows, M. M. .0176 Brown, F. M. .0295 Brown, G. J. .0573 Brown, K. L. .0523, 0541, 0542 <td>2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3 2 4 8 5</td>	2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3 2 4 8 5
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0162, 1088 Brock, M. .0162, 1088 Brok, M. .0162, 1088 Brok, M. .0162, 1088 Brok, M. .0162, 1088 Brock, M. S. .0162, 1088 Brock, M. .0162, 1088 Brock, M. .0162 Brock, M. S. .0399, 0803 Brodkin, E. S. .00974 Brodtmann, A. .0420 Bronas, U. .0471 Broks, J. .0471 Brooks, M. M. .0170 Brotman, L. .0964, 0992 Brown, F. M. .1101 Brown, G. J. .0523, 0541, 0542 Brown, K. L.	26685034109716551324859
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1083 Brinkman, T. M. .0112 Brock, M. .0162, 1083 Brinkman, T. M. .0112 Brock, M. .0162, 1083 Brok, M. .0162, 1083 Brok, M. S. .0162, 1083 Brock, M. S. .0162, 1083 Brock, M. S. .0399, 0803 Brock, M. S. .0399, 0803 Brodkin, E. S. .00974 Brodtmann, A. .0420 Bronas, U. .0471 Broks, J. .0047 Brooks, M. M. .0176 Browks, J. .0044, 1161 Browks, M. M. .0176 Brown, F. M. .0295, 0292 Brown, G	266850341097165513248593
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .0162, 1088 Brock, M. .0162, 1088 Brok, M. .0162, 1088 Brok, M. .0162 Brock, M. .0162 Brock, M. S. .0399, 0803 Brodkin, E. S. .00974 Brodkin, E. S. .0399, 0803 Brodkin, E. S. .00974 Brodtmann, A. .0420 Bronas, U. .0471 Broks, J. .00471 Brooks, M. M. .0076 Browks, J. .0044, 1161 Brooks, M. M. .0176 Brown, F. M. .0176 Brown, G. J. .0295, 0292 Brown, K. L. .05	2 6 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3 2 4 8 5 9 3 9
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1083 Brinkman, T. M. .0112 Brock, M. .0162, 1083 Brinkman, T. M. .0112 Brock, M. .0162, 1083 Brok, M. .0162, 1083 Brok, M. S. .0162, 1083 Brock, M. S. .0162, 1083 Brock, M. S. .0399, 0803 Brock, M. S. .0399, 0803 Brodkin, E. S. .00974 Brodkin, E. S. .0399, 0803 Brodkin, E. S. .0974 Brodkin, E. S. .0471 Broks, J. .0442 Brons, U. .0866 Broks, J. .0044, 1161 Brooks, M. M. .0176 Browks, J. .0044, 1061 Brown, F. M. .01076 Brown, G. J.	2 6 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3 2 4 8 5 9 3 9 3 9 3 9 3 9 3
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .00860 Brock, M. S. .0399, 0802 Brodkin, E. S. .0399, 0802 Brodkin, E. S. .0972 Brodmer, D. .0471 Brodtmann, A. .0420 Bronas, U. .0866 Brooks, J. .0097 Brooks, J. .0097 Brooks, J. .0471 Brooks, M. M. .0420 Brooks, J. .0097 Brooks, J. .0097 Brooks, M. M. .0420 Brows, J. .0097 Brows, M. M. .0176 Brows, M. M. .0170 Brows, M. M. .0170 Brown, F. M. .0104, 1161 Brown, G. J. .0295, 0292 Brown, K. L. .0523, 0541, 0542 Brown, S. .0338 Brown, S. A. .0212 Brundige, A	2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3 2 4 8 5 9 3 9 3 0
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1180 Brindle, R. C. .0162, 1083 Brinkman, T. M. .0112 Brock, M. .0162, 1083 Brinkman, T. M. .0112 Brock, M. .0162, 1083 Brinkman, T. M. .0112 Brock, M. .00162, 1083 Brokk, M. .0162, 1083 Brokk, M. .0162, 1083 Brokk, M. .0162, 1083 Brokkin, E. S. .0399, 0803 Brodkin, E. S. .0097 Brodkin, E. S. .0097 Brodkin, E. S. .0097 Brooks, J. .0047 Brooks, M. M. .0170 Brotnsa, L. .00964, 0999 Broussard, J. L. .0295, 0295 Brown, F. M. .0110 Brown, G. J. .0215 Brown, S. A. .0222 Brown, S. A.	2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3 2 4 8 5 9 3 9 3 0 7 7 1 6 5 5 1 3 2 4 8 5 9 3 9 3 0 7 7 1 6 5 5 1 3 2 4 8 5 9 3 9 3 0 7 7 1 6 5 5 1 3 2 4 8 5 9 3 9 3 0 7
Brennan, E. M. .0729 Brenowitz, W. .1152 Brier, L. M. .0120 Brim, W. .1186 Brindle, R. C. .0162, 1088 Brinkman, T. M. .0112 Brock, M. .00860 Brock, M. S. .0399, 0802 Brodkin, E. S. .0399, 0802 Brodkin, E. S. .0972 Brodmer, D. .0471 Brodtmann, A. .0420 Bronas, U. .0866 Brooks, J. .0097 Brooks, J. .0097 Brooks, J. .0471 Brooks, M. M. .0420 Brooks, J. .0097 Brooks, J. .0097 Brooks, M. M. .0420 Brows, J. .0097 Brows, M. M. .0176 Brows, M. M. .0170 Brows, M. M. .0170 Brown, F. M. .0104, 1161 Brown, G. J. .0295, 0292 Brown, K. L. .0523, 0541, 0542 Brown, S. .0338 Brown, S. A. .0212 Brundige, A	2 6 6 8 5 0 3 4 1 0 9 7 1 6 5 5 1 3 2 4 8 5 9 3 9 3 0 7 3 2 4 8 5 9 3 9 3 0 7 3

D 101 NO 1
Buchfuhrer, M. J
Buchman, A. S
Buck, C
Bucks, R. S
Budd, K
Budhiraja, R
Bugos, J
Bujanover, S
Bukartyk, J
Bukhtiyarova, O
Bullock, A
Bullock, M. M
Buman, M. P
Bunnell, B
Burdayron, R
Burgess, H
Burgess, H. J
Burke, J. F
Burke, L
Burke, S. L
Burke, T. A
Burke, T
Burke, T. M
Burns, A
Burns, A. I
Burns, M. P
Burns, T
Burr, L. E
Burschtin, O. E
Bush, M
Bussieres, J
Butler, B. P
Butler, M
Butler, M. P
Butz, D
Buxton, O
Buxton, O. M 0033, 0085, 0296, 0296, 0303, 0303, 0320, 0360,
0360, 0371, 0858
Buysse, D. J
Byars, K. C
Byrnes, W. C
Byun, E1140
Byun, JI

С

c, S
Cabrera, C. I
Cade, B
Cahn-Hidalgo, D
Cain, P
Cain, S. W
Cairney, S. A
Calero, K
Calhoun, C
Calhoun, D
Calhoun, S. L 0319, 0457, 0458, 0476, 0742, 0878, 0890, 0919,
0920, 0936
Callander, D
Camacho, M
Campbell, A. D
Campbell, I. G
Campbell, M. C

~		
Campo, D.		0579
	.0188, 0189, 0189, 0199, 0274, 0290,	
Caples, M.		0994
Caples, S. M		.0719, 0719
· ·		
Carlson, A		0388
Carlson, G		.0537, 0581
Carmichael, K. E		0186
Carmichael, O. T		0862
Carreon, J. D		0572
Carrier, J.		.0354. 1133
Carroll, C. M		0415
Carroll, L		0820
Carvalho, D. Z		0355
Casario, K		
Casoni, F		0522
Castelnovo A		1122 1122
Castillo, P		.0646, 0805
Castleberry, L. A		.0401, 0851
Castronovo, V		0526, 0841
Cauley, J. A.		0856
Cawthon, P. M.		
<i>,</i>		
		0810
Cedernaes, J		.0317, 0317
Cedernaes, J		.0317, 0317
Cedernaes, J Ceesay, P		.0317, 0317 .0487, 0488
Cedernaes, J Ceesay, P Cellini, N		.0317, 0317 .0487, 0488 1193
Cedernaes, J Ceesay, P Cellini, N Ceren, O		.0317, 0317 .0487, 0488 1193 .0487, 0488
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerny, A.		.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerny, A. Cetin, D.		.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerny, A. Cetin, D.		.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerny, A. Cetin, D. Cha, J.		.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583 0724
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerny, A. Cetin, D. Chabal, S. A.		.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583 0724 0284
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerny, A. Cetin, D. Chabal, S. A. Chachad, R.		.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583 0724 0284 .0078, 0174
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerny, A. Cetin, D. Cha, J. Chabal, S. A. Chachad, R. Chacko, A.		.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583 0724 0284 .0078, 0174 0645
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cereny, A. Cetin, D. Chabal, S. A. Chachad, R. Chacko, A. Chagas Miranda, R.	E	.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583 0724 0284 .0078, 0174 0645 0832
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cereny, A. Cetin, D. Chabal, S. A. Chachad, R. Chacko, A. Chagas Miranda, R.		.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583 0724 0284 .0078, 0174 0645 0832
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerny, A. Cetin, D. Cha, J. Chabal, S. A. Chachad, R. Chacko, A. Chagas Miranda, R. CHAI, Y.	E	.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583 0724 0284 .0078, 0174 0645 0832 0060
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerry, A. Cetin, D. Cha, J. Chabal, S. A. Chachad, R. Chacko, A. Chagas Miranda, R. CHAI, Y. Chakravorty, S.	E	.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583 0724 0284 .0078, 0174 0645 0832 0060 1093, 1096
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerry, A. Cetin, D. Cha, J. Chabal, S. A. Chachad, R. Chacko, A. Chagas Miranda, R. CHAI, Y. Chakravorty, S. Chalifour, K.	E	.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583 0724 0284 .0078, 0174 0645 0832 0060 1093, 1096 1041
Cedernaes, J. Ceesay, P. Cellini, N. Ceren, O. Cerny, A. Cetin, D. Cha, J. Chabal, S. A. Chachad, R. Chacko, A. Chagas Miranda, R. CHAI, Y. Chalfour, K. Chambers, A. M.	E	.0317, 0317 .0487, 0488 1193 .0487, 0488 .0435, 0436 .0583, 0583 0724 0284 .0078, 0174 0645 0832 0060 1093, 1096 1041 0099

Chan, H. L
Chan, J
Chan, K. C
Chan, L
Chan, SP
Chan, W. S
Chan, W
Chan-Chi, C
Chandler, J. F
Chandler, N. G
Chandler, P
Chang, AM
Chang, CC
Chang, J
Chang, S
Chang, SF
Chang, YP
Chang, YF
Chanko, N
Chapman, J
Chapman, J. L
Chappel-Farley, M. G
Charest, J
Charlesworth, J. D
Chase, M. H
Chasens, E
Chasens, E. R
Chatila, W
Chatterjee, R
Chatterton, B
Chatterton, B. D
Chaturvedi, S
Chauvette, S
Chediak, A. D
Chee, M. W
Chee, M
Chein, K
Chen, B
Chen, CC
Chen, D
Chen, G
Chen, GT
Chen, I. Y
Chen, JC
Chen, K
Chen, K
Chen, K. Y
Chen, L
Chen, M. L
Chen, M
Chen, M
Chen, PC
Chen, PC
Chen, PC
Chen, PC
Chen, PC. .0063 Chen, S. .0732 Chen, YC. .1027 Chen, Y. .0253
Chen, PC. .0063 Chen, S. .0732 Chen, YC. .1027 Chen, Y. .0253 Cheng, J. .0480
Chen, PC. .0063 Chen, S. .0732 Chen, YC. .1027 Chen, Y. .0253 Cheng, J. .0480 Cheng, J. .0652
Chen, PC. .0063 Chen, S. .0732 Chen, YC. .1027 Chen, Y. .0253 Cheng, J. .0480 Cheng, J. .0652 Cheng, K. Y. .0260
Chen, PC. .0063 Chen, S. .0732 Chen, YC. .1027 Chen, Y. .0253 Cheng, J. .0480 Cheng, J. .0652 Cheng, K. Y. .0260 Cheng, P. .0006, 0007, 0203, 0465
Chen, PC. .0063 Chen, S. .0732 Chen, YC. .1027 Chen, Y. .0253 Cheng, J. .0480 Cheng, J. .0652 Cheng, K. Y. .0260 Cheng, P. .0006, 0007, 0203, 0465 Cheng-Yu, L. .0660
Chen, PC. .0063 Chen, S. .0732 Chen, YC. .1027 Chen, Y. .0253 Cheng, J. .0480 Cheng, J. .0652 Cheng, K. Y. .0260 Cheng, P. .0006, 0007, 0203, 0465 Cheng-Yu, L. .0660 Chenini, S. .0750
Chen, PC. .0063 Chen, S. .0732 Chen, YC. .1027 Chen, Y. .0253 Cheng, J. .0480 Cheng, J. .0652 Cheng, K. Y. .0260 Cheng, P. .0006, 0007, 0203, 0465 Cheng-Yu, L. .0660 Chenini, S. .0750 Chennaoui, M. .0299, 1042
Chen, PC. .0063 Chen, S. .0732 Chen, YC. .1027 Chen, Y. .0253 Cheng, J. .0480 Cheng, J. .0652 Cheng, K. Y. .0260 Cheng, P. .0006, 0007, 0203, 0465 Cheng-Yu, L. .0660 Chenini, S. .0750

Cheong, F	9 1
1131, 113	1
Cheung, J. M	6
Chhangani, B. S	
CHHOR, V	
Chia, C. W	
CHIANG, A	
Chiang, RY	
Chiappetta, L	
Chiba, S	
Chiem, E	
Chieng, LH	
Chin, N	
Chin, W	
Chindamporn, P	
Chini, B	
Chinoy, E. D	
Chirica, A	
Chitnis, T	
Chiu, HC	6
Cho, G	
Cho, SE	
Cho, Y	
Cho, Y	
Choi, H	
Choi, J	
Choi, S	
Chon, K. H	
Chopra, S	
Choukas-Bradley, S	
Chouraki, A	
Chow, L	
Chow, M	
Chow, P	
Choynowski, J	9
Choynowski, J. J	
Christensen, H	
Christian, L. M	5
Christina, M	
Chu, H	
Chu, JH	
Chua, J. R	
Chung A	
Chung, A	
Chung, J	
Chung, M	
Chung, N	
Chung, P	
Chung, S	
Cicalese, O	
Cielo, C. M	
Ciesla, J	
Ciesla, J. A	
Cipriano, A	2
Clark, A	
Clark, B	
Clark, D	
Clark, D. B	
Clark, P. R	5

SLEEP, Volume 43, Abstract Supplement, 2020

Claudatos, S
Clauw, D. J
Clay, M. A
Clemons, N. A
Coborn, J. E
Cocos, A
Coelho, F. M
Coelho, G
Coffey, A
•
Cogswell, D. T
Cohen, R
Cohen, S
Cohenour, M
Cohn, A
Cohn, V
Coimbra, B
Colclasure, A
Cole, A
Cole, R. A
Coleman, T. L
Coles, M
Collen, J. F
Collins, B. T
Collins, M. B
Collins, M
Collins-Rancourt, M. A0202
Colon-Feliciano, M1143
Colrain, I
Colvonen, P. J
Colwell, C
Combs, D 0370, 0656, 0656, 0701, 0717, 1172, 1188, 1188, 1209
Conceicao, A. S
Concepcion, E
Concepcion, E
Concepcion, E
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, M. B. .0176
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Conroy, D. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Conroy, D. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coon, D. W. .0826
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coon, D. W. .0826 Cooper, L. .1044, 1044
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coon, D. W. .0826 Cooper, L. .1044, 1044 Cope, A. .0508
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Cooper, L. .1044, 1044 Cope, A. .0508 Corbett, Q. .0157
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Cooper, L. .1044, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196
Concepcion, E. 1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Cooper, L. .0044, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M. .0286
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coor, D. W. .0826 Cooper, L. .1044, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M. .0286 Cornman, E. .0628
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coor, D. W. .0826 Cooper, L. .1044, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M. .0286 Cornman, E. .0628 Cornman, F. E. .0252
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coon, D. W. .0826 Cooper, L. .1044, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M. .0286 Cornman, E. .0628 Corona, F. E. .0252 Corser, B. .0745
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. .0532 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coon, D. W. .0826 Cooper, L. .1044, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M. .0286 Cornman, E. .0286 Cornman, F. E. .0252 Corser, B. .0745 Corsino, P. .0471
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coor, D. W. .0826 Cooper, L. .1044, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M. .0286 Cornman, E. .0286 Corona, F. E. .0252 Corser, B. .0745 Corsino, P. .0471 Cortés, C. .0249
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coon, D. W. .0826 Cooper, L. .01044, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M. .0286 Cornman, E. .0286 Corona, F. E. .0252 Corser, B. .0745 Corsino, P. .0471 Cortés, C. .0242
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coon, D. W. .0826 Cooper, L. .0144, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M. .0286 Coroman, E. .0252 Corser, B. .0745 Corsino, P. .0471 Cortés, C. .0242 Cortes, C. .0242
Concepcion, E. .1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. .0532 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coor, D. W. .0826 Cooper, L. .00441, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M. .0286 Corona, F. E. .0286 Corona, F. E. .0252 Corser, B. .0745 Corsino, P. .0471 Cortés, C. .0249 Cortes, C. .0242 Costanzo, M. .0702 Costedoat, G. .0254
Concepcion, E. 1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. .0532 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coor, D. W. .0826 Cooper, L. .00441, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M. .0286 Cornman, E. .0286 Corona, F. E. .0252 Corser, B. .0745 Corsino, P. .0471 Cortés, C. .0249 Cortes, C. .0427, 0428 Costanzo, M. .0702 Costedoat, G. .0254 Cotter, V. .1137
Concepcion, E. 1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coon, D. W. .0826 Cooper, L. .0144, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M .0286 Corona, F. E. .0252 Corser, B. .0745 Corsino, P. .0471 Cortés, C. .0249 Cortes, C. .0242 Costanzo, M. .0702 Costedoat, G. .0254 Cotter, V. .1137 Couderc, JP. .0571, 0571
Concepcion, E. 1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coon, D. W. .0826 Cooper, L. .0144, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M .0286 Corona, F. E. .0252 Corser, B. .0745 Corsino, P. .0471 Cortés, C. .0249 Cortes, C. .0242 Costanzo, M. .0702 Costedoat, G. .0254 Cotter, V. .1137 Couderc, JP. .0571, 0571 Covarrubias, I. .0076, 0154
Concepcion, E. 1176 Conduit, R. .0926 Conkright, W. R. .0242 Conley, Y. P. .0020 Connaboy, C. .0118, 0242 Connors, C. .0279, 0279 Conroy, D. .0532 Conroy, D. .0532 Conroy, D. A. .0513 Conroy, M. B. .0176 Cook, J. W. .0401, 0851 Cook, J. D. .0168, 0267 Coombes, B. J. .0173, 0173 Coon, D. W. .0826 Cooper, L. .0144, 1044 Cope, A. .0508 Corbett, Q. .0157 Cordoza, M. .0196 Cori, J. M .0286 Corona, F. E. .0252 Corser, B. .0745 Corsino, P. .0471 Cortés, C. .0249 Cortes, C. .0242 Costanzo, M. .0702 Costedoat, G. .0254 Cotter, V. .1137 Couderc, JP. .0571, 0571

Cox, R. C	
Crabtree, V. M	
Craggs, J.	
Crainiceanu, C	
Crane, S. C	
Crane, T. E	
Cravalho, P. F	
Crawford, M	
Crawford, M	
Crawford, M. R	
Creamer, J. L	
Crean, H. F	
Crean, H. J	
Creswell, D	
Creswell, K	
Cribbet, M	
Criley, C	
Criner, G. J	
Crisostomo, M. I	
Cristol, JP.	
Critton, J	
Crowley, S. J	
Cruickshank-Quinn, C	
Cruz, J	
Cruz Basilio, A	
Cruz Martir, L	
Cuamatzi Castelan, A	
Cuamatzi Castelan, A. S	
Cuamatzi-Castelan, A	
Cuellar, J. A	
Cui, L	
Culbreth, J. L	
Culnan, E	
Culpepper, L	
Culver, J. P	
Culver, M. N	
Culver, N	
Cummings, R	
Cunningham, J	
Cunningham, L	
Curtis, A	
Curtis, A. F.	
Cusmano, D	
Cuthbert, V	
Czajkowski, S	
Czeisler, C	
Czeisler, C. A	
Czeisler, E	

D

Daffre, C	0065, 1067, 1071, 1080, 1081, 1116
Daftary, A.	
Dagan, Y.	
D'Agata, M. N.	
Dagdag, A	
Daghlas, I	
Dahdah, M	
Dahlquist, D. T.	
Daigle, K	
Dailey, N. S 0014, 0070, 0079, 0	0080, 0227, 0305, 0305, 0314, 1158,
	1158, 1160

Dalal, L	
Daley, M. S	0055
Daley, R. T	1,0111
D'Almeida, V	0022
D'Almeida, V.	
D'Almeida, V.	
D'Almeida, V	
D'Alonzo, G	
Dang, R	
Daniel, L. C	
Danoff-Burg, S	
D'Antonio, B	·
Darby, J.	
Darchia, N	
Daripelli, S	
Darko, P.	
Dashti, H. S	
Daue, M. L.	
Dautovich, N.	
Dautovich, N. D	
Dauvilliers, Y Dauvilliers, Y0026, 0693, 0740, 0750, 0752, 0753, 076	
Dave, A	
Davenport, M	
Davenport, N	
Davey, M. J	·
Davies, C. R.	
Daviglus, M	
Daviglus, M. L.	
Davis, C. W	
Davis, E	
Davis, E. M.	
Davis, J.	
Davis, J. E	
Davison, D	
Davison, K.	
Dawson, S. C	
Day, A. J	
Day, Y	
Dayno, J. M	
De, A	0, 1060
Dean, A	
Dean, G	
Dean, G. E	
Debellemaniere, E	2,0546
Decker, A	0138
De Cuntis, I	8, 0808
Dedhia, R	0674
Deering, S	3, 0696
de Godoy, L. B.	0563
DeGraba, T	1103
Deighan, M. K	0100
Dela Cruz, A. V	0725
Del Brutto, O. H.	0805
Del Brutto, V. J.	0805
De Leon, L. F	0725
de Leon, M	0011
Delgadillo, M. E	0375
Delgado, GI.	
Deligiannidis, K.	
Dell'Acqua, R	0298
DelRosso, L	0905
DelRosso, L. M	

· · · · · · · · · · · · · · · · · · ·	
Deng, Y	
Denis, D	
Denison, S	
Denoncin, K.	
Deol, L. I	
De Ovando, C. I.	
Depner, C. M	
de Queiroz Campos, G	
Derby, C. A	
De Sá Souza, H	
	0184, 0218, 0233, 0279, 0279
	0560.0690
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589 051
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589 051
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589 0589 1051 1091 0317, 0317
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589 051 1091 0317, 0317 0312
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589 0589 1051 1091 0317, 0317 0312 0178, 0398, 1063, 1186
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 0589 0589 051 091 0317, 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 0589 0589 051 0317, 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0445
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 0589 0589 051 0317, 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0135
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 0633, 0634 0589 0589 051 0317, 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0445 0135 0331, 0331
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 0633, 0634 0589 0589 051 0317, 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0445 0135 0331, 0331 0692
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589 0589 0101, 0317 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0135 0135 0331, 0331 0692 1196, 1196
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589 0589 0101, 0317 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0445 0135 0135 0331, 0331 0692 1196, 1196 0069
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589 0589 0101, 0317 018, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0135 0135 0135 0131 0331 0692
de Zambotti, M	$\begin{array}{cccccccccccccccccccccccccccccccccccc$
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589 0589 0101, 0317 0317, 0317 0317, 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0445 0135 0135 0131 0682 0227, 0328 0437, 0449 0688
de Zambotti, M	$\begin{array}{cccccccccccccccccccccccccccccccccccc$
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 0633, 0634 0589 0589 0101 0317, 0317 0317, 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0445 0135 0135 031, 0331 0692 0692 0692 0196, 1196 0688 0009, 0009 0009, 0009 0060, 1100, 1100
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 1136, 1136 0589 0589 0101 0317, 0317 0317 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0445 0135 0135 0331, 0331 0692 0196, 1196 0692 0217, 0328 0437, 0449
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 0633, 0634 0589 0589 0101 0317, 0317 0317, 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0445 0135 0135 0331, 0331 0692 0196, 1196 0069 0217, 0328 0437, 0449 0688 0009, 0009 0060, 1100, 1100
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 0633, 0634 0589 0589 0101 0317 0317 0317 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0445 0135 0135 0331, 0331 0692 0692 0178, 0398, 1063, 1186 0692 0135 0135 0069 0688 0009, 0009 0060, 1100, 1100 0294, 0294
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193 0738 0101, 0391 0633, 0634 0633, 0634 0589 0589 0101 0317, 0317 0317, 0317 0312 0178, 0398, 1063, 1186 0225, 0384, 0533, 0533, 0540 0445 0135 0135 0331, 0331 0692 0196, 1196 0692 0437, 0449 0688 0009, 0009 0060, 1100, 1100 0294, 0294 0941, 0951
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193
de Zambotti, M	0482, 0794, 0794, 0916, 0916, 1193

SLEEP, Volume 43, Abstract Supplement, 2020

DiTomasso R A	
Doerflein Fulk, D. L.	
Doghramji, K.	
Dokkedal-Silva, V.	
Dolan, D.	
Dominguez, J.	
-	
2	
-	
/	
Dow, H. C	
Dowling, M	
Drake, C	
Drake, C.	
Drake, C. L.	0006, 0007, 0203, 0465, 0470, 0470
Drasher-Phillips, L.	
-	
Driscoll B I	
Drogou, C	
Drogou, C	
Drogou, C Drori, A D'Rozario, A	
Drogou, C Drori, A D'Rozario, A D'Rozario, A. L	
Drogou, C Drori, A D'Rozario, A D'Rozario, A. L Drummond, S. P	
Drogou, C Drori, A D'Rozario, A D'Rozario, A. L Drummond, S. P Dube, J	
Drogou, C	
Drogou, C Drori, A D'Rozario, A D'Rozario, A. L Drummond, S. P Dube, J Dubois-Comtois, K Dubourdeau, M	
Drogou, C Drori, A D'Rozario, A D'Rozario, A. L Drummond, S. P Dube, J Dubois-Comtois, K Dubourdeau, M Dubow, J	
Drogou, C Drori, A D'Rozario, A. L D'Rozario, A. L Drummond, S. P Dube, J Dubois-Comtois, K Dubourdeau, M Dubow, J Dubrovsky, B	
Drogou, C Drori, A D'Rozario, A. L D'Rozario, A. L Drummond, S. P Dube, J Dubois-Comtois, K Dubourdeau, M Dubow, J Dubrovsky, B DuBuc, K	
Drogou, C	$\begin{array}{c}$
Drogou, C	$\begin{array}{c}$
Drogou, C	$\begin{array}{c}$
Drogou, C	$\begin{array}{c} .0323\\ .0299\\ .0299\\ .0584\\ .1127, 1127\\ .0669, 0672\\ .0204\\ .0354\\ .0161\\ .0275, 0275\\ .0743, 0745, 0747\\ .0712, 0733, 0736\\ .0039, 0513, 0532\\ .1133\\ .00917\\ .0409, 0778, 1024\\ .0033, 0117, 0302, 0302, 0382, 1128\\ .0669\\ .0402\\ .0074\\ .0916, 0916\\ .0024\\ .0053\\ .0368\end{array}$
Drogou, C	$\begin{array}{c}$
Drogou, C	
Drogou, C	$\begin{array}{c}$
Drogou, C	
Drogou, C. Drori, A. D'Rozario, A. L. D'Rozario, A. L. D'Rozario, A. L. Drummond, S. P. Dube, J. Dubois-Comtois, K. Dubois-Comtois, K. Dubourdeau, M. Dubow, J. Duborovsky, B. DuBuc, K. Dulos, C. Duffett-Leger, L. Duffy, J. Duffy, S. Dugan, K. A. Dubar, S. B. Dunbar, S. B. Dunham, K. Dunhsm, K. Dunnyer, L. Dunnyer, K. Dunye-McCauley, K.	$\begin{array}{c}$

Duraccio, K. M
Durley, I
Dutcher, J
Dutra, M
Dutt, N
Dutta, R
Dykstra-Aiello, C. J
Dzierzewski, J
Dzierzewski, J. M

E

Eapen, B	
Eastman, C. I	
Eastwood, P. R.	
Eaton, G.	
Ebben, M	
e	
Elias, M. N	
Elias, R. M.	
Eliasson, A. H.	
Eliozishvili, M	
-	
-	
1	
Erblang, M.	

Erwin, J. A
Esbit, S
Esbit, S. L
Escourrou, P
e Silva, L
Espie, C. A
Espinoza, K
Estep, L
Etzion, S
Eun, H
Evans, K
Evans-Lindquist, M. K
Even Tsur, J
Everaert, K
Everhart, E
Everse, C
Evert, L
Eyal, S

F

-
Facer-Childs, E. R
Fakhary, M
Fakhoury, A
Faler, W. E
Falzon, L
FAN, JQ
Fan, J
Fan, Q
Fan, Z
Fan, Z
Fang, F
Fang, J
Fann, J. R
Fan Yun, L
Farber, N
Farquhar, W. B
Fearon, D
Feeley, C
Feemster, J
Feemster, J. C
Feemster, L. C
Fei, W
Feick, N. H
Feinaigle, P
Feinberg, I
Feinstein, L
Fekedulegn, D
Feldner, M
Felmingham, K
Felt, B
Feltch, C
Feng, G
Fenn, K. M
Fenton, M
Ferber, R
Fergason, K
Feria, C. S
Ferini Strambi, L
Ferini-Strambi, L
Fernandes, G. L
Fernandes, G. B
Fernandes, R. M

Fernandez-Mendoza, J	0319, 0457, 0458, 0476, 0506, 0585	
	0878, 0890, 0919, 0920, 0936, 1107	
Ferri, R		2, 1122
Ferziger, R		8, 1185
Fierro, A		0249
Fietze, I		0603
Figetakis, K		1190
Fiks, A. G), 0952
Filice, A		0526
Filippov, G		8,0474
Fillmore, P		0101
5		
-		
	.0057, 0057, 0583, 0583, 0740, 0752	
-		
· · · · · · · · · · · · · · · · · · ·		
		· · · · ·
,		
-		
,		·
,		
,		
,		
,		0806
	0.000	0017

SLEEP, Volume 43, Abstract Supplement, 2020

Foust, J. .0820 Fowler, L. .0264 Fox, S. V. .0432 Fragala, M. S. .1010 Frain, J. A. .1018
Frange, C
Franklin, M
Franzen, P. L
Frauscher, B
Frédéric, S
Freegard, M
Freeman, L. B
Freire, A. X
Fremouw, T
Frenia, D
Fridman, M
Friedman, A. L
Frisco, D. J
Froese, R. E
Fu, Y
Fu-Hsin, L
Fukuda, T
Fukumura, K
Fuller, P. M
Funderburk, J. S
Fung, C. H
Fung, S. J
Funk, B
Furgal, A

G

Gaba, A	l
Gabryelska, A)
Gaddameedhi, S	5
Gagnadoux, F	5
Gagnon, K	7
Gagnon, R	2
Gahan, L	1
Gajula, R. P	5
Gakwaya, S	5
Galaska, B	l
Galbiati, A	5
Galduroz, J. F	3
Gall, A. J)
Galli, O)
Gallo, L	3
Gallo, L. C	7
Gander, P. H)
Gandotra, K	5
Gannon, K	l
Gao, C	l
Gao, H	3
Gao, L)
Gao, X	7
Gao, Y	5
Gao, Z)
Garaulet, M	2
Garbazza, C	l
Garcell, A	2
Garcia, A	5
Garcia, J)
Garcia, J	3

Garcia, W	
Garcia-Borreguero, D	
Garcia-Hansen, V	6
Garcia-Molina, G	15
Gardiner, A	
Garland, S	3
Garland, S. N	-5
Garrison, M. M	19
Gaskell, M	12
Gaston, S. A	6
Gaudreault, P	12
Gaultney, J	6
GAURIAU, C	
Gauthier-Gagne, G	8
Gavidia, R	3
Ge, W	
Gehring, S	
Gehringer, B	
Gehrking, T	
Gehrman, P	
Gehrman, P. R	
Geil, E. S	
Geller, P. A	
Gencarelli, A. M	
genest, C	
Geng, E	
Geng, X	
Genova, H	
Geoca, A	3
George, C	
Geraci, C	
Gerald, L. B	
Gerashchenko, D	
Gerashchenko, L044	
Gerdes, L	
Germain, A	
Germany, R	
Gerstenslager, B	
Gerwell, K	
Gerwien, R	
Gestsdottir, S	
Gever, D. H	
Ghani, S	
Gharib, H	
Gharraf, H	
Gharraf, H. S	
Ghiani, C	
Ghilotti, F	
Ghorai, A	
Ghose, S	
Gialdi, G	
Giannasi, L	
Gibbons, B	
Gigic, B	
Gigic, B	
Gilbert, C	
Gilbertson, D	
Gill, J	
Gill, J. M	
Giller, J	
Gilles, A	
Gillingham, M	5

Gillow, G. M
Ginovker, A
Gislason, T
Gizewski, E. R
Glattard, N
Glavin, E
Glaze, D
Glickenstein, D. A
Glickman, G. L
Glidewell, R
Gliske, S. V
Glodzik, L
Gloria, R
Glos, M
Glymour, M
Glynn, E
Glynn, L. M
Gobbi, C
Godbout, R
Godin, R
Godoy, L. B
Goel, N
1020, 1100, 1100
Goh, D
Goldbart, A
Golden, E
Goldstein, C
Goldstein, M. R
Goldstein, T. R
Goldstein, T
Goldstone, A
Golkashani, H. A
Gomes, M
Gomes, M
Gomes, M
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .1144
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .1144 Gonzalez, K. T. .0607, 0607
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .1144
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .1144 Gonzalez, K. T. .0607, 0607
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .1144 Gonzalez, K. T. .0607, 0607 Goodman, M. O. .0363 Goodman, S. .0048
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .1144 Gonzalez, K. T. .0607, 0607 Goodman, M. O. .0363 Goodman, S. .0048 Goodrich, J. A. .0228, 0231
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 González, H. M. .0607, 0607, 0609 González, H. M. .0144 Gonzalez, K. T. .0607, 0607, 0607 Goodman, M. O. .0363 Goodman, S. .0048 Goodrich, J. A. .0228, 0231 Gooneratne, N. .0543, 0861, 1185
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .1144 Gonzalez, K. T. .0607, 0607 Goodman, M. O. .0363 Goodman, S. .0048 Goodrich, J. A. .0228, 0231
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 González, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodman, S. .0048 Goodrich, J. A. .0228, 0231 Gooneratne, N. .0543, 0861, 1185 Gopas, J. .0027
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodran, S. .0048 Goodrich, J. A. .0228, 0231 Gooneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .0149, 0389 González, K. T. .0607, 0607, 0607 Goodman, M. O. .0363 Goodran, S. .0048 Goodrich, J. A. .0228, 0231 Gooneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodran, S. .0048 Goodrich, J. A. .0228, 0231 Gooneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 González, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodman, S. .0048 Goodrich, J. A. .0228, 0231 Goneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gossard, T. .0817 Gosselin, N. .1133
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 González, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodman, S. .0048 Goodrich, J. A. .0228, 0231 Goneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gossard, T. .0817 Gosselin, N. .1133 Gottlieb, D. J. .0419
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. .0149, 0389 González, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodrann, S. .0048 Goodrich, J. A. .0228, 0231 Goneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gossard, T. .0817 Gosselin, N. .1133 Gottlieb, D. J. .0419
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. D. .0149, 0389 González, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodrann, S. .0048 Goodrich, J. A. .0228, 0231 Goneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gossard, T. .0817 Gosselin, N. .1133 Gottlieb, D. J. .0419 Gottlieb, E. W. .0420 Gould, J. .0038, 0081
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. .0149, 0389 González, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodrann, S. .0048 Goodrich, J. A. .0228, 0231 Goneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gossard, T. .0817 Gosselin, N. .1133 Gottlieb, D. J. .0419
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzales, A. .0551, 0551 Gonzalez, A. .0553, 0551 Gonzalez, B. .1053 Gonzalez, H. .00607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodran, M. O. .0363 Goodrich, J. A. .0228, 0231 Gooreratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gosselin, N. .1133 Gottlieb, D. J. .0419 Gottlieb, E. W. .0420 Gould, R. .0038, 0081
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. .00607, 0607 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodman, S. .0048 Goodrich, J. A. .0228, 0231 Goneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gosselin, N. .1133 Gottlieb, D. J. .0419 Gottlieb, E. W. .0420 Gould, J. .0038, 0081 Gould, R. .0989 Gould, R. A. .0946
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. .0169 Gonzalez, H.M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodman, S. .0048 Goodrich, J. A. .0228, 0231 Goneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gossard, T. .0817 Gosselin, N. .1133 Gottlieb, D. J. .0419 Gottlieb, E. W. .0420 Gould, J. .0038, 0081 Gould, R. .0989 Gould, R. A. .0946 Gouws, A. D. .0086
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. .00607, 0607 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Goodman, S. .0048 Goodrich, J. A. .0228, 0231 Goneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gosselin, N. .1133 Gottlieb, D. J. .0419 Gottlieb, E. W. .0420 Gould, J. .0038, 0081 Gould, R. .0989 Gould, R. A. .0946
Gomes, M.
Gomes, M.
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Gooreratne, N. .0228, 0231 Gooneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gossard, T. .0817 Gosselin, N. .1133 Gottlieb, D. J. .0419 Gottlieb, E. W. .0420 Gould, J. .0038, 0081 Gould, R. .0989 Gould, R. .0989 Gould, R. A. .0946 Gouyal, V. .0759, 0760 Gozal, D. .06680, 0792, 1006
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzales, A. .0551, 0551 Gonzalez, A. .0551, 0551 Gonzalez, B. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Gooreatne, N. .0228, 0231 Gooneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gossard, T. .0817 Gosselin, N. .1133 Gottlieb, D. J. .0419 Gottlieb, E. W. .0420 Gould, J. .0038, 0081 Gould, R. .0989 Gould, R. A. .0946 Gouws, A. D. .0086 Goyal, W. .0759, 0760 Gozal, D. .06680, 0792, 1006 Gozar, A. .0013
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzalez, A. .0551, 0551 Gonzalez, B. .1053 Gonzalez, B. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Gooreratne, N. .0228, 0231 Gooneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gossard, T. .0817 Gosselin, N. .1133 Gottlieb, D. J. .0419 Gottlieb, E. W. .0420 Gould, J. .0038, 0081 Gould, R. .0989 Gould, R. .0989 Gould, R. A. .0946 Gouyal, V. .0759, 0760 Gozal, D. .06680, 0792, 1006
Gomes, M. .0657, 0804 GOMEZ-MERINO, D. .1042 Gong, N. N. .0069 Gonsalves, V. S. .0977 Gonzales, H. .0423 Gonzales, A. .0551, 0551 Gonzalez, A. .0551, 0551 Gonzalez, B. .0149, 0389 Gonzalez, H. M. .0607, 0607, 0609 González, H. M. .0607, 0607, 0607 Goodman, M. O. .0363 Gooreatne, N. .0228, 0231 Gooneratne, N. .0543, 0861, 1185 Gopas, J. .0027 Gordon, C. .0819 Gossard, T. .0817 Gosselin, N. .1133 Gottlieb, D. J. .0419 Gottlieb, E. W. .0420 Gould, J. .0038, 0081 Gould, R. .0989 Gould, R. A. .0946 Gouws, A. D. .0086 Goyal, W. .0759, 0760 Gozal, D. .06680, 0792, 1006 Gozar, A. .0013

Grandner, M
Grandner, M. A 0013, 0014, 0038, 0070, 0079, 0080, 0081, 0082,
0120, 0140, 0226, 0227, 0232, 0235, 0236, 0240, 0241, 0243, 0263,
0305, 0305, 0307, 0314, 0315, 0316, 0365, 0372, 0373, 0374, 0375,
0376, 0404, 0405, 0406, 0408, 0464, 0543, 0544, 0553, 0865, 1053,
1077, 1093, 1095, 1096, 1108, 1158, 1158, 1160, 1174, 1187
Granizo, J
Grant, L. K
Grant, R. W
Grant, S. F
Grassot, J
Gratsia, S
Green, A
Green, K
Greenberg, H
Greenberg, P
Greenfeld, M
Greenlund, K. J
Gregory, K
Grewal, R
Grey, M
Griesbach, G. S
Griffin, K0150, 0280
Griggs, S
Grigsby-Toussaint, D
Grill, J. D
Grimaldi, D
Grimaldo, D
Grinberg, A
Gronli, J
Grosicki, G. J
Gross, J. J
Gross, M
Gross, T
Gross, Y
Groton, D
Gruber, R
Grunstein, R. R
Grunstein, R. R
Grunvald, E
Gu, F
Gu, J
GU, W
Gualco, S. J
GUAN, J
Guan, L
Gudmundsdottir, S. L
Guedes, V
Guedes, V. A
Guerriero, R
Gui, Y
Guice, J. O
Guillard, M
Guillot, A
Guimaraes, T. M
Guimarães, T. M
Guimaraes, T. M
Gulati, G
Gummalla, P
Gunaratnam, B
Gundersen, H. S
Gunduz-Bruce, H

SLEEP, Volume 43, Abstract Supplement, 2020

Gunn, H. E
Gunnlaugsson, E
Gunstad, J
Guo, N
Gupta, A
Gupta, D
Gupta, G
Gupta, M. A
Gupta, S
Gurung, P
Gutierrez, R. L
Guttesen, A. V
Guttmann, C. R
Guzman, D
Gyamfi, L

H

	0058, 0233, 0275, 0275, 0279, 0279
Hackett, P. H	
Haddad, H	
Hagen, E. H	
Hagen, E. W	
Haghayegh, S	
Hahn, S	
Hakopian, S	
Halaby, L. M.	
. 0013, 0241, 0243, 0360, 03	60, 0376, 0406, 0408, 0544, 0865, 1095,
Hale, W.	1108, 1179, 1181
	009, 0009, 0588, 0623, 0716, 0761, 0765
2	
	0121, 0208, 0274, 0297, 0730, 1038
Hansen, S. L.	

Hantragool, S	
Hanyu, A	
Hao, W	.0727
Hao, Y	.0626
Haraldsson, H.	.0337
Hardikar, S.	.0044
Hardy, W	
Harkins, E.	
Harmon, E	
Haroutonian, C.	
Harris, M	
Harris, S	
Harrison, E	
Harrison, E. M.	
Harrison, K. A	
Harry-Hernandez, S.	
Harsh, J	0220
Harsh, J. R	
Harsn, J. K	
Hartescu, I.	
Hartman, A. R.	
Hartman, A. G.	
Hartmann, S	
Hartstein, L. E.	
Harvey, A. C.	
Harvey, A.	
Hasanaj, K	
Hasegawa, S.	
Hasler, B.	
Hasler, B. P	
Hassan, F.	.1014
Hassirim, Z	
Hassler, A. N.	.0224
Hassler, A. N	.0224 .0343
Hassler, A. N	.0224 .0343 .0162
Hassler, A. N Hatcher, K. M Hatfield, A Hawkins, S. M	.0224 .0343 .0162 .0876
Hassler, A. N	.0224 .0343 .0162 .0876
Hassler, A. N Hatcher, K. M Hatfield, A Hawkins, S. M	.0224 .0343 .0162 .0876 .1146
Hassler, A. N	.0224 .0343 .0162 .0876 .1146 1049 .0866
Hassler, A. N	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502
Hassler, A. N	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502
Hassler, A. N	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502 0936
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. .0554, 0554, 1047, Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920,	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502 0936 .0605
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. .0554, 0554, 1047, Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. He, M. .0437,	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502 0936 .0605 0449
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. .0554, 0554, 1047, Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. He, M. .0437, Heaps, E.	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502 0936 .0605 0449 .0929
Hassler, A. N. Hatcher, K. M. Hatther, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Haynes, P. L. .0554, 0554, 1047, Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. He, M. .0437, Heavner, J. J.	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502 0936 .0605 0449 .0929 .1167
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. Heavner, J. J. Heavner, M. S.	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502 0936 .0605 0449 .0929 .1167 .1167
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. Heavner, J. J. Heavner, M. S. Hébert, M.	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502 0936 .0605 0449 .0929 .1167 .1167 .0777
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Maynes, P. L. .0554, 0554, 1047, Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. Heavner, J. J. Heavner, J. J. Heavner, M. S. Hébert, M. Heckaman, E.	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502 0936 .0605 0449 .0929 .1167 .1167 .0777 .0592
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Maynes, P. L. .0554, 0554, 1047, Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. He, M. .0437, Heavner, J. J. Heavner, M. S. Hébert, M. Heckaman, E. Hedden, T.	0224 0343 0162 0876 1049 08866 0502 0936 0605 0449 0.929 1.1167 0.777 0.592 0.0144
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Maynes, P. L. Meavner, J. J. Heavner, M. S. Hébert, M. Heckaman, E. Hedden, T. Hedker, D.	0224 0343 0162 0876 1049 0866 0502 0936 0605 0449 0929 1.1167 0.777 0.592 0.0144
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. He, M. .0437, Heaps, E. Heavner, J. J. Hebert, M. Heckaman, E. Hedden, T. Hedge, M.	.0224 .0343 .0162 .0876 .1146 .1146 .049 .0866 .0502 .0936 .0605 .0449 .0929 .1167 .1167 .0777 .0592 .0144 .1151 .0165
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. He, M. .0437, Heaps, E. Heavner, J. J. Heker, M. Hedden, T. Hedden, T. Hedge, M. Hedner, J. .0641, 0693,	.0224 .0343 .0162 .0876 .1146 1049 .0866 0502 0936 .0605 .0449 .0929 .1167 .1167 .0777 .0592 .0144 .1151 .0165 0772
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. He, M. .0437, Heaps, E. Heavner, J. J. Hedener, M. S. Hedden, T. Hedden, T. Hedge, M. Hedner, J. .0641, 0693,	0224 0343 0162 0876 1146 1049 08866 0502 0936 0605 0449 0929 1167 1.1167 0.0777 .0144 1.151 0.0165 0772 0.010
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. He, M. .0437, Heaps, E. Heavner, J. J. Hebert, M. Heckaman, E. Hedden, T. Hedge, M. Hedrer, J. .0641, 0693, Heidbreder, A. Heinzer, R.	0224 0343 0162 0876 1146 1049 08866 0502 0936 0605 0449 0929 1167 1167 00777 00592 00144 1151 0165 0772 0010 0726
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0198, 0500, 0502, He, F. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. He, M. .0437, Heayner, J. Heavner, J. J. Heavner, M. S. Hébert, M. Hedden, T. Hedden, T. Hedge, M. Hedge, M. Heidbreder, A. Heinzer, R. .0697, 0697, 0697, 0699, Heiser, C.	0224 0343 0162 0876 1146 1049 08866 0502 0936 0605 0449 0929 1167 1167 0.0777 0.0592 0.0144 1151 0165 0772 0.0010 0726 0.0637
Hassler, A. N. Hatcher, K. M. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Haynes, P. L. Hays, C. C. He, E. 0198, 0500, 0502, He, F. He, K. He, M. He, M. Heavner, J. J. Heavner, M. S. Hedden, T. Hedden, T. Hedge, M. Heddrer, J. Heidbreder, A. Heinzer, R. 0697, 0697, 0699, Heiss, L.	0224 0343 0162 0876 1146 1049 0886 0502 0936 0605 0449 0929 1167 1167 0.0592 00144 1151 00752 00144 1151 00726 0010 0726 0.0637 0.0099
Hassler, A. N. Hatcher, K. M. Hatfield, A. Hawkins, S. M. Hayes, M. Hays, C. C. He, E. .0198, 0500, 0502, He, F. .0198, 0500, 0502, He, F. .0198, 0500, 0502, He, F. .0319, 0457, 0585, 0878, 0890, 0919, 0920, He, K. He, M. Heavner, F. .0437, Heasp, E. Heavner, J. J. Heavner, M. S. Hébert, M. Heckaman, E. Hedden, T. Hedder, D. Heddrer, J. Heidbreder, A. Heidbreder, A. Heinzer, R. .0697, 0697, 0699, Heiss, L. Helgadóttir, H. .0447,	0224 0343 0162 0876 1146 1049 0502 0936 0605 0449 0929 1167 0777 0.0592 0.0144 1.1151 0.0165 0772 0.010 0726 0.0637 0.0099 0448
Hassler, A. N	0224 0343 0162 0876 1146 1049 0502 0936 0605 0449 0929 1167 0777 0.0792 0144 1.1151 00165 0772 0010 0726 0.0637 0.0099 0448 0.853
Hassler, A. N	0224 0343 0162 0876 1146 1049 0502 0936 0605 0449 0929 1167 0777 0.0592 0144 1151 00165 0772 0010 0726 0.0637 0.0099 0448 0.0853 0.086
Hassler, A. N	0224 0343 0162 0876 1146 1049 0502 0936 0605 0449 0929 1167 0777 0.0592 0144 1151 0075 0772 0010 0726 0010 0726 0037 0099 0448 0.853 0.086 0194
Hassler, A. N	0224 0343 0162 0876 1146 1049 0502 0936 0605 0449 0929 1167 1167 0777 0.0592 0144 1151 0165 0772 0.010 0726 0.0637 0.0099 0448 0.0853 0.0866 0194 0519

Henzel, M. K	
Hermann, V.	
Hernandez, B	0652, 0781, 0781
Hernandez, P.	
Hernandez, R.	
Hernandez-Cardenache, R.	
Herring, W	
Herringa, R.	
Hershner, S	
Hershner, S. D	
Hertzberg, V. S.	
Herzig, M. X	
Hevener, W	
Hewitt, J.	
Hickey, J.	
Hickey, M. G.	
Hicks, A	
Higgins, J	
Highland, K.	
Hikichi, H	
Hilditch, C	
Hilditch, C. J.	
Hill, E. A	
Hill, N	
Hillary, R	
Hillman, D. R.	
Hiltner, R. K.	
Himali, J. J.	
Himbert, C.	
Hines, A	
Hino, N	
Hinson, J. M.	0121, 0125, 0301
Hinton, J. A.	
Hirai, N	
Hirose, M.	
Hirotsu, C.	
Hiroyama, S	
Hirsch, D. A	
Hiscock, H	
Hisler, G.	0216, 0276
Но, Ј. Q	
Но, S. S.	
Hockmeyer, T. R.	
-	
Hoddy, K. K.	
Hodgkins, P.	
Hoff, R	
Hoffman, J	
Hoffman, M	
Hoffmann, C. M.	
Hof zum Berge, A.	
-	
Högl, B.	
Holbert, C	
Holingue, C	
Holliday, M	0228, 0231
Hollimon, L	
Hollingshead, K.	
Holm, K	
Holmedahl, N	
Holmes, J. F.	
Holthouser, S	
Holty, JE	0652, 0781, 0781
Holty, JE.C.	1069, 1069
Hom, C	
· · · · · · · · · · · · · · · · · · ·	

	0921, 0924
Hong, J	
Hong, J	0845
Hong, S	0935
Hong, SC	0387
Honn, K. A	0301, 0308
Honomichl, R.	
Horgan, S.	
Horne, R. S	
Horvat, M.	
Hosamane, N	
Hossain, M. M.	
Hossain, S.	
Houser, L	
Howard, K	
Howard, L. J	
Howard, L. J	
Howard, M. E	
Howe, G	
Howell, M	
Howell, M.	
Howell, S	
Howell, S. N.	
Howladar, A	
Hoyle, S. P	
Hsia, J	
Hsiao, JR	
Hsieh, JC	
Hu, K0032, 0259, 1135, 1135,	1141, 1159
Hua, R. X	0126
Huang, A	1017
Huang, F	0656,0701
Huang, G	.0898, 0898
Huang, G	0843, 0844
Huang, J	0843, 0844 .0752, 0753
Huang, J	0843, 0844 .0752, 0753 0535
Huang, J	0843, 0844 .0752, 0753 0535 0615
Huang, J	0843, 0844 .0752, 0753 0535 0615 .0363, 1007
Huang, J	0843, 0844 .0752, 0753 0535 0615 .0363, 1007 0580
Huang, J	0843, 0844 .0752, 0753 0535 0615 .0363, 1007 0580 0995
Huang, J	0843, 0844 0752, 0753 0535 0615 .0363, 1007 0580 0995 0603
Huang, J	0843, 0844 0752, 0753 0535 0615 .0363, 1007 0580 0580 0995 0603 1134
Huang, J	0843, 0844 0752, 0753 0535 0615 0363, 1007 0580 0995 0603 1134 0026
Huang, J	0843, 0844 .0752, 0753 0615 .0363, 1007 0580 0995 0603 1134 0026 1074
Huang, J	0843, 0844 .0752, 0753 0615 .0363, 1007 0580 0603 0603 1134 0026 1074 0628
Huang, J	0843, 0844 0752, 0753 0615 0363, 1007 0580 0603 0603 1134 0026 1074 0628 0121, 0208
Huang, J	0843, 0844 0752, 0753 0615 0363, 1007 0580 0603 0603 026 0026 0628 .0121, 0208 1015
Huang, J	0843, 0844 0752, 0753 0615 .0363, 1007 0580 0995 0603 1134 0026 1074 0628 .0121, 0208 1015 0883
Huang, J	0843, 0844 0752, 0753 0615 .0363, 1007 0580 0995 0603 1134 0026 1074 0628 .0121, 0208 1015 0883 0952
Huang, J	0843, 0844 0752, 0753 0535 0615 .0363, 1007 0580 0995 0603 1134 0026 0628 .0121, 0208 0615 0883 0952 .0290, 0290
Huang, J	0843, 0844 0752, 0753 0535 0615 .0363, 1007 0580 0995 0603 1134 0026 1074 0628 .0121, 0208 1015 0883 0952 .0290, 0290 1165
Huang, J	0843, 0844 0752, 0753 0535 0615 .0363, 1007 0580 0995 0603 1134 0026 1074 0628 .0121, 0208 0952 .0290, 0290 1165 1074
Huang, J	0843, 0844 0752, 0753 0535 0615 .0363, 1007 0580 0995 0603 1134 0026 1074 0628 .0121, 0208 0952 .0290, 0290 1165 074 .0001, 0001
Huang, J. .0843, Huang, L. .0843, Huang, L.	0843, 0844 0752, 0753 0535 0615 .0363, 1007 0580 0995 0603 1034 026 1074 0628 .0121, 0208 0952 .0290, 0290 1165 074 .0001, 0001 0602
Huang, J. .0843, Huang, L. .0843, Huang, L.	0843, 0844 0752, 0753 0535 0615 .0363, 1007 0580 0995 0603 1034 026 1074 0628 .0121, 0208 0952 .0290, 0290 1165 0883 0952 .0290, 0290 1165 0602 0601
Huang, J	0843, 0844 0752, 0753 0535 0615 .0363, 1007 0580 0995 0603 1034 026 1074 0628 .0121, 0208 0952 .0290, 0290 1165 0883 0952 .0290, 0290 1165 0602 0601
Huang, J. .0843, Huang, L. .0843, Huang, L.	0843, 0844 0752, 0753 0615 0363, 1007 0580 0995 0095 0603 1134 026 1074 0628 .0121, 0208 1015 0952 .0290, 0290 165 1074 .0001, 0001 0602 0671 0834
Huang, J. .0843, Huang, L. .0843, Huang, L.	0843, 0844 0752, 0753 0615 0363, 1007 0580 0995 0603 1134 026 1074 0628 .0121, 0208 1015 0952 .0290, 0290 1165 0612 0952 0628 0952 0952 0952 0952 0952 0615 003 026 063 003
Huang, J. .0843, Huang, L. .0843, Huang, L.	0843, 0844 0752, 0753 0615 0363, 1007 0580 0995 0603 1134 026 1074 0628 .0121, 0208 1015 0952 .0290, 0290 1165 0612 0952 .0290, 0290 1165 0615 0637 0637 0637 0731
Huang, J. .0843, Huang, L. .0843, Huang, L.	0843, 0844 0752, 0753 0615 0363, 1007 0580 0995 0603 1134 026 1074 0628 .0121, 0208 1015 0883 0952 .0290, 0290 1165 0615 0615 0833 026 0628 0952 0952 0615 0637 0637 0637 0731 0731 0731
Huang, J. .0843, Huang, L. .0843, Huang, L.	0843, 0844 0752, 0753 0615 .0363, 1007 0580 0995 0603 1134 026 1074 0628 .0121, 0208 1015 0883 0952 .0290, 0290 105 0671 0637 0637 0731 1167 .0184, 0218
Huang, J. .0843, Huang, L. .0843, Huang, L.	0843, 0844 0752, 0753 0615 0363, 1007 0580 0995 0603 1134 0026 1074 0628 0121, 0208 1015 0883 0952 .0290, 0290 1165 0671 0834 0637 0731 0167 .0184, 0218 0223
Huang, J. .0843, Huang, L. .0843, Huang, L.	0843, 0844 0752, 0753 0615 0363, 1007 0580 0995 0603 1134 026 1074 0628 .0121, 0208 1015 0883 0952 .0290, 0290 1165 0671 0637 0637 0731 0637 0731 0167 0223 0298

SLEEP, Volume 43, Abstract Supplement, 2020

Hutzelmann, J.	0487, 0488
Hwangbo, Y	0935
Hyman, D	0751

I

Iakoubova, O. A
Iakovou, A
Iao, S
Ibarra-Hernandez, J. M
Iber, C
Ibrahim, S
Ida, H
Ihemeremadu, N
Ikeda, A
Ikeda, K
Im, H
Im, K
Imadera, Y
Imanishi, A
Imes, C
Imes, C. C
Immen, R
Inamac, A
Inge, T. H
Ingram, D
Ingram, D. G
Inoue, Y
110 ac, 111 111 111 111 111 111 111 111 111 1
Inslicht, S
Inslicht, S
Inslicht, S
Inslicht, S. .1074 Iranmanesh, A. .0131 Irfan, M. .1169
Inslicht, S. .1074 Iranmanesh, A. .0131 Irfan, M. .1169 Irish, L. A. .0247
Inslicht, S. .1074 Iranmanesh, A. .0131 Irfan, M. .1169 Irish, L. A. .0247 Ishii, T. .0147
Inslicht, S. 1074 Iranmanesh, A. .0131 Irfan, M. .1169 Irish, L. A. .0247 Ishii, T. .0147 Ishikawa, T. .0141, 0142
Inslicht, S. 1074 Iranmanesh, A. 0131 Irfan, M. 1169 Irish, L. A. 0247 Ishii, T. 0147 Ishikawa, T. 0141, 0142 Isidean, S. D. 1028
Inslicht, S. 1074 Iranmanesh, A. 0131 Irfan, M. 1169 Irish, L. A. 0247 Ishii, T. 0147 Ishikawa, T. 0141, 0142 Isidean, S. D. 1028 Ismail, M. 0794, 0794
Inslicht, S. 1074 Iranmanesh, A. 0131 Irfan, M. 1169 Irish, L. A. 0247 Ishii, T. 0147 Ishikawa, T. 0141, 0142 Isidean, S. D. 1028 Ismail, M. 0794, 0794 Ito, H. 0618
Inslicht, S. 1074 Iranmanesh, A. 0131 Irfan, M. 1169 Irish, L. A. .0247 Ishii, T. .0141, 0142 Isdean, S. D. .0128 Ismail, M. .0794, 0794 Ito, H. .0618 Ito, K. .0453, 0491 Ito, Y. .0575 Itoh, T. .0001, 0001
Inslicht, S. 1074 Iranmanesh, A. 0131 Irfan, M. 1169 Irish, L. A. 0247 Ishii, T. 0141, 0142 Isdean, S. D. 1028 Ismail, M. 0794, 0794 Ito, H. 0618 Ito, X. 0453, 0491 Ito, Y. 0575 Itoh, T. 0001, 0001 Ito Uemura, S. 0757
Inslicht, S. 1074 Iranmanesh, A. 0131 Irfan, M. 1169 Irish, L. A. .0247 Ishii, T. .0141 Ishikawa, T. .0141, 0142 Isidean, S. D. .1028 Ismail, M. .0794, 0794 Ito, H. .0618 Ito, K. .0453, 0491 Ito, Y. .0575 Itoh, T. .0001, 0001 Ito Uemura, S. .0757 Ivers, H. .0511, 0512, 0536
Inslicht, S. 1074 Iranmanesh, A. 0131 Irfan, M. 1169 Irish, L. A. .0247 Ishii, T. .0141, 0142 Isdean, S. D. .0128 Ismail, M. .0794, 0794 Ito, H. .0618 Ito, X. .0453, 0491 Ito, Y. .0575 Itoh, T. .0001, 0001 Ito Uemura, S. .0757 Ivers, H. .0511, 0512, 0536 Ives, S. J. .0134
Inslicht, S. 1074 Iranmanesh, A. 0131 Irfan, M. 1169 Irish, L. A. .0247 Ishii, T. .0141, 0142 Isdean, S. D. .0128 Ismail, M. .0794, 0794 Ito, H. .0618 Ito, X. .0453, 0491 Ito, Y. .0575 Itoh, T. .0001, 0001 Ito Uemura, S. .0757 Ivers, H. .0511, 0512, 0536 Ives, S. J. .0134
Inslicht, S. 1074 Iranmanesh, A. 0131 Irfan, M. 1169 Irish, L. A. .0247 Ishii, T. .0141 Ishikawa, T. .0141, 0142 Isidean, S. D. .1028 Ismail, M. .0794, 0794 Ito, H. .0618 Ito, K. .0453, 0491 Ito, Y. .0575 Itoh, T. .0001, 0001 Ito Uemura, S. .0757 Ivers, H. .0511, 0512, 0536

J

Jablin, T. .0338 Jack, C. R. .0355 Jackson, C. L. .0361, 0366, 0367, 0367, 0988, 1016, 1016 Jackson, M. L. .0926, 1171 Jackson, W. .0361 Jaffe, F. .0567, 0610, 1048 Jahani Kondori, M. .0738 Jain, S. V. .1172 Jain, V. .0782, 0783 Jaioo, A .003
Jain, V
Jakubowski, K. P
Jambhekar, S

Jamison, A. L
Janevski, K
Jang, E
Jang, H
Jang, JW
Janney, R
Jansen, E
Jansen, E. C
Janssen, H
Jardin, P. B
Jaroenying, R
Jarrin, D. C
Jaskiw, G. E
Jasko, J
Jasko, J. G
Jasper, A
Jaussent, I
Javaheri, S
Javaheri, S
Jayarajan, P
Jean-Louis, G
Jean-Louis, G
Jean-Louis, G 0011, 0232, 0235, 0372, 0373, 0374, 0376, 0377
0404, 0406, 0410, 0621, 0863, 0964, 0995, 1046, 1046, 1058, 1058
1062, 1062, 1082, 1089, 1102, 1113, 1114, 1117, 1150, 1153, 1179
1181, 1189, 1212, 1213
Jeanne, H
Jean-Pierre, P
Jecmen, D
Jee Hyun, K
Jellis, C
Jen, R
Jenkins, D
Jenks, C
Jennum, P0451, 1208
Jeon, B
Jeon, J
Jeon, S
Jeon, S
Jeoung, S
C .
Jetta, S
Jhoo, J
Ji, M
Ji, X0400
Ji, X
Jiang, F
Jiao, J. L
Jiao, L
Jiao, N
Jimenez, L
Jin, P
Jobe, S
Jobe, S. L
Joffe, H
Johannsson, E
John, S. E
Johnson, A
Johnson, A. M
Johnson, A. K
Johnson, A. H
Johnson, A. H
Johnson, D

Johnson, R. L
Johnson-Akeju, O
Jones, A. M
Jones, A
Jones, A. C
Jones, C
Jones, C. W
Jones, S
Jones, S. G
Jónsson, S. Æ
Joo, E
Jordan, K
Jorge, M. C
Josephson, K
Josephson, K. R
Joshi, M. J
Jovet, G
Joyce, D. S
Jubran, A0734
Jun, T
Junco, B
Jung, C. M
Junge, M
Jungquist, C

K

Kaali, S
Kaar, J. L
Kadotani, H
Kaffashi, F
Kahane, A
Kaiser, U. B
Kaizer, A
Kaizi-Lutu, M
Kajiyama, Y
Kak, I
Kalaydjian, S
Kales, S. N
Kallweit, U
Kalmbach, D
Kalmbach, D. A
Kalra, M
Kalyan, B
Kam, K
Kamarck, T
Kamarck, T
Kambe, D
Kambe, D
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757 Kandil, M. .0795
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757 Kandil, M. .0795 Kanes, S. J. .0535 Kaneshiro, K. N. .0893 Kang, H. .0840
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757 Kandil, M. .0795 Kanes, S. J. .0535 Kaneshiro, K. N. .0893 Kang, H. .0840 Kang, J. .0073
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757 Kandil, M. .0795 Kanes, S. J. .0535 Kaneshiro, K. N. .0893 Kang, H. .0840 Kang, J. .0073 Kang, SG. .0073
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757 Kandil, M. .0795 Kanes, S. J. .0535 Kaneshiro, K. N. .0893 Kang, H. .0073 Kang, SG. .0073 Kansagra, S. .0950
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757 Kandil, M. .0795 Kanes, S. J. .0535 Kaneshiro, K. N. .0893 Kang, H. .0073 Kang, SG. .0073 Kansagra, S. .0950 Kantner, C. .1167
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757 Kandil, M. .0795 Kanesh, S. J. .0535 Kaneshiro, K. N. .0893 Kang, H. .0073 Kang, SG. .0073 Kansagra, S. .0950 Kantner, C. .1167 Kao, CH. .0669
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757 Kandil, M. .0795 Kanesh, S. J. .0535 Kaneshiro, K. N. .0893 Kang, H. .0073 Kang, SG. .0073 Kansagra, S. .0950 Kantner, C. .1167 Kao, CH. .0669 Kapella, M. .0529
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757 Kandil, M. .0795 Kanesh, S. J. .0535 Kaneshiro, K. N. .0893 Kang, H. .0073 Kang, SG. .0073 Kansagra, S. .0950 Kantner, C. .1167 Kao, CH. .0669 Kapella, M. .0529 Kapil, R. .0198, 0500, 0502, 0502
Kambe, D. .0004, 0146 Kampakis, S. .1201, 1201 Kamuju, V. .0072 Kanady, J. C. .0503 Kanbayashi, T. .0518, 0757 Kandil, M. .0795 Kanesh, S. J. .0535 Kaneshiro, K. N. .0893 Kang, H. .0073 Kang, SG. .0073 Kansagra, S. .0950 Kantner, C. .1167 Kao, CH. .0669 Kapella, M. .0529

Kapoor, A	
Karhu, T	
Kark, S. M	
Karlen, W	
Karlson, E	
Karp, H	
Karroum, E. G.	
Kashyap, S.	
Kaskie, R	
Kasper, J.	
Kassner, A	
Kataria, L.	
Kato, M	
-	
-	
Keane, T.,	
Keele, L	
Keele, L	
Keele, L Keenan, B Keenan, B. T	
Keele, L Keenan, B Keenan, B. T	
Keele, L Keenan, B	
Keele, L Keenan, B	
Keele, L Keenan, B. Keenan, B. T. .0053 Keens, T. G. Kellar, D. C. Keller, C. Keller, T.	
Keele, L Keenan, B. Keenan, B. T. .0053 Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A.	
Keele, L Keenan, B. Keenan, B. T. .0053 Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A. Kelly, K.	
Keele, L Keenan, B. Keenan, B. T. .0053 Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A. Kelly, K. Kelly, M.	
Keele, L Keenan, B. Keenan, B. T. .0053 Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Kelly, M. R.	
Keele, L Keenan, B. Keenan, B. T. .0053 Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Kelly, M. R. Kelly, M. B.	
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Kelly, M. R. Kelly, M. B. Kenner, G.	
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Kelly, M. R. Kelly, M. B. Kenner, G. Kempke, N.	
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Kelly, M. R. Kelly, M. B. Kenner, G. Kempke, N. Kenney, K.	$\begin{array}{c} 1073\\0054, 0054\\0237, 0424, 0562, 1057\\0874, 0054, 0054, 0569, 0613, 0691, 1184\\0874, 0887\\0523, 0541, 0542\\0244, 0255\\0596\\0896\\0225, 0384, 0398, 1063\\0581\\0131, 0467, 0475, 0537\\0160\\0529\\0583, 0583\\0046, 0046, 0416, 1155, 1155\end{array}$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Kelly, M. R. Kelly, M. B. Kemner, G. Kemney, K. Kenney, K.	$\begin{array}{c} 1073 \\$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Kelly, M. R. Kelly, M. B. Kemner, G. Kenney, K. Kenney, K. Kenney, K. Kenniger, E. A. Kent, D.	$\begin{array}{c} 1073 \\$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A. Kelly, A. Kelly, M. Kethy, M. Kethy, M. Kethy, M. Kethy, M. Kempke, N. Kenney, K. Kensinger, E. Kent, D. Keshavarzian, A.	$\begin{array}{c} 1073 \\$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, C. Keller, R. Kelly, A. Kelly, M. Keelly, M. Keelly, M. Keelly, M. Kempke, N. Kensinger, E. Kensinger, E. Keshavarzian, A. Ketchum, J.	$\begin{array}{c} 1073 \\$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Kellar, D. C. Keller, T. Kelly, A. Kelly, A. Kelly, M. Keenye, N. Kenney, K. Kensinger, E. Keshavarzian, A. Kezirian, E.	$\begin{array}{c} 1073 \\$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Kellar, D. C. Keller, T. Kelly, A. Kelly, A. Kelly, M. Keelly, M. Keelly, M. Keelly, M. Keelly, M. Kenney, K. Kensinger, E. Keshavarzian, A. Kezirian, E. Khader, S.	$\begin{array}{c} 1073 \\$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Kellar, D. C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Kenney, G. Kenney, K. Kensinger, E. A. Keshavarzian, A. Kezirian, E. Khader, W. S.	$\begin{array}{c} 1073 \\0054, 0054 \\0237, 0424, 0562, 1057 \\0874, 0054, 0054, 0569, 0613, 0691, 1184 \\0874, 0887 \\0874, 0887 \\0232, 0541, 0542 \\0244, 0255 \\0596 \\0896 \\0225, 0384, 0398, 1063 \\0581 \\0131, 0467, 0475, 0537 \\0160 \\0529 \\0583, 0583 \\0109, 0109, 0111, 0111, 0311 \\0681 \\0780 \\0606, 1136, 1136, 1177, 1177 \\0637 \\0374 \\0232 \end{array}$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Kellar, D. C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Keelly, M. Keelly, M. Keelly, M. Keelly, M. Keelly, M. Kenney, K. Kenney, K. Kenney, K. Keeninger, E. A. Keeninger, E. A. Keeninger, E. Khader, W. S.	$\begin{array}{c} 1073 \\$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, T. Keller, R. Kelly, A. Kelly, K. Kelly, M. Ketly, M. Keth, D. Kenney, K. Keshavarzian, A. Ketchum, J. Kezirian, E. Khader, W. S. Khader, W. S. Khan, M.	$\begin{array}{c} 1073 \\$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Kellar, D. C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Ketly, M. Ketly, M. Kethy, M. Kesper, S. Kenney, K. Kent, D. Keshavarzian, A. Ketchum, J. Kezirian, E. Khader, W. S. Khan, M. Khan, S.	$\begin{array}{c} 1073 \\$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Ketly, M. Ketly, M. Kespke, N. Kenney, K. Kenney, K. Kensinger, E. A. Ketchum, J. Kezirian, E. Khader, W. S. Khan, M. Khan, M.	$\begin{array}{c} 1073 \\0054, 0054 \\0237, 0424, 0562, 1057 \\0874, 0054, 0054, 0569, 0613, 0691, 1184 \\0874, 0887 \\0874, 0887 \\0223, 0541, 0542 \\0244, 0255 \\0596 \\0896 \\0225, 0384, 0398, 1063 \\0581 \\0131, 0467, 0475, 0537 \\0160 \\0529 \\0583, 0583 \\0583$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, T. Keller, R. Kelly, A. Kelly, A. Kelly, M. Kenney, G. Kensinger, E. A. Kent, D. Keshavarzian, A. Ketchum, J. Keator, W. S. Khader, W. S. Khan, M.	$\begin{array}{c} 1073 \\$
Keele, L Keenan, B. Keenan, B. T. .0053 Keens, T. G. Kellar, D. C. Kellar, D. C. Keller, T. Keller, K. Kelly, A. Kelly, M. Kemner, G. Kemner, G. Kenney, K. Kensinger, E. Kent, D. Keshavarzian, A. Kezirian, E. Khader, W. S. Khan, M. Khan, W. <td< td=""><td>$\begin{array}{c} 1073 \\0054, 0054 \\0237, 0424, 0562, 1057 \\0874, 0054, 0054, 0569, 0613, 0691, 1184 \\0874, 0887 \\0874, 0887 \\0523, 0541, 0542 \\0244, 0255 \\0596 \\0896 \\0225, 0384, 0398, 1063 \\0581 \\0131, 0467, 0475, 0537 \\0160 \\0529 \\0583, 0583 \\0046, 0046, 0416, 1155, 1155 \\ .0109, 0109, 0111, 0111, 0311, 0311 \\0681 \\0780 \\0232 \\0243, 1096 \\0420 \\0702, 0703 \\0702, 0703 \\0702, 0703 \\0702, 0703 \\0702, 0703 \\0702, 0703 \\0702, 0703 \\0105, 0105, 0157 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\ .$</td></td<>	$\begin{array}{c} 1073 \\0054, 0054 \\0237, 0424, 0562, 1057 \\0874, 0054, 0054, 0569, 0613, 0691, 1184 \\0874, 0887 \\0874, 0887 \\0523, 0541, 0542 \\0244, 0255 \\0596 \\0896 \\0225, 0384, 0398, 1063 \\0581 \\0131, 0467, 0475, 0537 \\0160 \\0529 \\0583, 0583 \\0046, 0046, 0416, 1155, 1155 \\ .0109, 0109, 0111, 0111, 0311, 0311 \\0681 \\0780 \\0232 \\0243, 1096 \\0420 \\0702, 0703 \\0702, 0703 \\0702, 0703 \\0702, 0703 \\0702, 0703 \\0702, 0703 \\0702, 0703 \\0105, 0105, 0157 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\0105, 0105, 0105 \\ .$
Keele, L Keenan, B. Keenan, B. T. Keens, T. G. Kellar, D. C. Keller, C. Keller, T. Kelly, A. Kelly, K. Kelly, M. Kemner, G. Kemner, G. Kenney, K. Kenney, K. Kensinger, E. A. Kent, D. Ketchum, J. Ketchum, J. Khader, W. S. Khader, W. S.	$\begin{array}{c} 1073 \\0054, 0054 \\0237, 0424, 0562, 1057 \\0874, 0054, 0054, 0569, 0613, 0691, 1184 \\0874, 0887 \\0874, 0887 \\0874, 0887 \\0223, 0541, 0542 \\0244, 0255 \\0596 \\0896 \\0225, 0384, 0398, 1063 \\0581 \\0131, 0467, 0475, 0537 \\0160 \\0529 \\0583, 0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0583 \\0646, 0046, 0416, 1155, 1155 \\0109, 0109, 0111, 0111, 0311 \\0681 \\0780 \\0606, 1136, 1136, 1177, 1177 \\0637 \\0243 \\0243 \\0420 \\0702 \\0702 \\0702 \\0420 \\0420 \\0420 \\0420 \\0420 \\0420 \\0420 \\0420 \\0420 \\0420 \\0420 \\0420 \\0420 \\0420 \\$

Khosrof, A	
Kiang, J. B	0887
Kidwell, S	0907
Kieley, J	1031
Kille, T.	
Killgore, W. D 0013, 0014, 0038, 0070, 0079, 0080,	0081, 0082,
0140, 0226, 0227, 0232, 0235, 0236, 0240, 0241, 0243,	
0305, 0306, 0307, 0314, 0315, 0316, 0372, 0374, 0375,	
0430, 0544, 0553, 0865, 1053, 1077, 1093, 1095, 1108,	
0100, 0011, 0000, 0000, 1000, 1011, 1000, 1000, 1000,	1160
Killgore, W.	
Kim, B.	
Kim, D	
Kim, D	
Kim, D. S.	
Kim, D	
KIM, G	
Kim, H	
Kim, H	
Kim, HS	
Kim, J	0394
Kim, J	0409
Kim, J	0971
Kim, K. N	1092
Kim, K	0791
Kim, L	
Kim, M	
Kim, N	
Kim, S. Y	
Kim, S	
Kim, S	
KIM, S.	
Kin, S	
Kim, S.	
Kim, S	
Kimura, H.	
Kindel, B. C.	
King, J. A.	
King, R	
Kinnunen, H.	
Kinoshita, L	
Kirkpatrick, J. N	
Kirwan, J. P	0862
Kishi, A	0746
Kishma, E. E	0851
Kishman, E. E.	.0127, 0401
Kitajima, T	
Kite, K	
Kleckner, I	
Kleim, B.	
Klerman, E. B	
Kleva, C.	
Kline, C. E	
Klingaman, E. A.	· · · · · · · · · · · · · · · · · · ·
Klingman, K	
Klingman, K. J.	
Kloss, J.	
KLS Working Group,	
Kluding, P.	
Knappenberger, T.	
Kneeland-Szanto, E	
Knopman, D. S.	
Knowland, V. C	0086

Knutson, K. L	
Ko, NY	/
Ko, WC	/
Koba, S)
Kobayashi, U	
Kodali, L	/
Koh, K	
Kohli, N)
Kohyama, J	/
Koike, T	/
Koirala, A	ŀ
Kolla, B	3
Kollins, S. H	
Kolotovska, V	ŀ
Komatsuzaki, K	;
Kominsky, A	
Kondapalli, K	2
Kondo, H	
Konerman, M	
Konkoly, K	
Konkoly, K. R	
Konno, Y	
Koo, B	
Koo, D	
Koopman-Verhoeff, M	
Koren, O	
Koritala, B	
Korotun, M	
Korthas, H. T	
Kortykowski, M	
Koshorek, G	7
Koskimaki, H	
Kotagal, S	
Kotecha, B	
Kothare, S	
KOUTENTAKI, E	
Krachman, S	
Krahn, L	
Krahn, L. E	
Kram Mendelsohn, A	
Kratz, A. L	
Kräuchi, K	
Kreibig, S	
Kreitinger, K	
Kremerskothen, K	
Kretzmer, T	
Krieger, A	
Krieger, A. C	
	·
KTIEISCH K UZ09)
Krietsch, K	
Krietsch, K. N	
Krietsch, K. N	7
Krietsch, K. N	7)
Krietsch, K. N. .0271 Krishnamurthy, V. B. .0458, 0585, 1107 Krishnan, J. .1209 Kriska, A. M. .0176	7
Krietsch, K. N. .0271 Krishnamurthy, V. B. .0458, 0585, 1107 Krishnan, J. .1209 Kriska, A. M. .0176 Krizan, Z. .0189, 0189, 0230, 0276	7
Krietsch, K. N. .0271 Krishnamurthy, V. B. .0458, 0585, 1107 Krishnan, J. .1209 Kriska, A. M. .0176 Krizan, Z. .0189, 0189, 0230, 0276 Kroeger, D. .0005, 0156, 0156	7
Krietsch, K. N. .0271 Krishnamurthy, V. B. .0458, 0585, 1107 Krishnan, J. .1209 Kriska, A. M. .0176 Krizan, Z. .0189, 0189, 0230, 0276 Kroeger, D. .0005, 0156, 0156 Kroeker, K. A. .0231	7
Krietsch, K. N. .0271 Krishnamurthy, V. B. .0458, 0585, 1107 Krishnan, J. .1209 Kriska, A. M. .0176 Krizan, Z. .0189, 0189, 0230, 0276 Kroeger, D. .0005, 0156, 0156 Kroeker, K. A. .0231 Kronish, I. M. .1154	555
Krietsch, K. N. .0271 Krishnamurthy, V. B. .0458, 0585, 1107 Krishnan, J. .1209 Kriska, A. M. .0176 Krizan, Z. .0189, 0189, 0230, 0276 Kroeger, D. .0005, 0156, 0156 Kroeker, K. A. .0231 Kronish, I. M. .1154 Krueger, J. M. .0017	
Krietsch, K. N. .0271 Krishnamurthy, V. B. .0458, 0585, 1107 Krishnan, J. .1209 Kriska, A. M. .0176 Krizan, Z. .0189, 0189, 0230, 0276 Kroeger, D. .0005, 0156, 0156 Kroeker, K. A. .0231 Kronish, I. M. .1154 Krueger, J. M. .0017 Krull, K. R. .1015	
Krietsch, K. N. .0271 Krishnamurthy, V. B. .0458, 0585, 1107 Krishnan, J. .1209 Kriska, A. M. .0176 Krizan, Z. .0189, 0189, 0230, 0276 Kroeger, D. .0005, 0156, 0156 Kroeker, K. A. .0231 Kronish, I. M. .1154 Krueger, J. M. .0017 Krull, K. R. .1015 Kryger, M. .0707	7) 5 5 5 1 7 5 7
Krietsch, K. N. .0271 Krishnamurthy, V. B. .0458, 0585, 1107 Krishnan, J. .1209 Kriska, A. M. .0176 Krizan, Z. .0189, 0189, 0230, 0276 Kroeger, D. .0005, 0156, 0156 Kroeker, K. A. .0231 Kronish, I. M. .1154 Krueger, J. M. .0017 Krull, K. R. .0017 Kryger, M. .0707 Krystal, A. .0533, 0533, 0540	
Krietsch, K. N. .0271 Krishnamurthy, V. B. .0458, 0585, 1107 Krishnan, J. .1209 Kriska, A. M. .0176 Krizan, Z. .0189, 0189, 0230, 0276 Kroeger, D. .0005, 0156, 0156 Kroeker, K. A. .0231 Kronish, I. M. .1154 Krueger, J. M. .0017 Krull, K. R. .1015 Kryger, M. .0707	

Ku, H
Kubin, L
Kubo, M
Kubota, N
Kuhlman, K. R
Kukafka, D
Kulkas, A
Kumar, D0477, 0478, 0481, 0484
Kumar, R
Kummerow, E
Kun, S
Kun, S. S
Kuna, S. T
Kuna, S. T
Kundel, V
Kunik, M. E1156, 1157
Kuo, CE
Kuo, PY
Kuo, T
Kurdziel, L. B
Kurth, S
Kuschner, W
Kushida, C
Kushida, C. A
Kwak, I
Kwok, K
Kwon, D
Kwon, M
Kwon, S
Kwon, Y
Kyle, D. J
Kyle, S. D

L

Laberge, L
LaFleur, B
LaFollette, K
Laganière, C
Lage, C
Lageman, S. K
Laghi, F
LaGoy, A. D
Lai, C
Lai, Y. J
Laing, K
LaJambe, C. M
Lambiase, M. J
Lambrasko, L. K
Lammers, G
Lammers, G. J
Lammers, G
Lammers-van der Holst, H. M
Lamp, A
Lance, C
Landsness, E. C
Lane, J
Lang, R
Langsetmo, L

	.0030
LaRosa, K. N.	.0994
Larson, O. R.	.0304
Lasko, N. B	
Lasser, R	
Lassonde, J. M.	
Lau, T	
Laughton, A	
Laughon, A.	
Lauren, S.	
Lauteslager, T	
Laureshager, I	
Lavinder, W. G	
Lavigne, A. A.	
Lavigne, A. A.	
Lawrence-Sidebottom, D	
Lawrence-Sidebottolii, D	
Laxinarayan, S	
Layton, M. E	
Lazır, J. M	
Lazar, J. M	
Lazzeroni, L	
Le, B	
Le, T	
Le, W	
Leary, E	
Leary, E. B	
LeBourgeois, M. K	
LEBRUN, MP.	
Lecendreux, M.	
Le-Dong, NN.	
Lee, A	.0966
Lee, A	.0675
Lee, B	.0675 .0789
Lee, B	.0675 .0789 .1013
Lee, B. Lee, CH. Lee, CN.	.0675 .0789 .1013 .0819
Lee, B	.0675 .0789 .1013 .0819 .0321
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0285 .0598
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0285 .0598 .0455
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, B. Lee, B. Lee, B. Lee, H. Lee, H. Lee, H. Lee, J. Lee, J. Lee, J. Lee, J. Lee, K. Lee, N. Lee, N. Lee, M. Lee, M. Lee, M. Lee, M. Lee, M. Lee, N. Lee, M. Lee, M. Lee, M. Lee, N. Lee, N. Lee, N. Lee, N. Lee, M. Lee, N. Lee, N.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0598 .0455 .0285 .0598
Lee, B	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0285 .0598 .0455 .1206 .0916
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, B. Lee, B. Lee, H. Lee, H. Lee, H. Lee, JM. Lee, J. Lee, K. Lee, S. .0064 Lee, M. .0064 Lee, M. .0064 Lee, P-L. .0064 Lee, P-L. .0064 Lee, S. .00647	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0285 .0285 .0285 .1206 .0916 .0835
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, B. Lee, B. Lee, H. Lee, H. Lee, H. Lee, J. Lee, J. Lee, K. Lee, S. Lee, M. Lee, S. Lee, M. Lee, M. Lee, M. Lee, S. Lee, S. Lee, S.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0285 .0285 .0285 .1206 .0835 .0192
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, B. Lee, B. Lee, H. Lee, H. Lee, H. Lee, J. Lee, J. Lee, J. Lee, K. Lee, S. Lee, M. Lee, S. Lee, M. Lee, S. Lee, M. Lee, M. Lee, M. Lee, S. Lee, S.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0289 .0287 .0280 .0271 .0280 .0271 .0280 .0271 .0280 .0271 .0280 .0271 .0280 .0271 .0285 .0271 .0285 .0271 .0285 .0271 .0285 .0271 .0285 .0271 .0285 .0275 .0285 .0389
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, D. Lee, H. Lee, H. Lee, H. Lee, H. Lee, J. Lee, J. Lee, J. Lee, J. Lee, K. Lee, K. Lee, K. Lee, K. Lee, K. Lee, R. Lee, S. Lee, M. Lee, M. Lee, M. Lee, M. Lee, S. Lee, S.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0288 .02888 .02888 .02888 .02888 .02888 .02888 .02888 .02888 .02888 .02888 .02888 .02888 .02888 .02888 .02888 .028888 .02888 .02888 .02888 .02888 .028888 .02888 .02888 .02888 .02888 .028888 .02888 .02888 .02888 .02888 .028888 .02888 .02888 .02888 .028888 .028888 .02888 .028888 .0288888 .028888 .028888888 .028888888888
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, D. Lee, E. Lee, H. Lee, H. Lee, H. Lee, J. Lee, K. Lee, K. Lee, K. Lee, K. Lee, K. Lee, S. Lee, S. Lee, M. Lee, M. Lee, M. Lee, M. Lee, S.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0265 .02855 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, D. Lee, H. Lee, H. Lee, H. Lee, H. Lee, J. Lee, K. Lee, K. Lee, K. Lee, K. Lee, R. Lee, R. Lee, M. Lee, M. Lee, PL. Lee, S. Lee, S.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0092
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, B. Lee, H. Lee, H. Lee, H. Lee, H. Lee, J. Lee, K. Lee, K. Lee, K. Lee, K. Lee, R. Lee, R. Lee, M. Lee, M. Lee, R. Lee, R. Lee, S. Lee, Y.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0097
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, D. Lee, B. Lee, H. Lee, H. Lee, H. Lee, H. Lee, J. Lee, K. Lee, K. Lee, K. Lee, K. Lee, K. Lee, K. Lee, S. Lee, M. Lee, M. Lee, M. Lee, M. Lee, S. Lee, Y. Lee, Y. LEE, Y.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0097 .02766 .0276 .0276 .0276 .0276 .0276 .0276 .0276 .0276 .0276 .0276
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, B. Lee, H. Lee, H. Lee, H. Lee, H. Lee, J. Lee, K. Lee, S. Lee, M. Lee, M. Lee, M. Lee, S. Lee, X. Lee, Y. Lee, Y. Lee, Y. Lee, Y. Lee, Y.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0271 .0285 .0271 .0285 .0092 .0276 .0277 .0276 .0277 .0277 .0092 .0771 .0789 .0455
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, B. Lee, H. Lee, H. Lee, H. Lee, H. Lee, J. Lee, K. Lee, M. Lee, M. Lee, M. Lee, M. Lee, Q. Lee, Q. Lee, S. Lee, Y. Lee, Y. Lee, Y. Lee, Y. Lee, Y. Lee, Y. Lee, Y.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0126 .0389 .1176 .0047 .0092 .0791 .0789 .0455 .0284
Lee, B. Lee, CH. Lee, CN. Lee, D. Lee, B. Lee, H. Lee, H. Lee, H. Lee, H. Lee, J. Lee, K. Lee, S. Lee, M. Lee, M. Lee, M. Lee, S. Lee, X. Lee, Y. Lee, Y. Lee, Y. Lee, Y. Lee, Y.	.0675 .0789 .1013 .0819 .0321 .0800 .0455 .0971 .0845 .0126 .0459 .0047 .0868 .0455 .02855 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285 .0285

Leete, J. J
LEGER, D
Léger, D
LEGER, D
Lehoux, S. D
Lehrer, H. M
Lehrer, M
LEI, H
Leichman, E. S
Leitner, C
Lemieux, R
Lemon, S
Leng, Y
Lengenfelder, J
Leone, M. J
Leow, Z
Lepage, C
Lepäge, C
11
Lequerica, A
Leroux, A
Lester, R
Lett, J
LEUNG, L
Leu-Semenescu, S
Levenson, J. C
Leverenz, J
Levin, B. E
Levine, R. S
Levri, J
Levri, J. M
Lewis, R
Lewis, S. J
Lewis, T. T
Li, A. M
Li, CH
Li, C
Li, C
Li, CS
Li, C
Li, C. I
Li, CY
Li, F
Li, J
Li, J
Li, L
Li, P
Li, Q
Li, S
Li, W
Li, WY
Li, W
Li, X
Li, X
Li, Y
Li, Y
Li, Z
Liang, J
Liang, J
Liang, SF
Liang, Z
Liao, D
Liao, J
Liao, WC

Lim, D. C	
	.0143, 0145, 0287, 0519, 0519, 0824, 0824
Lim, L. M	
Limbekar, N.	
Limone, N	
Lin, C	
Lin, L	
Link, D. G	
Liu, MH	
Liu, M	
-	
-	
-	
Loputo, 12	

Lopera, F. .0421 Lopes, MC. .0937 Lopez, R. .0750 Lopez, R. .1166, 1168 Lopez, S. .0370 Lopez-Jimenez, F. .0719, 0719 Lorenz, R. .0229 Lorenz, R. A. .0854, 0857 Lotierzo, M. .0750 Lott, I. T. .0425 Lough, L. .1046, 1046, 1058, 1058 Lovato, N. .1192, 1199 Low, D. .0787 Low, V. J. .0355 Lozano, A. .0861 Lu, M. .0721, 0721 Lubas, M. M. .1015 Lucchesi, L. M. .1126 Lucena, L. R. .0498 Lui, K. K. .0325, 0335, 0422
Lui, M. M
Lunsford-Avery, J. R
Lunyera, J
Luo, Y
Luo*, G
Lupini, F
Luster, 1
Luyster, F
Luyster, F. S
Luz, G. P
Lv, C
Lv, Q
Lyamin, O
Lyng, P. J

Μ

Ma, C
Ma, C
Ma, H
Ma, N
Ma, X
Ma, Y
Macauley, S. L
Macey, P. M
MacKay, S. G
MacKintosh, E
MacMullen, L. E
Madden, E
Madden, E
Maddison, K
Maddison, K
Maddison, K
Maddison, K. .0615 Madera, C. .0864 Maeder, T. .0552, 1067 Magalang, U. .0606, 0608, 1177, 1177
Maddison, K. .0615 Madera, C. .0864 Maeder, T. .0552, 1067 Magalang, U. .0606, 0608, 1177, 1177 Maghsoudipour, M. .0690
Maddison, K. .0615 Madera, C. .0864 Maeder, T. .0552, 1067 Magalang, U. .0606, 0608, 1177, 1177 Maghsoudipour, M. .0690 Maguen, S. .0503
Maddison, K. .0615 Madera, C. .0864 Maeder, T. .0552, 1067 Magalang, U. .0606, 0608, 1177, 1177 Maghsoudipour, M. .0690 Maguen, S. .0503 Mahakit, P. .0625
Maddison, K. .0615 Madera, C. .0864 Maeder, T. .0552, 1067 Magalang, U. .0606, 0608, 1177, 1177 Maghsoudipour, M. .0690 Maguen, S. .0503 Mahakit, P. .0625 Maher, B. S. .0988
Maddison, K. .0615 Madera, C. .0864 Maeder, T. .0552, 1067 Magalang, U. .0606, 0608, 1177, 1177 Maghsoudipour, M. .0690 Maguen, S. .0503 Mahakit, P. .0625

Mahoney, M. M	
Maier E	
Maki, P. M	
Malanga, E	
	.0560, 0611, 0641, 0690, 0693, 0772
Mallett, R.	
Malone, S. K.	
Mamikonyan, E	
	,0499,0510,0533,0533,0534,0540
	$\ldots \ldots .0091, 0325, 0335, 0422, 0425$
Manderscheid, R	
Mandrell, B	
Mandrell, B. N.	
Manoach, D	
Manoogian, E. N.	
Mansukhani, M. P	
Mansukhani, M. P	
Mansukhani, M. P	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B	
Mansukhani, M. P	
Mansukhani, M. P	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C Marchetti, D	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C Marchetti, D Marelli, S	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C Marchetti, D Marcletti, S Marcich, Y	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C Marchetti, D Marchetti, S Marcich, Y Marino, V. R	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C Marchetti, D Marchetti, S Marcich, Y Marino, V. R	
Mansukhani, M. P Manthei, M Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C Marchetti, D Marelli, S Maricich, Y Marino, V. R Markland, A	
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetti, D.Marelli, S.Maricich, Y.Marino, V. R.Markland, A.Markun, L. C.	
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetti, D.Marelli, S.Maricich, Y.Marino, V. R.Markland, A.Markun, L. C.Markunas, C. M.	
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetta, D.Marchetti, D.Marelli, S.Maricich, Y.Marino, V. R.Markland, A.Markunas, C. M.Markwald, R. R.	$\begin{array}{c}1029\\0173, 0173, 0719, 0719, 0738\\0330\\0188, 0199, 0219, 1028\\0189, 0189\\0250\\0680\\0579\\0685\\0985\\0551, 0551\\0525, 0728\\0524\\0524\\0524\\0524\\0524\\0524\\0524\\0524\\0524\\0524\\0524\\0524\\0524\\0117\\1147\\0211\\0050, 0097, 0284\\ \end{array}$
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetta, C.Marchetti, D.Marelli, S.Maricich, Y.Markland, A.Markunas, C. M.Markwald, R. R.Marshall, N. S.	$\begin{array}{c}1029\\0173, 0173, 0719, 0719, 0738\\0330\\0188, 0199, 0219, 1028\\0189, 0189\\0250\\0680\\0579\\0685\\0985\\0551, 0551\\0525, 0728\\0524\\0524\\0524\\0345\\1017\\1147\\0211\\0050, 0097, 0284\\0672\end{array}$
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetta, C.Marchetti, D.Maricich, Y.Marino, V. R.Markland, A.Markunas, C. M.Markwald, R. R.Marsland, A. L.	
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetta, C.Marchetti, D.Maricich, Y.Marino, V. R.Markland, A.Markunas, C. M.Markwald, R. R.Marsland, A. L.Marsland, A. L.Martin, A.	
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetta, C.Marchetti, D.Marcich, Y.Marino, V. R.Markund, A.Markund, A.Markund, A.Markulad, R. R.Marshall, N. S.Marsland, A.Martin, A.Martin, B. J.	
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetta, C.Marchetti, D.Marcich, Y.Marino, V. R.Markund, A.Markund, A.Markund, A.Markulad, R. R.Marshall, N. S.Marsland, A.Martin, A.Martin, B. J.	
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetta, C.Marchetti, D.Maricich, Y.Marino, V. R.Markland, A.Markunas, C. M.Markunas, C. M.Marshall, N. S.Marsland, A. L.Martin, A.Martin, B. J.Martin, J. L.0131, 0467	
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetta, C.Marchetti, D.Marcich, Y.Marino, V. R.Markland, A.Markunas, C. M.Marshall, N. S.Marsland, A. L.Martin, B. J.Martin, J. LMartinez, D.	
Mansukhani, M. P.Manthei, M.Mantua, J.Mantua, J. R.Manuck, S. B.Manzar, M.Marais, L.Marceau, S.Marchetta, C.Marchetta, C.Marchetti, D.Marcich, Y.Marino, V. R.Markland, A.Markunas, C. M.Marshall, N. S.Marsland, A. L.Martin, B. J.Martin, J. L.Ol 31, 0467Martinez, F.	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C Marchetta, C Marchetti, D Marchetti, D Marchetti, S Marchetti, S Marchetti, S Marchetti, C Marchetti, C Marchetta, C Marchetta, C Marchetta, C Marchetta, C Marchetta, C Marchetta, C Marchetta, C Marchetta, C Marchetta, C Markland, A Markland, A Markunas, C. M Markwald, R. R Markwald, R. R Marshall, N. S Marshall, N. S Martin, A Martin, B. J Martin, J. L	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C Marchetta, C Marchetti, D Marchetti, D Marchetti, S Marchetti, S Marchetti, S Marchetti, C Marchetta, C Markland, A Markland, A Markunas, C. M Markwald, R. R Markwald, R. R Marthand, A. L Martin, A Martin, B. J Martin, J. L	
Mansukhani, M. P Manthei, M Mantua, J Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C Marchetti, D Marchetti, D Marchetti, S Marchetti, S Marchetti, S Marchetti, C Marchetti, C Marchetti, C Marchetti, C Marchetti, C Marchetti, C Markland, A Markland, A Markunas, C. M Markwald, R. R Marshall, N. S Marshall, N. S Martin, A Martin, B. J Martin, J. L	
Mansukhani, M. P Manthei, M Mantua, J. R Mantua, J. R Manuck, S. B Manzar, M Marais, L Marceau, S Marchetta, C Marchetta, C Marchetti, D Marchetti, D Marchetti, D Marchetti, C Marchetti, C Marchetti, C Marchetti, C Marchetti, C Markland, A Markland, A Markunas, C. M Markwald, R. R Markwald, R. R Marshall, N. S Martin, A Martin, A Martin, B. J Martinez, D Martinez, S Martinez, S Martinez, S Martinez, Garcia, AM	$\begin{array}{c} 1029\\0173, 0173, 0719, 0719, 0738\\0330\\0188, 0199, 0219, 1028\\0189, 0189\\0250\\0680\\0579\\0685\\0985\\0551, 0551\\0525, 0728\\0524\\0524\\0524\\0345\\0117\\1147\\0211\\0050, 0097, 0284\\0672\\0820\\0445\\0242\\0445\\0242\\0445\\0242\\0445\\0242\\0445\\0242\\0445\\0242\\0556\\0421\\0556\\0421\\0556\\0421\\0683, 0696\\0238, 0995\\0551, 0551\\$
Mansukhani, M. P Manthei, M Mantua, J. R Mantua, J. R Manuck, S. B Manzar, M Marcais, L Marceau, S Marchetta, C Marchetti, D Marchetti, D Marchetti, D Marchetti, C Marchetti, C Marchetti, C Marchetti, C Marchetti, C Markland, A Markland, A Markunas, C. M Markwald, R. R Markwald, R. R Marshall, N. S Marshall, N. S Martin, A Martin, B. J Martin, J. L0131, 0467 Martinez, F Martinez, S Martinez, S Martinez, S Martinez, Miller, E. E	

Mashaqi, S	
Mashek, D. G	
Maski, K	
Maski, K. P	
Maski*, K. P	
Maslik, M.	
Mason, B.	
*	
Mason, G	
Mason, G. M	
Mason, I. C	
Massar, S. A.	
Masse, JF.	
Master, L	
Mastin, D	
Mastin, D. F.	
Matadiaby, F.	
Matar, E	
Mathew, E. V.	
Mathew, G. M.	
Mathur, S. K.	
Matsangas, P.	
Matsuda, A.	
Matsuo, M	
Matsuo, Y	
Matthews, C.	
Matthews, E.	
Matthews, K. A.	
Maurer, J	
May, A	
May, A. M.	
Mayer, C. M.	
-	
Mayne, S	
Mayne, S. L.	
Mayo, P	
Mazimba, S	
Mazurek, M	
Mazzotti, D. R	
Mbah, A. K	
McArdle, N	
McArthur, B.	
McCall, W. V.	
McCann, D	
McCarter, S.	
McCarter, S. J.	
McCarthy, J.	
McCarty, D. E.	
McCauley, E.	
McCauley, M. E.	
McClintock, H. F.	
McComb, G. J	
McCrae, C	
McCrae, C. S	
McCurry, S.	
McCurry, S. M.	
McDermott, J. E.	
McFarlane, S.	
McGhee, V.	
McGlashan, E. M.	
	· · · · · · · · · · · · · · · · · · ·
McGlinchev F	1170 1101
McGlinchey, E	
McGovney, K.	
McGovney, K	
McGovney, K	
McGovney, K	

McGrath, J
McGuire, K
McHill, A. W
McIntyre, E
McIntyre, E. A
McKane, S
McKenzie-Hartmann, T
McKeon, A
McKeon, A. B
McLaughlin, L. E
McMillan, L
McNally, J. M
McNally, J
McQuillen, A
McRae-Clark, A. L
Md Hossain, M
Mead, M. P
Meaklim, H
Mebust, K. A
Mechal, R
Medina-Inojosa, J
Mednick, S
Mednick, S. C
Meers, J. M
Mehra, R 0057, 0057, 0463, 0463, 0543, 0558, 0583, 0583, 0614,
0650, 0681, 0687, 0906, 1185
0650, 0681, 0687, 0906, 1185 Mehra, R
Mehta, B
Mehta, I
Mehta, R
Mehta, T
Mehta, T. R
Meinhausen, C. E
Meinhausen, C. E
Meinhausen, C. E
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079 Mello, M. F. .1079
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079 Mello, W. F. .0272 Mello, V. .0272
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079 Mello, W. F. .0272 Mello, V. .0272 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0072 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079 Mello, W. F. .0272 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981 Meltzer, L. J. .0924, 0993, 1171
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0072, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079 Mello, M. F. .0072 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981 Meltzer, L. J. .0924, 0993, 1171 Memarian, N. .0669
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0072, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079 Mello, M. F. .1079 Mello, V. .0272 Meltzer, C. .0769 Meltzer, L. J. .0921, 0921, 0981 Meltzer, L. J. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0072, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079 Mello, M. F. .1079 Mello, V. .0272 Meltzer, C. .0769 Meltzer, L. J. .0921, 0921, 0981 Meltzer, L. J. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Mendelevich, E. .0207
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0072, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .0079 Mello, M. F. .0079 Mello, V. .0272 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981 Meltzer, L. .0272 Meltzer, L. .0201 Meltzer, L. .02021, 0921, 0931, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Mendez, M. .0605 Meng, A. .0837
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079 Mello, M. F. .0272 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981 Meltzer, L. J. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Mendelevich, E. .0207 Mendel, M .0663 Meng, A. L. .0723
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .0079 Mello, M. F. .0079 Mello, V. .0272 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981 Meltzer, L. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Mendalevich, E. .0207 Mendel, M. .0605 Meng, A. L. .0723 Meng, H. .0341
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .0079 Mello, M. F. .0079 Mello, V. .0272 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981 Meltzer, L. J. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Mendelevich, E. .0207 Mendel, M. .0663 Meng, A. L. .0723 Meng, H. .0341
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .0079 Mello, M. F. .0079 Mello, V. .0272 Melzer, C. .0769 Melzer, L. .0921, 0921, 0981 Meltzer, L. .0924, 0993, 1171 Memarian, N. .06605 Meng, A. L. .0723 Meng, H. .0341 Meng, L. .0713 Meng, NH. .1134
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .0079 Mello, M. F. .0079 Mello, V. .0272 Melzer, C. .0769 Melzer, L. .0921, 0921, 0981 Meltzer, L. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Meng, A. L. .0723 Meng, H. .0341 Meng, L. .0713 Menghini, L. .1134
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .0079 Mello, M. F. .0079 Mello, V. .0272 Melzer, C. .0769 Melzer, L. .0921, 0921, 0981 Meltzer, L. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Meng, A. L. .0723 Meng, H. .0341 Meng, L. .0341 Meng, NH. .1134 Menghini, L. .1193 Menno, D. .0641, 0693, 0751, 0772, 0950
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .0079 Mello, M. F. .0079 Mello, V. .0272 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981 Meltzer, L. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Meng, A. L. .0723 Meng, A. L. .0723 Meng, NH. .0341 Meng, NH. .1134 Menghini, L. .1134 Menno, D. .0641, 0693, 0751, 0772, 0950 Menon, P. .1213
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .0079 Mello, M. F. .0079 Mello, V. .0272 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981 Meltzer, L. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Mendel, A. L. .0723 Meng, A. L. .0723 Meng, H. .0341 Meng, L. .0713 Meng, NH. .1134 Menghini, L. .1193 Menno, D. .0641, 0693, 0751, 0772, 0950 Menon, P. .1213 Mentch, L. .0850
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079 Mello, M. F. .0272 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981 Meltzer, L. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Mende, M. .0605 Meng, A. L. .0723 Meng, A. L. .0723 Meng, NH. .0341 Meng, NH. .0341 Meng, NH. .0341 Menghini, L. .0713 Menon, D. .0641, 0693, 0751, 0772, 0950 Menon, P. .1213 Mentch, L. .0850
Meinhausen, C. E. 0307 Meira e Cruz, M. 0657, 0804 Meissner, C. 0230 Meissner, S. 0587 Mekala, V. 0072 Melaku, Y. A. 0722, 0722 Melanson, E. L. 0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. 1079 Mello, M. F. .0072 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0921 Meltzer, L. .0921, 0921, 0931 Meltzer, L. J. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Mendelevich, E. .0207 Mende, M. .0669 Mendelevich, E. .0207 Mende, M. .0605 Meng, A. L. .0723 Meng, H. .0341 Meng, NH. .1134 Menghini, L. .1134 Mendpinini, L. .1133 Menno, D. .0641, 0693, 0751, 0772, 0950 Menon, P. .1213 Mentch, L. .08850 Merchant, G.
Meinhausen, C. E. .0307 Meira e Cruz, M. .0657, 0804 Meissner, C. .0230 Meissner, S. .0587 Mekala, V. .0072 Melaku, Y. A. .0722, 0722 Melanson, E. L. .0050, 0132 Mellman, T. .0454, 0454, 0490 Mello, A. F. .1079 Mello, M. F. .0272 Meltzer, C. .0769 Meltzer, L. .0921, 0921, 0981 Meltzer, L. .0924, 0993, 1171 Memarian, N. .0669 Mendelevich, E. .0207 Mende, M. .0605 Meng, A. L. .0723 Meng, H. .0341 Meng, NH. .1134 Menghini, L. .1134 Menghini, L. .1133 Menno, D. .0641, 0693, 0751, 0772, 0950 Menon, P. .1213 Mentch, L. .0850

Mesa, J	
Wiesa, J	5
Meskill, G. J	5
Meskill, S. D	
Meslier, N	
Messman, B	
Messman, B. A	
Messnick, R)
Metlaine, A	2
Meyer, T. E	
Meysing, A	
Michelle, S	
Michelson, D0487, 0488	
Michelson, K. P	2
Mickelson, C)
Mickelson, C. A	
Mielke, M. M	
Mignot, E	
Milanak, M	
Miles, J)
Miles, S. R	3
Milinovich, A	3
Miller, A. M	
Miller, A	
Miller, E	
Miller, J. E	3
Miller, K	5
Miller, K. E	2
Miller, M	
Miller, M	
Mills, B	
Mindell, J. A	
Miner, B	
Mintz, J	3
Minville, C	5
Miquel, S	3
Miranda, N. W	
Mishima, K	
Mishra, S	,
Mitchell, H. D	
	5
Mitchell, J. A	5
	5
Mitchell, J. A	5
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017	5
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. N. .0475, 0581, 0644	5 2 1 7 4
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. N. .0475, 0581, 0644 Mitchell, M. .0467	5 2 7 1 7
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, S. .0021, 0021, 0024	5 2 7 4 7
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0021, 0024 Mitmesser, S. H. .0545	5 2 7 4 7 7 4
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535	5 2 7 7 4 7 7 4 5 5
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0021, 0024 Mitmesser, S. H. .0545	5 2 7 7 4 7 7 4 5 5
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010	5 2 7 7 4 5 5 5
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575	5 2 7 4 7 7 4 5 5 5 5
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mittahai, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172	5 2 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0467 Mitchell, M. .0467 Mithani, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167	5 2 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0467 Mithani, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Mo, L. .0281	5 2 7 4 7 4 5 5 5 5 2 7
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mittahl, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Moelter, S. .0861	5 2 7 7 7 7 7 7 7 7 1
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0467 Mithani, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Mo, L. .0281	5 2 7 7 7 7 7 7 7 7 1
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mittahl, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Moelter, S. .0861	5 2 1 7 4 7 7 4 5 5 5 2 7 1 1 9
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mithani, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Moelter, S. .0281 Moelter, S. .0861 Mohd, A. .0969	5 2 1 7 7 4 7 7 7 7 7 7
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0467 Mittahl, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Mo, L. .0281 Moelter, S. .0861 Mohd, A. .0965 Mok, Y. .0574, 0627 Moline, M .0574, 0627	5 2 1 7 7 4 7 7 1 5 5 5 2 7 1 1 9 7 7
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0467 Mitchell, M. .0467 Mitchell, M. .0467 Mithani, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Moelter, S. .0861 Mohd, A. .0969 Mok, Y. .0574, 0627 Moline, M .0473, 0474, 0477, 0478, 0479, 0480, 0481, 0484, 0485, 0486	
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mittahai, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Mo, L. .0281 Moelter, S. .0861 Mohd, A. .0969 Mok, Y. .0574, 0627 Moline, M .0574, 0627 Moline, M .0167 0473, 0474, 0477, 0478, 0479, 0480, 0481, 0484, 0485, 0486 Mollicone, D. J. .0196, 0261	5 2 1 7 7 4 7 7 4 5 5 5 2 7 1 1 9 7 5
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0467 Mitchell, M. .0467 Mithani, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Mo, L. .0281 Moelter, S. .0861 Mohd, A. .0969 Mok, Y. .0574, 0627 Moline, M .00574, 0627 Moline, M .0196, 0261 Monbelli, S. .0196, 0261 Mombelli, S. .0728	5 2 1 7 4 7 4 7 4 7 7 1 1 9 7 7 1 1 9 7 7 1 1 9 7 7 1 1 9 7 7 1 1 9 7 7 1 9 7 7 7 7
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mithani, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Mo, L. .0281 Moelter, S. .0861 Mohd, A. .0969 Mok, Y. .0574, 0627 Moline, M	5 2 1 7 7 4 5 5 2 7 1 1 7 7 5 2 7 1 1 9 7 5 2 7 1 1 7 7 4 5 5 9 7 1 7 7 4 5 5 9 7 7 4 7 7 4 5 5 9 7 7 4 7 7 7 4 5 5 9 7 7 4 5 5 9 7 7 4 5 5 9 7 7 4 5 5 9 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0467 Mitchell, M. .0467 Mitchell, M. .0467 Mithani, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Mo, L. .0281 Moelter, S. .0861 Mohd, A. .0969 Mok, Y. .0574, 0627 Moline, M .0167 Moline, M .01066 Mollicone, D. J. .0196, 0261 Mombelli, S. .0728 Monaghan, T. F. .0822, 0823, 1012 Monden, K. .0606	5 2 1 7 4 5 5 0 5 2 7 1 1 0 7 5 1 8 2 5
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mithani, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Mo, L. .0281 Moelter, S. .0861 Mohd, A. .0969 Mok, Y. .0574, 0627 Moline, M	521774755) 527177455) 5271197513259
Mitchell, J. A. .0390, 0397, 0952 Mitchell, J. .0038, 0081 Mitchell, M. .1017 Mitchell, M. .1017 Mitchell, M. .0475, 0581, 0644 Mitchell, M. .0467 Mitchell, M. .0467 Mitchell, M. .0467 Mitchell, M. .0467 Mithani, S. .0021, 0021, 0024 Mitmesser, S. H. .0545 Mittal, A. .0535 Mitterling, T. .0010 Miyata, S. .0575 Mizhquiri Barbecho, J. S. .0172 Mizrahci, N. .0167 Mo, L. .0281 Moelter, S. .0861 Mohd, A. .0969 Mok, Y. .0574, 0627 Moline, M .0167 Moline, M .01066 Mollicone, D. J. .0196, 0261 Mombelli, S. .0728 Monaghan, T. F. .0822, 0823, 1012 Monden, K. .0606	521774755) 527177455) 5271197513259

	0743, 0745
5.	
Moore, J. M	
Morgenthaler, T. I.	0173, 0173, 0668, 0695, 0698
Morimoto, M	
Morin, C	
Morin, C. M	
-	
Moskowitz, A.	
Moss, K	
Mossavar-Rahmani, Y	
· · · · · · · · · · · · · · · · · · ·	
Mott, C. G	
-	
·	
wuudana, N	

Mudrakola, H. V	5
Muench, A. L	
Mukerji, S	
Mullen, L	
Muller, D	
Mullington, J	
Mullington, J. M	
Mullins, A0090, 0144, 1150	
Mullins, A. E	
Mullins, K. M	
Mun, J. G	
Munafo, D	
Munafo, D. A	
Münch, M. Y	
Mundt, J. M	
Munoz, M	
Munro, C. L	
Muntner, P	
Murphy, A. S	
Murray, C. F	
Murugan, N	
Musliu, T	
Musso, M	
Mwendwa, D. T	
Myers, A	
Myers, S. A	
Mylonas, D	
Mysliwiec, V	J

Ν

Na, M
Nacif, S
Nadhim, A
Nadhim, A. N
Nadorff, M
Nagaoka, T
Nagy, S
Nahmod, N. G
Naik, A. D
Naik, T
Naime, S
Nair, N
Naismith, S. L
Naji, M
Najimi, N
Nakagawa, E
Nakamura, K. P
Nakase-Richardson, R
0416.0729.1073.1124.1136.1136.1177.1177

0416, 0729, 1073, 1124, 1136, 1136, 1177, 1177
Nakayama, H
Nallu, S1124
NAM, H
Nan, B
Nápoles, A
Naqeeb, B
Narang, I
Nasr, S. Z
Nasser, R
Nathan, M. D0190

Naufel, M. F.	
Navidi, W	
Nawabit, R	
Nayak, M. M.	
Naylor, J. A	
Nazário, L.	
Nazmi, A.	
Neikrug, A. B.	
Neill, S. E.	
Nelson, M. D.	
Nelson, S. M.	
Nene, Y	
Ness, K	
Ness, K. K	
Nettel-Aguirre, A.	
Newton, R. L	
Neylan, T. C	
Ng, L	
Ng, T. H.	
Ngari, W	
Nguyen, A.	
Nguyen, D. D.	
Nguyen, J	
Nguyen-Phan, AH	
Nguyen-Rodriguez, S	
Nguyen-Rodriguez, S. T	
Ni, X	
Ni, Y	
Ni, YL	
Nichols, I	
Nichols, I	
Nichols, I. S	
Nichols, I	0309 0426 0483, 0483, 1073 0157 0088 0403 0403 0403
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. .0008	
Nichols, I	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. .0008	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishimura, Y.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishimura, Y. Nishino, S.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishimura, Y. Nishino, S. Nixon, G. M.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishihinura, Y. Nixon, G. M. Nixoda, A.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishinon, S. Nixon, G. M. Noda, A.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Niels, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishihinura, Y. Nixon, G. M. Noda, A. Nofzinger, E.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishino, S. Nixon, G. M. Noda, A. Nofzinger, E. Nogi, T.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishino, S. Nixon, G. M. Noda, A. Nofzinger, E. Nogi, T. Norman, S.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishino, S. Nixon, G. M. Noda, A. Nofzinger, E. Nogi, T. Norman, S.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nirogi, R. Nirogi, R. Nishikawa, K. Nishino, S. Nixon, G. M. Noda, A. Nofzinger, E. Nogi, T. Norman, S. Nouraie, S. M.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nirogi, R. Nirogi, R. Nishikawa, K. Nishino, S. Nixon, G. M. Noda, A. Nofzinger, E. Nogi, T. Norman, S. Nouraie, S. M. Novack, V.	$0309 \\0426 \\0483, 0483, 1073 \\0157 \\0088 \\0403 \\0403 \\0403 \\0403 \\0403 \\0403 \\0453 \\0518 \\0561 \\0518 \\0518 \\0575 \\0652 \\ .0482, 0514, 0515, 1198 \\0146 \\0582 \\0989 \\0357 \\0891 \\0891 \\0146 \\0582 \\0989 \\0357 \\0899 \\0357 \\0899 \\0899 \\0357 \\0899 \\0890 \\080$
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nirogi, R. Nirogi, R. Nishikawa, K. Nishino, S. Nixon, G. M. Noda, A. Nofzinger, E. Nogi, T. Norman, S. Nouraie, S. M.	$0309 \\0426 \\0483, 0483, 1073 \\0157 \\0088 \\0403 \\0403 \\0403 \\0403 \\0403 \\0403 \\0453 \\0518 \\0561 \\0518 \\0518 \\0575 \\0652 \\ .0482, 0514, 0515, 1198 \\0146 \\0582 \\0989 \\0357 \\0891 \\0891 \\0146 \\0582 \\0989 \\0357 \\0899 \\0357 \\0899 \\0899 \\0357 \\0899 \\0890 \\080$
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nirogi, R. Nirogi, R. Nishikawa, K. Nishino, S. Nixon, G. M. Noda, A. Nofzinger, E. Nogi, T. Norman, S. Nouraie, S. M. Novack, V.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishihikawa, K. Noda, A. Nofzinger, E. Norman, S. Novack, V. Nowakowski, S. Noyes, E. <td></td>	
Nichols, I. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishihikawa, K. Nishihino, S. Nixon, G. M. Noda, A. Noda, A. Nofzinger, E. Norman, S. Novack, V. Nowakowski, S. Nowakowski, S. Nunes, J.	
Nichols, I. Nicholson, K. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishihikawa, K. Nishinura, Y. Nishino, S. Nixon, G. M. Noda, A. Nofzinger, E. Nogi, T. Nouraie, S. M. Novack, V. Nowakowski, S. Nunes, J.	
Nichols, I. Nichols, I. S. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishihura, Y. Nishino, S. Nixon, G. M. Noda, A. Nofzinger, E. Nogi, T. Nouraie, S. M. Novack, V. Nowakowski, S. Nunes, R. Nusbaum, A. T.	
Nichols, I. Nicholson, K. Nicholson, K. Nicoletta, A. Nielsen, T. Nielson, S. Nieto, J. Nindl, B. C. Nirogi, R. Nishikawa, K. Nishihikawa, K. Nishinura, Y. Nishino, S. Nixon, G. M. Noda, A. Nofzinger, E. Nogi, T. Nouraie, S. M. Novack, V. Nowakowski, S. Nunes, J.	

0

O'Brien, L	
O'Brien, L. M	.0221, 0221, 0470, 0470, 0877, 1014
O'Byrne, N	

Ochsner Margolies, S
O'Connor, D
O'Connor, P
O'Connor, S. G
O'Donovan, A
,
Ogna, A
Ogo, H
O'Gorman, C
Oh, A
Oh, S
O'Hara, B
O'Hara, R
Ohayon, M. M
Ohmichi, M
Oji, E
Okoye, S
Okun, M
Ólafsdóttir, G. H
Olatunji, B. O
Oldani, A0522
Oles, S. K
Olesen, A. N
Oliveira, E
Oliveira, G. P
Oliveira, L
Oliveira, L. S
Oliveira, L
Oliveira, M. M
Oliveira, W
Oliver, K. I
Ollila, H
Ollinger, J
Olsen, M
Olsen, S
Olsen, S
Olson, T
Olson, T
Olson, T
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Oppy, K. .0883, 0907
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Oppy, K. .0883, 0907 Orff, H. J. .0866
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Oppy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0560
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Oppy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0560 Osburn, S. .0508, 0756
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Oppy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0560
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Oppy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0560 Osburn, S. .0508, 0756
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .00107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Opp De Beeck, S. .0568, 0568 Oppy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0560 Osburn, S. .0508, 0756 Ose, J. .0044
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .00107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Opry, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0508, 0756 Ose, J. .0044 Osorio, R. S. .1150
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .00107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Sonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Opy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0560 Osburn, S. .0508, 0756 Ose, J. .0044 Osorio, R. S. .0011 Osorio, R. S. .0863, 0864, 1153
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .00107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Sonka, K. .0740, 0753 Ono, T. .0757 Onyconwu, C. .0865 Op De Beeck, S. .0568, 0568 Opy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0560 Osburn, S. .0508, 0756 Ose, J. .0044 Osorio, R. S. .0011 Osorio, R. S. .0863, 0864, 1153 Ostan, A. .0929
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .00107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Sonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Opy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0508, 0756 Oserio, R. S. .0508, 0756 Oserio, R. S. .0863, 0864, 1153 Osorio, R. S. .0863, 0864, 1153 Ostan, A. .0929 Overeem, S. .0673, 0673, 0763
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .00107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Opy, K. .0883, 0907 Orff, H. J. .08866 Orr, J. E. .0508, 0756 Oserio, R. S. .0508, 0756 Osorio, R. S. .0044 Osorio, R. S. .0863, 0864, 1153 Ostan, A. .0929 Overeem, S. .0673, 0673, 0763 Owens, J. .0408, 1179, 1181
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .00107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Opy, K. .0883, 0907 Orff, H. J. .08866 Orr, J. E. .0508, 0756 Oserio, R. S. .0508, 0756 Oserio, R. S. .0863, 0864, 1153 Ostrio, R. S. .0863, 0864, 1153 Ostrio, R. S. .0673, 0673, 0763 Overeem, S. .0673, 0763 Owens, J. .0408, 1179, 1181 Owens, R. .0611, 0690
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Opy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0560 Osburn, S. .0508, 0756 Oserio, R. S. .1150 Osorio, R. S. .0011 Osorio, R. S. .0673, 0673, 0763 Overeem, S. .0673, 0673, 0763 Owens, J. .0408, 1179, 1181 Owens, R. L. .0560
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Sonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Oppy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0560 Osburn, S. .0508, 0756 Ose, J. .0044 Osorio, R. S. .0663, 0864, 1153 Ostan, A. .0929 Overeem, S. .0673, 0673, 0763 Owens, J. .0408, 1179, 1181 Owens, R. L. .0560 Owusu, J. T. .0560
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. .0107 Ong, J. .00519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Šonka, K. .0740, 0753 Ono, T. .0757 Onyconwu, C. .0865 Op De Beeck, S. .0568, 0568 Oppy, K. .0883, 0907 Orff, H. J. .08866 Orr, J. E. .0560 Osburn, S. .0508, 0756 Ose, J. .00444 Osorio, R. S. .1150 Osorio, R. S. .0673, 0673, 0763 Overeem, S. .0673, 0673, 0763 Owens, J. .0408, 1179, 1181 Owens, R. L. .0560 Owens, R. L. .0560 Owens, R. L. .0560
Olson, T. .0719, 0719 Omlin, X. .0548 Omori, Y. .0757 O'Mullane, B. .0412, 0412, 0443, 1214 Ong, J. .0107 Ong, J. C. .0519, 0519, 0655, 0754, 0824, 0824, 1151 Ong, J. .0092, 0143, 0519, 0519 Oniani, N. .0327 Oniani, T. .0327 Sonka, K. .0740, 0753 Ono, T. .0757 Onyeonwu, C. .0865 Op De Beeck, S. .0568, 0568 Oppy, K. .0883, 0907 Orff, H. J. .0866 Orr, J. E. .0560 Osburn, S. .0508, 0756 Ose, J. .0044 Osorio, R. S. .0663, 0864, 1153 Ostan, A. .0929 Overeem, S. .0673, 0673, 0763 Owens, J. .0408, 1179, 1181 Owens, R. L. .0560 Owusu, J. T. .0560

Ozaki, N	575
Ozuru, Y	06

P

Pace, D	.0513, 053	32
Pace-Schott, E. F		
Pack, A	.0054, 005	54
Pack, A. I	0569, 102	20
Paech, G. M.	074	18
Page, J	098	39
Pahwa, V.	060)0
Pain, S.		
Paine, SJ.		
Pak, V. M		
Pal, A		
Palacharla, R		
Palchik, G. A.		
Paller, K. A.		
Palling, D.		
Palme, C. E.		
Palmer, C. A.		
Palombini, L.		
Palombini, L. O.		
Palomo, R		
Palotai, M.		
PANAGIOTAKIS, S.		
Panda, S		
Pandey, A		
Pandey, A		
Pandey, S.		
Pandya, V.		
Panek, D		
Paquet, C		
Paquette, T		
Parathasarathy, s	117	
Pardilla-Delgado, E		
	042	21
Parekh, A		21 53
Parekh, A		21 53 44
Parekh, A		21 53 44 97
Parekh, A		21 53 44 97
Parekh, A		21 53 44 97 28
Parekh, A		21 53 44 97 28 58 79
Parekh, A		21 53 44 97 28 58 79
Parekh, A. .0838, 0838, 0966, Parekh, A. A.		21 53 44 97 28 58 79 95 72
Parekh, A. .0838, 0838, 0966, Parekh, A. A.		21 53 44 97 28 58 79 95 72
Parekh, A. .0838, 0838, 0966, Parekh, A. A.		21 53 44 97 28 58 79 55 72 47
Parekh, A		21 53 44 97 28 58 79 95 72 47 51
Parekh, A. .0838, 0838, 0966, Parekh, A. A.		21 53 44 97 28 57 28 57 28 57 28 57 28 57 29 57 27 27 71
Parekh, A. .0838, 0838, 0966, Parekh, A. A. Parizi-Robinson, M. Park, C. J. Park, E. Park, H. Park, J. G. Park, K. J. Park, K. J. Park, M. Park, S. Park, YM.M.		21 53 44 97 28 57 95 72 47 51 71 16
Parekh, A		21 53 44 97 28 57 28 57 28 57 28 57 28 57 28 57 28 57 28 57 29 57 27 27 17 71 16 71
Parekh, A. .0838, 0838, 0966, Parekh, A. A. Parizi-Robinson, M. Park, C. J. Park, E. Park, H. Park, J. G. Park, J. N. Park, K. J. Park, K. J. Park, S. Park, YM.M. Park', K. Park, J. L.		21 53 44 97 28 58 79 572 47 51 71 16 71 22
Parekh, A. .0838, 0838, 0966, Parekh, A. A.		21 53 44 97 28 57 28 57 28 57 28 57 27 51 71 6 71 22 33
Parekh, A. .0838, 0838, 0966, Parekh, A. A.		21 53 44 97 28 57 95 72 71 71 71 71 71 71 71 71 71 71 71 71 71
Parekh, A. .0838, 0838, 0966, Parekh, A. A.		21 53 44 77 86 79 72 87 70 71 71 71 71 71 71 71 71 71 71 71 71 71
Parekh, A. .0838, 0838, 0966, Parekh, A. A.		21 53 44 77 88 79 72 87 70 71 71 71 71 71 71 71 71 71 71 71 71 71
Parekh, A. .0838, 0838, 0966, Parekh, A. A.		21 53 44 72 88 79 57 27 71 71 71 71 23 88 13 73 8,
Parekh, A		21 53 44 72 86 79 57 71 71 71 71 71 71 71 71 73 88 73 89 73 89
Parekh, A		21 53 44 97 28 57 95 72 71 71 71 71 71 71 71 73 88 73 8, 97 73 73 73 73 73 73 73
Parekh, A		21 53 44 97 28 57 95 72 77 71 71 71 71 71 71 71 73 88 73 89 72 73 73 73 72 73 73 73 72 73
Parekh, A		21 54 72 54 72 58 75 71 71 71 72 33 81 73 80 72 70 72 70 72 70 72 70 72 70 72 70 70 70 70 70 70 70 70 70 70 70 70 70
Parekh, A		21 53 54 44 97 72 28 86 87 9 95 57 22 87 72 72 72 72 72 73 88 83 83 83 83 73 88,99 96 838 837 99 96 838

Patel, A
Patel D 0747
$1 a_{1} a_{1} a_{2} a_{3} a_$
Patel, R
Patel, R. K
Patel, S
Patel, S. I
Patel, S. L
Patel, S. R
Pathan, Z
Patroneva, A
Patten, L
Patterson, C. M
Patterson, F
Pattinson, C. L
Pattinson, C
Paul, K
Pavlova, M
Pavuluri, H
Pawlowska-Wajswol, S
Pawlowska-Wajswol, S. J
Paxton Willing, M. M
Payano, L
Payne, J. D
-
Peach, H
Pearson, H
Pearson, S
Pedersen, S
Peedin, M
Peine, N. A
Peker, Y
Peleckis, A. J
Penedo, F
Peng, V
Pennell, C. E
Pennestri, MH
Penzel, T
Peoples, A. R
PEPIN, E
Pepin, JL
•
Peppard, P
Peppard, P. E
Peprah, R
Perdomo, C
Perez, E
Perez, E
Perez, E
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456 Perlis, M. L. .0373, 0456, 0456
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456 Perlis, M. L.
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456 Perlis, M. L. .0120, 0152, 0153, 0351, 0404, 0405, 0464, 0544, 0868, 1093, 1096,
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456 Perlis, M. L. .0373, 0456, 0456
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perni, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456 Perlis, M. L. .0373, 0456, 0456 Perlis, M. L. .0373, 0454, 0868, 1093, 1096, 1108, 1109, 1111, 1174, 1175 Perreault, L. .0132
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456 Perlis, M. L. .0373, 0456, 0456
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perni, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456 Perlis, M. L. .0373, 0456, 0456 Perlis, M. L. .0373, 0454, 0868, 1093, 1096, 1108, 1109, 1111, 1174, 1175 Perreault, L. .0132
Perez, E. .0213 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456 Perlis, M. L. .0373, 0456, 0456 Perlis, M. L. .0120, 0152, 0153, 0351, 0404, 0405, 0464, 0544, 0868, 1093, 1096, 1108, 1109, 1111, 1174, 1175 Perreault, L. .0132 Perrin, P. B. .1123
Perez, E. .0213 Perez, E. .0537, 1123 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456 Perlis, M. L. .0120, 0152, 0153, 0351, 0404, 0405, 0464, 0544, 0868, 1093, 1096, 1108, 1109, 1111, 1174, 1175 Perreault, L. .0132 Perrin, P. B. .1123 Peszka, J. .0180, 0194 Petelle, B. .0665
Perez, E. .0213 Perez, E. .0537, 1123 Perez, E. .0537, 1123 Perez, I. A. .0874, 0887 Perez, J. .0749 Perez, N. .1020 Pérez-Stable, E. J. .0361 Perini, F. .0519, 0519, 0824, 0824 Peris, T. .0997 Peris Sempere*, V. .0773 Perlis, M. .0373, 0456, 0456 Perlis, M. L. .0120, 0152, 0153, 0351, 0404, 0405, 0464, 0544, 0868, 1093, 1096, 1108, 1109, 1111, 1174, 1175 Perreault, L. .0132 Perrin, P. B. .1123 Peszka, J. .0180, 0194

Petersen, R. C.	
Peters-Mathews, B.	
Peterson, A	.0483, 0483, 0860, 1073, 1186
Peterson, A. L	
Peterson, B	
Peterson, S	
Petitjean, M	
Petitto, L	
Petlu, S	
Petrov, M. E	
Petrovick, M	
Pfaffinger, B	
Phadnis, M	
Pham, H	
Pham, J	
Phan, S	
Phillips, A. J.	
Phillips, K	
Phipps, A	
Phung, A	
Piacentini, J	
Picard-Deland, C	
Picchietti, D	
Pichardo, Y	
Pickett, S. M.	
Pickett, T. C	
Pien, G	
Pigeon, W.	
Pigeon, W. J.	
Pigeon, W. R.	
Pike, K. C	
Pilcher, J. J.	
Pillai, J	
Piltch, O	
Pinaud, C	
Pineda, L	
Pinner, K	
Pinnington, D. M	
Piovezan, R. D.	
Piper, D	
Pires, G.	
Pires, G. N.	
Pirner, M	
Piro, B	
Pitt, S	
Pizza*, F	
Plante, D. T.	
Platt, J	
Platt, R. S	
Platter, L	
Plazzi, G	
Plazzi**, G	
Plog, A. E	
Plunkett, K.	
Png, C. A	
Poe, A. R	
Poewe, W	
Pogach, M	
Poh, Y	
Poke, P	
Pollet, D. P	
Pollet, E. P	
Polos, P	

Polymeropoulos, C
Polymeropoulos, M
Poornima, S
Porro, A
Porter, C
Porter, K. I
Posada-Quintero, H0055
Postolache, T. T
Potvin, J
Poupore-King, H
Powell, E
Powell, T
Powell, W
Poyares, D
Pradeepan, S
Pradhan, S
Prasad, B
Prather, A
Prats, N
Prerau, M
Prerau, M. J
PRESSNITZER, D
Price, S. N
Price, S
Profant, J
Prouty, D
Provencio-Dean, N
Pruiksma, K
Pruiksma, K. E
Pruss, K. K
Przybelski, S. A
Punjabi, N. M
Punnett, L
Puravath, F. M
Purnell, J. Q
Puzino, K
Puzino, K
Tuzino, K
0458, 0476, 0506, 0585, 0742, 0878, 0919, 0936, 1107, 1130
PVDOMICS, P
1.201.200, 1.1.1.1.1.1.1.1.1.1.1.1.1.1.1.1.1.1.1.

Q

Qadri, S
Quach, J
QUAN, P
Quan, S
Quan, S. F0033, 0251, 0572, 0578, 0717, 0826, 1187, 1188, 1188
Quartana, P. J
Quattom, M
Quattrucci, J
Quevedo, Y. L
Quigley, D
Quin, N
Quinn, K
Quintero, L
Quintos, A
Quiquempoix, M
Quiroz, Y. T
Quispe, K. A
Quock, R. M
Qureshi, M
Qutub, A. A

R

Rabat, A	
Rader, D. J	
Radha, M	
Radix, A	
Radom-Aizik, S	0325, 0335
Raffin Bouchal, S	
Ragas, T. L	
Raghav, P	
Raghunathan, R	
Raghunathan, S	
Rahill, G. J	
Rahman, S. A.	
Rahman, S. N.	
Raikes, A. C	
0014, 0070, 0079, 0080, 0082, 0227, 0305, 030	5, 0314, 1158, 1158,
	1160
Raine, P	
Rains, J. C.	
Raj, A	2, 0412, 0443, 1214
Rajan, P	
Rajaratnam, S. M.	
Rajaratnam, S. W.	
Rakus, A	
Ralston, K.	
Ramaekers, J.	
RAMAGOPAL, M	
Ramakrishnan, S	
Raman, S	
Ramirez Gomez, L.	
Ramirez-Ruiz, M.	
Ramos, A	
Ramos, A.	

Raynor, H. A
Razjouyan, J
Reballi, V
Rebok, G. W
Rech, M
Rechul, D
Rechul, K
Redding, G
Reddy, A
Reddy, A
Redeker, N. S
Redline, S 0048, 0318, 0350, 0363, 0388, 0419, 0423, 0593, 0593,
0607, 0607, 0609, 0624, 0641, 0784, 0850, 1007, 1097, 1144, 1164
Rehm, I. C
Reichenberger, D. A
Reichert, J
Reid, K
Reid, K. J
Reid, M
Reid, M. J
Reid, M
Reidy, J
Reifman, J
Reiling, K
Reisdorph, N
Reitz, S
Reljic, T
-
Rengan, R
Repasky, E
Resick, P
Reuveny, A
Reyner, L
Reynolds, A. M
Reynolds, C
Reynolds, C
Powelds D 1029
Reynolds, D
Reynolds, M. A1061
Reynolds, M. A
Reynolds, M. A1061
Reynolds, M. A
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Riccit, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .1074
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .00438
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .00438 Richards, K. C. .0861
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .0438 Richards, K. C. .0861 Richards, S. .1212
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .00438 Richards, K. C. .0861
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .0438 Richards, K. C. .0861 Richards, S. .1212
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Riccit, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .1074 Richards, K. .0438 Richards, S. .1212 Richards, R. .0240 Richards, S. .1212 Richards, R. .02606, 0608, 0620, 1155, 1155
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .1074 Richards, K. .0438 Richards, S. .1212 Richardson, R. .0606, 0608, 0620, 1155, 1155 Richmond, T. S. .0365
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .0071 Richards, K. .0438 Richards, K. .0438 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, M. .020 Rickels, M. R. .020 Ricketti, P. .0608
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .00714 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, M. .0274 Richards, S. .1212 Richards, M. .0438 Richards, S. .1212 Richards, S. .1212 Richards, S. .1212 Richardson, R. .0606, 0608, 0620, 1155, 1155 Richerds, M. R. .020 Ricketti, P. .0608 Ricketts, E. .0997
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .00438 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, S. .1212 Richards, S. .1212 Richards, M. R. .0606, 0608, 0620, 1155, 1155 Richmond, T. S. .0365 Rickels, M. R. .1020 Ricketti, P. .0608 Ricketts, E. .0997 Riedner, B. .0340, 1003
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .00714 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, S. .0606, 0608, 0620, 1155, 1155 Richerds, M. R. .00365 Rickets, E. .0997 Riedner, B. .0340, 1003 Riedner, B. A. .0422, 0808, 0808
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .00714 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, M. R. .0606, 0608, 0620, 1155, 1155 Richerds, M. R. .00606 Ricketti, P. .0608 Ricketts, E. .0997 Riedner, B. A. .0422, 0808, 0808 Riedy, S. M. .0179
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .0071 Richards, K. .0438 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, K. .0438 Richards, S. .1212 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, M. .0606, 0608, 0620, 1155, 1155 Richerds, M. R. .00365 Ricketts, E. .0997 Riedner, B. .0340, 1003 Riedner, B. A. .0422, 0808, 0808 Riedy, S. M. .0179 Riggins, T. .0089, 0089, 0334
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .0174 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, S. .1212 Richards, S. .1212 Richards, K. .0438 Richards, S. .1212 Richards, S. .1212 Richards, S. .1212 Richards, M. R. .0606, 0608, 0620, 1155, 1155 Ricketts, E. .0997 Ricketts, E. .0997 Riedner, B. A. .0422, 0808, 0808 Riedy, S. M. .0179 Riggins, T. .0089, 0089, 0334 Ripple, C. H. .1179, 1181
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .0071 Richards, K. .0438 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, K. .0438 Richards, S. .1212 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, M. .0606, 0608, 0620, 1155, 1155 Richerds, M. R. .00365 Ricketts, E. .0997 Riedner, B. .0340, 1003 Riedner, B. A. .0422, 0808, 0808 Riedy, S. M. .0179 Riggins, T. .0089, 0089, 0334
Reynolds, M. A. .1061 Rezayat, T. .0715 Rezende, T. .0657, 0804 Rheingold, A. .1186 Rho, YA. .0124 Rhodes, C. .1103 Ricci, A. .0319 Ricci, E. .0359 Riccitelli, G. .1122, 1122 Rice, B. .1132 Rice, C. .1064 Rice, L. .0870 Richards, A. .0174 Richards, K. .0438 Richards, K. .0438 Richards, S. .1212 Richards, S. .1212 Richards, S. .1212 Richards, K. .0438 Richards, S. .1212 Richards, S. .1212 Richards, S. .1212 Richards, M. R. .0606, 0608, 0620, 1155, 1155 Ricketts, E. .0997 Ricketts, E. .0997 Riedner, B. A. .0422, 0808, 0808 Riedy, S. M. .0179 Riggins, T. .0089, 0089, 0334 Ripple, C. H. .1179, 1181

Ritchie, L
Ritterband, L
Ritterband, L. M
Rivera, G
Rivera, S
Riviere, L. A
Roache, J
Roane, B
Robbins, G
Robbins, R.
0251, 0408, 0444, 0964, 1046, 1046, 1058, 1058, 1179, 1181, 1187,
1212
Roberto, L
Roberts, A. C
Roberts, S. A
Roberts, Z
Robins, C
Robinson, M
Robinson, S. E
Robison, L. L
Rocha, S
Rode, N
Rode, S
Rođe, S
Rodriguez, R
Roecklein, K. A
Roehrs, T
Rogers, A. E
Rogers, A. E
Rognvaldsdottir, V
P_{olemon} (-
Roisman, G
Roizenblatt, S
Roizenblatt, S
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .1089
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .1089 Rosen, C. L. .0950
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .1089 Rosen, C. L. .0950 Rosen, I. M. .1184
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .0950 Rosen, C. L. .0950 Rosenberg, C. .0576
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .0950 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. .0404, 0405, 1187
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .0950 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. A. .0120
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .0950 Rosen, C. L. .0950 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. A. .0120 Rosenberg, R. .0428, 0481, 0751
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronka, S. .1200 Rorie, K. .0749 Rosen, C. L. .0950 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. A. .0120 Rosenberg, R. .0404, 0441, 0751 Rosendahl-Garcia, K. M. .0041, 0041, 0041, 0042
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .06950 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. A. .0120 Rosenberg, R. .0478, 0481, 0751 Rosendall-Garcia, K. M. .0041, 0041, 0042 Rosenfield, B. .0152, 0351, 1109, 1111
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .06950 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. A. .0120 Rosenberg, R. .0404, 0405, 1187 Rosenberg, R. .0478, 0481, 0751 Rosendahl-Garcia, K. M. .00478, 0431, 0041, 0042 Rosenthal, M. .0152, 0351, 1109, 1111 Rosenthal, M. .0645
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .00950 Rosen, C. L. .0950 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. A. .0120 Rosenberg, R. .0478, 0481, 0751 Rosenberg, R. .0478, 0481, 0751 Rosenfield, B. .0152, 0351, 1109, 1111 Rosenthal, M. M. .1044, 1044
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .00950 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. A. .0120 Rosenberg, R. .0404, 0405, 1187 Rosenberg, R. .0478, 0481, 0751 Rosenberg, R. .0478, 0481, 0751 Rosenthal, M. .0152, 0351, 1109, 1111 Rosenthal, M. .1044, 1044 Rosenthal, Z. P. .0126
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .00950 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. .0404, 0405, 1187 Rosenberg, R. .0120 Rosenberg, R. .0120 Rosenberg, R. .0441, 0041, 0042 Rosenberg, R. .0152, 0351, 1109, 1111 Rosenthal, M. .1044, 1044 Rosenthal, M. .1044, 1044 Rosenthal, Z. P. .0126 Roseus, J. .0373, 0377
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .0749 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. A. .0120 Rosenberg, R. .0448, 041, 0042 Rosenberg, R. .0478, 0481, 0751 Rosenberg, R. .0152, 0351, 1109, 1111 Rosenthal, M. .0645 Rosenthal, M. .1044, 1044 Rosenthal, Z. P. .0126 Rosenthal, Z. P. .0126 Rosens, M. .0435, 0436
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .0749 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. .0448, 0481, 0751 Rosenberg, R. .0120 Rosenberg, R. .0478, 0481, 0751 Rosenberg, R. .0041, 0041, 0042 Rosenthal, M. .0152, 0351, 1109, 1111 Rosenthal, M. .1044, 1044 Rosenthal, M. .1044, 1044 Rosenthal, Z. P. .0126 Rosens, M. .0435, 0436 Rostain, A. .1111
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .06950 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. .0448, 0481, 0751 Rosenberg, R. .0478, 0481, 0751 Rosenthal, M. .0041, 0041, 0042 Rosenthal, M. .0152, 0351, 1109, 1111 Rosenthal, M. .1044, 1044 Rosenthal, M. .1026 Rosenthal, Z. P. .0126 Rosenthal, Z. P. .0126 Rosenthal, Z. P. .0126 Rosenthal, A. .0117 Rosenthal, A. .1111 Rostain, A. .1111
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .06950 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. .0448, 0481, 0751 Rosenberg, R. .0478, 0481, 0751 Rosenthal, M. .0041, 0041, 0042 Rosenthal, M. .0152, 0351, 1109, 1111 Rosenthal, M. .0044, 1044 Rosenthal, M. .0126 Rosenthal, M. .0043, 0435, 0436 Rosenthal, Z. P. .0126 Rosens, M. .0435, 0436 Rostain, A. .1111 Rostain, A. .1111 Rostain, A. .1111 Rostain, A. .0128
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .06950 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. .0449, 0405, 1187 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. A. .0120 Rosenberg, R. .0478, 0481, 0751 Rosenberg, R. .0478, 0441, 0042 Rosenthal, M. .0041, 0041, 0042 Rosenthal, M. .0152, 0351, 1109, 1111 Rosenthal, M. .0126 Rosenthal, Z. P. .0126
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .0108 Rosen, C. L. .0950 Rosen, I. M. .1184 Rosenberg, C. .0576 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. .0448, 0751 Rosenberg, R. .0478, 0481, 0751 Rosenthal, Garcia, K. M. .0041, 0041, 0042 Rosenthal, M. .0152, 0351, 1109, 1111 Rosenthal, M. .0126 Rosenthal, M. .0435, 0436 Rosenthal, M. .0435, 0436 Rosenthal, A. .01170 Rosenthal, M. .0435, 0436 Rosenthal, A. .01111 Rosenthal, M. .0435, 0436 Rosenthal, M. .0435, 0436 Rosenthal, A. .0128 Roth, A.
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .1089 Rosen, C. L. .0950 Rosenberg, C. .0576 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. A. .0120 Rosenberg, R. .0478, 0481, 0751 Rosenherg, R. .0478, 0481, 0751 Rosenthal, M. .0041, 0041, 0042 Rosenthal, M. .0152, 0351, 1109, 1111 Rosenthal, M. .0126 Rosenthal, M. .0126 Rosenthal, M. .0126 Rosenthal, M. .0126 Rosenthal, A. .0110 Rosenthal, M. .0126 Rosenthal, M. M. .01435, 0436 Rosenthal, A. .01109 Rosenthal, A. .0110
Roizenblatt, S. .0937 Rojo-Wissar, D. M. .0139, 0353, 0988 Rojrattanadumrong, R. .0992 Rollman, B. L. .0489, 0489 Romero, E. K. .1154 Romero, H. E. .0573 Ronda, J. M. .0117, 0382 Ronka, S. .1200 Rorie, K. .0749 Rosal, M. .0108 Rosen, C. L. .0950 Rosen, I. M. .1184 Rosenberg, C. .0576 Rosenberg, E. .0404, 0405, 1187 Rosenberg, E. .0448, 0751 Rosenberg, R. .0478, 0481, 0751 Rosenthal, Garcia, K. M. .0041, 0041, 0042 Rosenthal, M. .0152, 0351, 1109, 1111 Rosenthal, M. .0126 Rosenthal, M. .0435, 0436 Rosenthal, M. .0435, 0436 Rosenthal, A. .01170 Rosenthal, M. .0435, 0436 Rosenthal, A. .01111 Rosenthal, M. .0435, 0436 Rosenthal, M. .0435, 0436 Rosenthal, A. .0128 Roth, A.

Rothenberger, S. .0216 Rottapel, R. .0624 Rowatt, W. .0193 Rowe, M. .0501 Rowlett, J. K. .0151 Roy, A. .0740, 0768
Roy, J
Roy, M. J
Royer, A
Rozenman, M
Rubens, S. L
Rucker, J
Rudd, E
Ruder, M0412, 0412, 0443, 1214
Rueschman, M
Ruggero, C
Ruggero, C. J
Ruggero, C. R
Ruggiero, K
Ruggiero, L
Rumble, M0472
Ruoff, L
Rupp, H. R
Rus, H
Rus, H. M
Rusinek, H
Rusk, S
Russell, A
Russell, J. A
Ruth, C
Rye, D

S

Sabet, S
Sabet, S. M
Sabla, G
Sabzpoushan, A
Saconi, B
Sadato, N
Sagong, C
Sahlem, G. L
Sahota, P
Sahota, P. K
Sakamoto, Y
Sakhelashvili, I
Sakthiakumaran, A
Sakurai, Y
Salazar, J
Saletin, J. M
Salgado, M
Salinas, G
Salk, R. H
Salloum, A
Salsone, M
Samman, H
Sampat, A
Sampogna, S
Sanchez, E
Sanchez, H
Sanchez, H. O
Sander, H. H
Sanders, M

Sandhu, A.	
Sandler, D. P.	
Sandness, D. J.	
Sands, S	
Sands, S. A	
Sangha, R	
Sankari, A.	
Santamaria, A	
Santiago, A.	
Santiago, B. P.	0384
Santisteban, J.	
Santos, A. B	
Santos, A. A.	
Santos-Fernandez, E	0210
Santos-Junior, J. G	
Sanzo, G	
Saoud, J. B	
Saper, C. B	0148
Sarmiento, K.	
Sarzetto, F	
Sasmita, K	
Satake, M.	0518
Satoh, M.	
Sattari, N	
Satterfield, B. C	0274, 0306, 0308, 0316
Satyanarayana, S	
Saul, C	
Sauvet, F	
Savenkova, M. I	
Savitz, A.	
Sawyer, A. M.	
Saxena, R	.0016, 0016, 0259, 1164
G 1 I	
Sayed, J	
Sayed, J	
Saylor, J	
Saylor, J	
Saylor, J Sbarboro, J Scammell, T	
Saylor, J Sbarboro, J Scammell, T	
Saylor, J. Sbarboro, J. Scammell, T. Scammell, T. E.	
Saylor, J Sbarboro, J Scammell, T Scammell, T. E Scammell, T. E.	
Saylor, J Sbarboro, J Scammell, T Scammell, T. E Scammell, T. E Scammell, T Scammell, T	
Saylor, J. Sbarboro, J. Scammell, T. Scammell, T. E. Scammell, T. Scammell, T. Scammell, T. Scammell**, T. Schaaf, C.	
Saylor, J. Sbarboro, J. Scammell, T. Scammell, T. E. Scammell, T. Scammell, T. Scammell, T. Scammell**, T. Schaaf, C.	
Saylor, J	

Scholz, S.		
Schonberg, M. .0624 Schousboe, J. .0388 Schrack, J. A. .0135, 0139, 0353, 1137 Schredl, M. .0112 Schurz, King, P. .0044 Schutte, M. .0801 Schutz, S. G. .0622 Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwatz, A. R. .0702, 0703 Schwatz, J. D. .0606, 1136, 1177, 1177 Schwatz, D. .06061, 136, 1136, 1177, 1177 Schwatz, J. C. .0708 Schwatz, S. W. .0667, 0806, 0816 Schwatz, S. W. .0667, 0807 Scott. Stuherland, J.	Scholz, S	0750
Schonberg, M. .0624 Schousboe, J. .0388 Schrack, J. A. .0135, 0139, 0353, 1137 Schredl, M. .0112 Schurz, King, P. .0044 Schutte, M. .0801 Schutz, S. G. .0622 Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwatz, A. R. .0702, 0703 Schwatz, J. D. .0606, 1136, 1177, 1177 Schwatz, D. .06061, 136, 1136, 1177, 1177 Schwatz, J. C. .0708 Schwatz, S. W. .0667, 0806, 0816 Schwatz, S. W. .0667, 0807 Scott. Stuherland, J.	Schommer, J.	
Schott, A. .0071, 0074 Schousboe, J. .0388 Schrack, J. A. .0135, 0139, 0353, 1137 Schrack, J. A. .0135, 0139, 0353, 1137 Schrack, J. A. .0135, 0139, 0353, 1137 Schutt, M. .0044 Schuttz, S. G. .0642 Schutz, S. G. .0662 Schwab, R. J. .0527, 0424, 0637, 0674, 1057 Schwatz, S. G. .0666, 0569 Schwartz, D. J. .1124 Schwartz, D. J. .1124 Schwartz, D. J. .1124 Schwartz, S. W. .0667, 0866, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, C. G. .0355 Schwarz, J. .04460, 0460 Schweitzer, P. K. .0150, 0280, 0282 Scictle, E. J. .0171, 0280, 0282 Scictle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .192, 1199 Scott, P. W. .1056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .06608 Sebastiao, Y. V.		
Schousboe, J. .0388 Schrack, J. A. .0135, 0139, 0353, 1137 Schredl, M. .0112 Schratz, K. J. A. .0135, 0139, 0353, 1137 Schredl, M. .0044 Schutz, S. G. .0602 Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwatz, A. R. .0702, 0703 Schwartz, D. .0606, 1136, 1136, 1177, 1177 Schwartz, D. J. .1124 Schwartz, J. J. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .06641, 0693, 0772 Schwartz, S. W. .06641, 0693, 0772 Schwartz, P. K. .0150, 0280, 0282 Scircle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, H. .192, 1199 Scott, P. W. .0664 Scutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scutherland, J. .0233, 0279, 0279 Scuderi, N. .0664 Scutherland, J. .0233, 0279, 0279 Sc	6,	
Schrack, J. A. .0135, 0139, 0353, 1137 Schrotz-King, P. .0044 Schutte, M. .0801 Schutz, S. G. .0602 Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwab, R. J. .0562, 0569 Schwatz, A. R. .0702, 0703 Schwatz, D. .0606, 1136, 1136, 1177, 1177 Schwatz, D. .0606, 1136, 1136, 1177, 1177 Schwatz, I. P. .0144, 0218 Schwatz, S. W. .0667, 0806, 0816 Schwatz, I. P. .0144, 0218 Schwatz, S. W. .0667, 0806, 0816 Schwatz, P. M. .0150, 0280, 0282 Scitlee, F. J. .0171 Scott. .0673, 0772 Schwatz, P. K. .0150, 0280, 0282 Scott, H. .1192, 1199 Scott, S. .0641, 0693, 0772 Schwatz, P. K. .0150, 0280, 0282 Scott, S. .0641, 0693, 0772 Schwatz, P. K. .0167, 0398 </td <td></td> <td>,</td>		,
Schredl, M. .0112 Schutz-King, P. .0044 Schutte, M. .0801 Schutz, S. G. .0622 Schwab, R. J. .0237, 0424, 0637, 0674, 1057 Schwatz, A. R. .0702, 0703 Schwatz, A. R. .0702, 0703 Schwatz, D. J. .1124 Schwatz, D. J. .1124 Schwatz, JC. .0768 Schwatz, J. P. .0184, 0218 Schwatz, S. W. .0667, 0806, 0816 Schwatz, S. W. .0667, 0806, 0816 Schwatz, S. W. .0667, 0806, 0816 Schwatz, S. W. .0661, 0630, 0722 Schwatz, P. S0150, 0280, 0282 Scircle, E. J. Schweitzer, P. K. .0150, 0280, 0282 Scircle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0604 Scullin, M. K. .0667 Scela, M. .037, 1045 Sebastião, Y. V. .0667, 0806 <td></td> <td></td>		
Schrotz-King, P. .0044 Schunacher, S. .0184, 0218 Schütz, S. G. .0602 Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwatz, A. R. .0702, 0703 Schwartz, D. .0666, 1136, 1136, 1177, 1177 Schwartz, D. .0666, 1136, 1136, 1177, 1177 Schwartz, D. .0666, 0816 Schwartz, L. P. .0184, 0218 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, I. .0460, 0460 Schwartz, P. Schwartz, S. W. .06641, 0693, 0772 Schweitzer, P. Scott, B. .0178, 0398 Scott, E. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0067	Schrack, J. A	0353, 1137
Schulte, M. .0801 Schuracher, S. .0184, 0218 Schütz, S. G. .0602 Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwab, R. J. .0562, 0569 Schwartz, A. R. .0702, 0703 Schwartz, D. .0606, 1136, 1136, 1177, 1177 Schwartz, D. .0606, 1136, 1136, 1177, 1177 Schwartz, D. .0184, 0218 Schwartz, S. W. .0607, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, C. G. .03355 Schwartz, P. .0641, 0693, 0772 Schweitzer, P. K. .0150, 0280, 0282 Scictel, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .0233, 0279, 0279 Scullin, M. K. .0233, 0279, 0279 Scullin, M. K.	Schredl, M	0112
Schulte, M. .0801 Schuracher, S. .0184, 0218 Schütz, S. G. .0602 Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwab, R. J. .0562, 0569 Schwartz, A. R. .0702, 0703 Schwartz, D. .0606, 1136, 1136, 1177, 1177 Schwartz, D. .0606, 1136, 1136, 1177, 1177 Schwartz, D. .0184, 0218 Schwartz, S. W. .0607, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, C. G. .03355 Schwartz, P. .0641, 0693, 0772 Schweitzer, P. K. .0150, 0280, 0282 Scictel, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .0233, 0279, 0279 Scullin, M. K. .0233, 0279, 0279 Scullin, M. K.	Schrotz-King P	0044
Schumacher, S. .0184, 0218 Schütz, S. G. .0602 Schwab, R. J. .0237, 0424, 0637, 0674, 1057 Schwab, R. J. .0237, 0424, 0637, 0674, 1057 Schwah, R. J. .00066, 1136, 1136, 1177, 1177 Schwartz, D. J. .1124 Schwartz, JC. .0702 Schwartz, S. W. .0667, 0866, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schweitzer, P. .0440, 0460 Schweitzer, P. K. .0150, 0280, 0282 Scircle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, H. .1026 Scouderi, N. .0664 Scullin, M. K. .00176, 0369, 0369, 0391 Scwhartz, D. .0608 Selastiao, Y. V. .0667, 0806 Sebastiao, Y. V. .0667, 0806 Sebastiao, Y. V. .0667, 0806 Seewald, M. .0152, 0153, 1109, 1111 Segust, S. .0152 Sedky, K. .00000 Seewald, M. .0152, 0153, 1109, 1111	e.	
Schütz, S. G. .0602 Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwab, R. J. .0562, 0569 Schwartz, A. R. .0702, 0703 Schwartz, D. .0606, 1136, 1136, 1177, 1177 Schwartz, JC. .0768 Schwartz, L. P. .0184, 0218 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwarz, C. G. .03355 Schwarz, S. W. .0641, 0693, 0772 Schweitzer, P. K. .0150, 0280, 0282 Scicte, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .10266 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .06604 Scullin, M. K.		
Schwab, R. .0237, 0424, 0637, 0674, 1057 Schwab, R. J. .0502, 0569 Schwartz, A. R. .0702, 0703 Schwartz, D. .0606, 1136, 1136, 1177, 1177 Schwartz, D. .0164, 0218 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0663, 0772 Schwartz, P. .0150, 0280, 0282 Scircle, F. J. .0171 Scott, B. .0178, 0398 Scott, P. W. .0126 Scott, P. W. .0126 Scullin, M. K. .00233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0667, 0806 Sebastião, Y. V. .0816 Sebastião, Y. V. .0816 </td <td></td> <td></td>		
Schwab, R. J. .0562, 0569 Schwartz, A. R. .0702, 0703 Schwartz, D. J. .1136, 1136, 1177, 1177 Schwartz, D. J. .1124 Schwartz, L. P. .0184, 0218 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, I. .0460, 0460 Schwartz, P. K. .0150, 0280, 0282 Scircle, F. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .0056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0064 Scullin, M. K. .0667, 0806 Sebastião, Y. V. .0672, 0153, 1109, 1111 Sebastião, Y. V. .0673, 00745, 0747 Sedastião, Y. V. .0152, 0153, 1109, 1111 Segastruiriya, C. .0623 Segust, S. .0159 Seiden, D. .0128 Segastruiriya, C. .0622 Segust, S. .0159 Seiden, D. .0128 Segastruiriya, C. .06252		
Schwartz, A. R. .0702, 0703 Schwartz, D. .0606, 1136, 1136, 1177, 1177 Schwartz, D. J. .1124 Schwartz, L. P. .0768 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwarz, I. .0460, 0460 Schwarz, I. .0461, 0693, 0772 Schweitzer, P. K. .0150, 0280, 0282 Scircle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, F. W. .1056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0101, 0193, 0197, 0201, 0201, 0212, 0344, 0369, 0369, 0391 Schwartz, D. .0608 Sebastiao, Y. V. .0667 Seboskiao, Y. V. .0521 Sedky, K. .0521 Sedky, K. .0521 Sedward, M. .0152, 0153, 1109, 1111 Segarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seiden, D.	Schwab, R	0674, 1057
Schwartz, A. R. .0702, 0703 Schwartz, D. .0606, 1136, 1136, 1177, 1177 Schwartz, D. J. .1124 Schwartz, L. P. .0768 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwartz, S. W. .0667, 0806, 0816 Schwarz, I. .0460, 0460 Schwarz, I. .0461, 0693, 0772 Schweitzer, P. K. .0150, 0280, 0282 Scircle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, F. W. .1056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0101, 0193, 0197, 0201, 0201, 0212, 0344, 0369, 0369, 0391 Schwartz, D. .0608 Sebastiao, Y. V. .0667 Seboskiao, Y. V. .0521 Sedky, K. .0521 Sedky, K. .0521 Sedward, M. .0152, 0153, 1109, 1111 Segarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seiden, D.	Schwab, R. J.	0562, 0569
Schwartz, D.		
Schwartz, D. J. 1124 Schwartz, JC. 0768 Schwartz, L. P. 0184, 0218 Schwartz, S. W. 0667, 0806, 0816 Schwartz, C. G. 0355 Schwartz, I. 0460, 0460 Schwartz, I. 0460, 0460 Schwartz, R. 0150, 0280, 0772 Schweitzer, P. 0641, 0693, 0772 Schweitzer, P. K. 0150, 0280, 0282 Scott, E. J. 0171 Scott, B. 0178, 0398 Scott, H. 1192, 1199 Sctut, P. W. 1056 Scott-Sutherland, J. 0233, 0279, 0279 Scculeri, N. 0604 Scullin, M. K. 0604 Scullin, M. K. 0608 Seal, M. 1037, 1045 Sebastião, Y. V. 06667, 0806 Sebastião, Y. V. 0816 Sedox K. 0900 Seewald, M. 0152, 0153, 1109, 1111 Segust, S. 0159 Seigust, S. 0159 Seigust, S. 0159 Seigust, S. 0159 Seigust, S. 0159 <		
Schwartz, JC. .0768 Schwartz, L. P. .0184, 0218 Schwartz, S. W. .0667, 0806, 0816 Schwarz, C. G. .0355 Schwarz, J. .0460, 0460 Schwarz, I. .0460, 0460 Schweitzer, P. K. .0150, 0280, 0282 Scictle, E. J. .0178, 0398 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0017, 0469 Sceal, M. .0037, 1045 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0816 Seboak Kinter, D. .0521 Sedky, K. .0900 Seeal, M. .0152, 0153, 1109, 1111 Segesury, Y. .0128 Segasarnviriya, C. .0625 Segust, S. .0159 Sidog, 1062, 1002, 1002, 1002, 1014, 1046, 1046, 1053, 1058, 1058, 1058, 1058, 1052, 0153, 1109, 1111 Seiras, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1052, 0153, 1009, 1102, 1113, 1114, 1189, 1212, 1213 Seitas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046,		
Schwartz, L. P. .0184, 0218 Schwartz, S. W. .0667, 0806, 0816 Schwarz, C. G. .0355 Schwarz, J. .0460, 0460 Schweitzer, P. .0641, 0693, 0772 Schweitzer, P. K. .0150, 0280, 0282 Scircle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0101, 0193, 0197, 0201, 0201, 0212, 0344, 0369, 0369, 0391 Scwhartz, D. .0608 Seal, M. .0037, 1045 Sebastião, Y. V. .0608 Sebastião, Y. V. .0816 Sebeck Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifitz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1045, 1053, 1058, 1058, 1052, 1062, 1093, 1102, 1113, 114, 1189, 1212, 1213 Seixas, A. <td< td=""><td></td><td></td></td<>		
Schwartz, S. W. .0667, 0806, 0816 Schwarz, C. G. .0355 Schwarz, J. .0460, 0460 Schweitzer, P. .0641, 0693, 0772 Schweitzer, P. K. .0150, 0280, 0282 Scricle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .056 Scott-Sutherland, J. .0233, 0279, 0279 Sculeri, N. .0604 Scullin, M. K. .0013, 0197, 0201, 0201, 0212, 0344, 0369, 0369, 0391 Scwhartz, D. .0608 Seal, M. .1037, 1045 Sebastião, Y. V. .0816 Seboek Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Segesyrnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifitz, E. .0522 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1045, 1053, 1058, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 <		
Schwarz, C. G. 0355 Schwarz, J. 0460, 0460 Schweitzer, P. 0.641, 0693, 0772 Schweitzer, P. K. 0150, 0280, 0282 Scircle, E. J. 0171 Scott, B. 0178, 0398 Scott, H. 1192, 1199 Scott, P. W. 1056 Scott-Sutherland, J. 0233, 0279, 0279 Scuderi, N. 0604 Scullin, M. K. 0604 Scullin, M. K. 0604 Scwhartz, D. 06608 Seal, M. 1037, 1045 Sebastião, Y. V. 0667, 0806 Sebastião, Y. V. 0667, 0806 Sebastião, Y. V. 0667, 0806 Seewald, M. 0152, 0153, 1109, 1111 Segsarnviriya, C. 0621 Segsarnviriya, C. 0625 Segust, S. 0159 Seiten, D. 0743, 0745, 0747 Seifen, A.	Schwartz, L. P	0184, 0218
Schwarz, J. .0460, 0460 Schweitzer, P. .0641, 0693, 0772 Schweitzer, P. K. .0150, 0280, 0282 Scircle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0604 Scullin, M. K. .0604 Scullin, M. K. .0608 Seal, M. .037, 1045 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0667, 0806 Seboek Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Segev, Y. .0128 Segasmrviriya, C. .0625 Seitan, D. .0743, 0745, 0747 Seiferitz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. .0232, 0372, 0374, 0864 Sekartini, R. .0947 Semenescu, S. L. .0026	Schwartz, S. W	0806, 0816
Schwarz, J. .0460, 0460 Schweitzer, P. .0641, 0693, 0772 Schweitzer, P. K. .0150, 0280, 0282 Scircle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0604 Scullin, M. K. .0604 Scullin, M. K. .0608 Seal, M. .037, 1045 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0667, 0806 Seboek Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Segev, Y. .0128 Segasmrviriya, C. .0625 Seitan, D. .0743, 0745, 0747 Seiferitz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. .0232, 0372, 0374, 0864 Sekartini, R. .0947 Semenescu, S. L. .0026	Schwarz, C. G.	0355
Schweitzer, P. .0641, 0693, 0772 Schweitzer, P. K. .0150, 0280, 0282 Scircle, E. J. .0171 Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0101, 0193, 0197, 0201, 0201, 0212, 0344, 0369, 0369, 0391 Scwhartz, D. .0608 Seal, M. .1037, 1045 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0816 Seboek Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Segev, Y. .0128 Segasrnviriya, C. .0625 Seiden, D. .0743, 0745, 0747 Seifitz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0844 Sekartini, R. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. <t< td=""><td></td><td></td></t<>		
Schweitzer, P. K		
Scircle, E. J. 0171 Scott, B. 0178, 0398 Scott, H. 1192, 1199 Scott, P. W. 056 Scott-Sutherland, J. 0233, 0279, 0279 Scuderi, N. 0604 Scullin, M. K. 0604 Scwhartz, D. 0608 Seal, M. 1037, 1045 Sebastião, Y. V. 0667, 0806 Sebastião, Y. V. 0816 Sebastião, Y. V. 0816 Sebastião, Y. V. 0816 Sebastião, Y. V. 0816 Sebastiao, Y. V. 0816 Segarnviriya, C. 0625 Segust, S. 0159 Seiden, D. 0743, 0745, 0747 Seifritz, E. 0552 Seixas, A. 0013, 0140, 0235, 0406, 0621, 1046, 1046, 1046, 1053, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. 00158, 1062, 1063, 1067, 1071, 1080, 1081 Sereiti, R. 0054, 0		
Scott, B. .0178, 0398 Scott, H. .1192, 1199 Scott, P. W. .056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0604 Scwhartz, D. .0608 Seal, M. .1037, 1045 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0816 Seboek Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Seges, Y. .0128 Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixa, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0054, 0054, 0054, 0054, 0346 Senthilvel, E. .0054, 0054, 0346 Senthilvel, E. .0054, 0054, 0346 Sereika, S. M. .0065, 1067, 1071, 1080, 1081 Sereika, S. M. .0666 Serieka, S. M. .0666 <td></td> <td></td>		
Scott, H. .1192, 1199 Scott, P. W. .0233, 0279, 0279 Scuderi, N. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0604 Scwhartz, D. .0608 Seal, M. .1037, 1045 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0816 Seboek Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Segev, Y. .0128 Segarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0054, 0054, 0054, 0346 Senthilvel, E. .0057 Send, R. .0158 Sendy, R. .0054, 0054, 0346 Senthilvel, E. .0056 Sereika, S. M. .0065, 1067, 1071, 1080, 1081 <t< td=""><td></td><td></td></t<>		
Scott, P. W. 1056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0604 Scwhartz, D. .0608 Seal, M. .1037, 1045 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Seges, Y. .0128 Segarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0054 Send, R. .0513, 0532 Senf, R. .0054, 0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. M. .0686 Sériès, F. .0564 <td< td=""><td>Scott, B</td><td>0178, 0398</td></td<>	Scott, B	0178, 0398
Scott, P. W. 1056 Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0604 Scwhartz, D. .0608 Seal, M. .1037, 1045 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Seges, Y. .0128 Segarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0054 Send, R. .0513, 0532 Senf, R. .0054, 0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. M. .0686 Sériès, F. .0564 <td< td=""><td>Scott, H</td><td>1192, 1199</td></td<>	Scott, H	1192, 1199
Scott-Sutherland, J. .0233, 0279, 0279 Scuderi, N. .0604 Scullin, M. K. .0608 Seal, M. .0369, 0369, 0391 Scwhartz, D. .0608 Seal, M. .037, 1045 Sebastião, Y. V. .0667, 0806 Sebastião, Y. V. .0816 Sebost Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Seges, Y. .0128 Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0247 Semenescu, S. L. .0026 Sen, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. M. .0668 Sériès, F. .0564 Seriek, S. M. .06685 Sériès,		
Scuderi, N.		
Scullin, M. K.		
Scwhartz, D.		
Scwhartz, D.		0369, 0391
Seal, M.		
Sebastião, Y. V. .0667, 0806 Sebastiao, Y. V. .0816 Seboek Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Seges, Y. .0128 Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0026 Sen, A. .00513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Sereika, S. .0065, 1067, 1071, 1080, 1081 Sereika, S. .0065 Sereika, S. M. .0686 Sériès, F. .0564 Sereis, F. .0566 Sériès, F. .0566		
Sebastiao, Y. V. .0816 Seboek Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Segev, Y. .0128 Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1052, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0947 Semenescu, S. L. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .0056 Seriès, F. .0564 Series, F. .0564 Series, F. .0566 Sériès, F. .0566 Seriès, F. .0566 Seriès, F. .0566 Seriès, F. .0566 Seriès, F. .0566		,
Seboek Kinter, D. .0521 Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Segsernviriya, C. .0128 Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0026 Sen, A. .0026 Sen, A. .00513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Sereika, S. .0065, 1067, 1071, 1080, 1081 Sereika, S. .0056 Serieka, S. M. .0686 Sériès, F. .0566 Sériès, F. .0566 Series, F. .0566 Series, F. .0566 Series, F. .0566 Series, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0380 <td></td> <td><i>,</i></td>		<i>,</i>
Sedky, K. .0900 Seewald, M. .0152, 0153, 1109, 1111 Segev, Y. .0128 Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0026 Sen, A. .0513, 0532 Senft, R. .0026 Sen, A. .0513, 0532 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .0686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0566 Sériès, F. .0566 Sériès, F. .0566 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .03859		
Seewald, M. .0152, 0153, 1109, 1111 Segev, Y. .0128 Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. .0232, 0372, 0374, 0864 Sekartini, R. .0026 Sen, A. .00232, 0372, 0374, 0864 Senenescu, S. L. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. .0564 Series, F. .0566 Sériès, F. .0566 Sériès, F. .0566 Series, F. .0380 Servot, S. .0380		
Segev, Y. .0128 Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1058, 1058, 1052, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. .0232, 0372, 0374, 0864 Sekartini, R. .0232, 0372, 0374, 0864 Sekartini, R. .0026 Sen, A. .0513, 0532 Sengupta, A. .00513, 0532 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .0686 Śriès, F. .0564 Series, F. .0566 Sériès, F. .0566 Seriek, S. M. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0380	Sedky, K	0900
Segev, Y. .0128 Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1058, 1058, 1052, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. .0232, 0372, 0374, 0864 Sekartini, R. .0232, 0372, 0374, 0864 Sekartini, R. .0026 Sen, A. .0513, 0532 Sengupta, A. .00513, 0532 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .0686 Śriès, F. .0564 Series, F. .0566 Sériès, F. .0566 Seriek, S. M. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0380	Seewald, M	1109, 1111
Segsarnviriya, C. .0625 Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0232, 0372, 0374, 0864 Sekartini, R. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .0686 Śriès, F. .0564 Series, F. .0566 Sériès, F. .0566 Seriek, S. F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0380		<i>,</i>
Segust, S. .0159 Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0232, 0372, 0374, 0864 Sekartini, R. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .0566 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		
Seiden, D. .0743, 0745, 0747 Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0232, 0372, 0374, 0864 Sekartini, R. .0026 Sen, A. .0513, 0532 Sengupta, A. .0513, 0532 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .0566 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		
Seifritz, E. .0552 Seixas, A. .0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1058, 1052, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0232, 0372, 0374, 0864 Sekartini, R. .0026 Sen, A. .0513, 0532 Senft, R. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .0566 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		
Seixas, A 0013, 0140, 0235, 0406, 0621, 1046, 1046, 1053, 1058, 1058, 1058, 1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A		
1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189, 1212, 1213 Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0947 Semenescu, S. L. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .0686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0566 Series, F. .0566 Series, F. .0566 Series, F. .0564 Series, F. .0566 Series, F. .0566 Series, F. .0566 Series, F. .0566 Series, F. .0380 Service, S. .0380 Servot, S. .0859		
Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0947 Semenescu, S. L. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .0686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0566 Serik, S. .0191 Sereika, S. .0564 Series, F. .0564 Series, F. .0566 Sériès, F. .0566 Series, S. .0380 Service, S. .0380	Seixas, A 0013, 0140, 0235, 0406, 0621, 1046, 1046, 1	1053, 1058,
Seixas, A. A. .0232, 0372, 0374, 0864 Sekartini, R. .0947 Semenescu, S. L. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .0686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0566 Serik, S. .0191 Sereika, S. .0564 Series, F. .0564 Series, F. .0566 Sériès, F. .0566 Series, S. .0380 Service, S. .0380	1058, 1062, 1062, 1093, 1102, 1113, 1114, 1189,	1212, 1213
Sekartini, R. .0947 Semenescu, S. L. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. .0065 Series, F. .0564 Series, F. .0566 Sériès, F. .0566 Seriek, S. .1191 Service, S. .0380 Servot, S. .0859		
Semenescu, S. L. .0026 Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .006686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		
Sen, A. .0513, 0532 Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .0686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		
Senft, R. .1182 Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. .0065, 1067, 1071, 1080, 1081 Sereika, S. .0065 Sereika, S. .0066 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859	,	
Sengupta, A. .0054, 0054, 0346 Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. .00686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0566 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		<i>,</i>
Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .0686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859	Senft, R	1182
Senthilvel, E. .0879 Seo, J. .0065, 1067, 1071, 1080, 1081 Sereika, S. .1056 Sereika, S. M. .0686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859	Sengupta, A	0054, 0346
Seo, J		
Sereika, S. .1056 Sereika, S. M. .0686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		
Sereika, S. M. .0686 Sériès, F. .0564 Series, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		
Sériès, F. .0564 Series, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		
Series, F. .0566 Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		
Sériès, F. .0685 Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859		
Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859	Series, F	0566
Sert Kuniyoshi, F. .1191 Service, S. .0380 Servot, S. .0859	Sériès, F	0685
Service, S		
Servot, S	•	
Sesnauri, S	·	
	Stenauli, S	

Sewall, C	
Sewell, C	
Sexias, A.	
Sforza, M	
Sgambati, F	
Shaarawy, H. m.	
Shafazand, S.	
Shaffer, V. N.	
Shah, K.	
Shah, N.	
Shah, V	
Shahim, P	
Shainin, I	
Shakkottai, A	
Shalowitz, E. L.	
Shaltout, H. A.	
Shamim-Uzzaman, Q.	
Shams-White, M. M.	
Shanholtz, C.	
Shannon, M. A.	
Shapiro, C. M	
Shapiro, T	1063
Sharafkhaneh, A	
Shariff, T	
Sharkey, K. M	
Sharma, A	
Sharma, M	
Sharma, P	
Sharman, R	
Sharp, C.	
Shattuck, N. L	
Shaughnessy, G. F	
Shaw, P	
Shaw, I	
Shea, J. A.	
Shea, S. A	
Shea, T	
Shechter, A	.0172, 0253, 1154
Shedden, K	
Sheedy, C	
Sheehan, D	
Sheehan, O. C	
Sheinkopf, S. J.	
Shekari Soleimanloo, S	
Sheldon, S. S.	
Sheline, Y	
Shen, L	
Shenker, J.	
Sher, S.	
Sherman, S. G	
Shi, C	
Shih, C	
Shiferaw, B.	
Shiloh, A.	
Shimomura, K	
Shin, C	
Shin, H. J	
Shin, H	
Shin, J	
SHIN, W	

Shinde, A	
Shine, L	
Shinke, T.	
Shioya, T	
Shirahama, R	
Shochat, T	0863, 0864
Shokoueinejad, M	0636, 0636
Shokri-Kojori, E	
Shoval, H. A.	
Shukla, G	
Shumard, T	
Siddiqui, F	
Siegel, E. M	
Siegel, J.	
Siegel, M.	
Siengsukon, C.	
Sierra-Gonzalez, A.	
Signal, L.	
Signal, T. L.	
-	
Silber, J. H	
Silber, M	
Silber, M. H	
Sillah, A	
Silva, A.	
Silva, B. M.	
Silva, G. E.	
Silva, L. O	
Silva, M	
Silva, M. A.	
Silvestre, P	
Simakajornboon, N	
Simmons, B	
Simmons, J. H	
Simmons, R	
Simmons, R. O	
Simmons, Z	
Simon, K. C	
Simon, K	
Simon, K	
Simon, S. L	
Simon, S. L	
Simon, S. L	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	0115 $0110, 0110$ $I, 0924, 0982, 1029$ $0385, 0385, 1144$ $0135, 0139, 0353$ $1120, 1130$ $0636, 0636$ $0499, 0534$ $0279, 0279$ 0842 0178 1146 0787 1025 $0794, 0794$ 0731 0007
Simon, S. L. .0876, 0886, 0921, 092 Simonslik, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonslik, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonslik, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonslik, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonelli, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonslik, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonslik, G.	
Simon, S. L. .0876, 0886, 0921, 092 Simonslik, G.	

Skoulos, MC	
Skowronski, R	
Skwara, A. K	
Slavish, D	
Slavish, D. C	225, 0384, 1063
Sletten, T. L	
Sliwinski, M. J	
Sloley, S. S	
Slota, K. A	
Smagula, S	
Smagula, S. F	
Small, B. J	
Smallwood, S	
Smarr, B.	
Smernoff, Z	
Smieszek, S. P.	
Smith, A. M	
Smith, A. A	
Smith, A.	
Smith, J. P.	
Smith, J. R.	
Smith, J.	
Smith, K. J.	
Smith, M	
Smith, M. G	
Smith, R. L	
Smith, S. S	
Smith, S. S	
Snider, M. N	
Snyder, E	
Snyder, E	
Sobremonte-King, M.	
Sochal, M.	
Soehner, A. M	
Soffia, H. M.	
Soh, L	
Sohn, MW.	
Solar, X.	
Solomonova, E	
Soltani, S	
Somers, V. K	
Somerville, G	328 0018 0050
Somma, A	
Song, Y	
Sonia, AI	
Sorensen, H	
Sørensen, H. B	
Soriano, S	
Soriano-Smith, R	
Sorrell, A	
Sosa, J	
Sosnowski, D. W	
Sotelo, M. I	
Soto, P	
D4/3 D0/ (160/ 0)	600 1007 1144
Soubrier, M	

Sowden, W. J. .0188, 0189, 0189, 0199 Spadola, C. .0624, 1044, 1044, 1168 Spadola, C. E. .1166 Spadola, C. E. .0187, 0401, 0851 Sparks, J. R. .0127, 0401, 0851 Sparks, K. .0106 Spece, L. J. .0596 Speed, K. J. .0810, 0945, 1087, 1104, 1104 Spencer, R. .0122, 0222 Spencer, R. M. .0087, 0089, 0089, 0100, 0207, 0334
Spadola, C. E. .1166 Spaeth, A. M. .0867 Sparks, J. R. .0127, 0401, 0851 Sparks, K. .0106 Spece, L. J. .0596 Speed, K. J. .0810, 0945, 1087, 1104, 1104 Spencer, R. .0122, 0222
Spaeth, A. M. .0867 Sparks, J. R. .0127, 0401, 0851 Sparks, K. .0106 Spece, L. J. .0596 Speed, K. J. .0810, 0945, 1087, 1104, 1104 Spencer, R. .0122, 0222
Sparks, J. R. .0127, 0401, 0851 Sparks, K. .0106 Spece, L. J. .0596 Speed, K. J. .0810, 0945, 1087, 1104, 1104 Spencer, R. .0122, 0222
Sparks, J. R. .0127, 0401, 0851 Sparks, K. .0106 Spece, L. J. .0596 Speed, K. J. .0810, 0945, 1087, 1104, 1104 Spencer, R. .0122, 0222
Sparks, K. .0106 Spece, L. J. .0596 Speed, K. J. .0810, 0945, 1087, 1104, 1104 Spencer, R. .0122, 0222
Spece, L. J. .0596 Speed, K. J. .0810, 0945, 1087, 1104, 1104 Spencer, R. .0122, 0222
Speed, K. J. .0810, 0945, 1087, 1104, 1104 Spencer, R. .0122, 0222
Spencer, R
Spencer, R
Spencer, R. M
Spencer, R. W
• • • • • • • • • • •
Spielberg, D
Spira, A. P0135, 0139, 0353, 0843, 0843, 0844, 0872, 0988, 1137
Spray, B. J
Sprecher, K. E
Squires, L
Srikanchana, R
Srinivasan, S
Srisawart, P
Srivastava, D
Stanley, M
Stanley, N
Stanley, N
•
Stansbury, R
Staud, R
Stearns, M
Steele, T
Stefani, A
Stefansdottir, R
Stefansdottir, R. S
Stefanski, V
Steffen, A
Steffen, A
Stehli, A
Stein, M
Steinberg, K
Steinhart, E
Stensel, D. J
Stepan, M. E
C4
Stephan, J. T
Stephan, J. 1
Stepnowski, C0568, 0568
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0113, 0278, 0278, 0311, 0311
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0113, 0278, 0278, 0311, 0311
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609 Stickgold, R. .0113, 0278, 0278, 0311, 0311 Stitt, C. J. .0357
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0113, 0278, 0278, 0311, 0311 Stitt, C. J. .0357 Stiver, J. .1115 St Laurent, C. W. .0222
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0113, 0278, 0278, 0311, 0311 Stitt, C. J. .0357 Stiver, J. .1115 St Laurent, C. W. .0222 St Louis, E. K. .0277, 0277
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609 Stickgold, R. .0113, 0278, 0278, 0311, 0311 Stitt, C. J. .0357 Stiver, J. .1115 St Laurent, C. W. .0222 St Louis, E. K. .0277, 0277 St. Louis, E. .0787, 0817
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609 Stickgold, R. .0113, 0278, 0278, 0311, 0311 Stitt, C. J. .0357 Stiver, J. .1115 St Laurent, C. W. .0222 St Louis, E. K. .0277, 0277 St. Louis, E. K. .0787, 0817 St. Louis, E. K. .0003, 0355, 0695
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609 Stickgold, R. .0113, 0278, 0278, 0311, 0311 Stitver, J. .1115 St Laurent, C. W. .0222 St Louis, E. K. .0277, 0277 St. Louis, E. K. .0787, 0817 St. Louis, E. K. .0003, 0355, 0695 Stocks, A. .0112
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609 Stickgold, R. .0113, 0278, 0278, 0311, 0311 Stitt, C. J. .0357 Stiver, J. .1115 St Laurent, C. W. .0222 St Louis, E. K. .0277, 0277 St. Louis, E. K. .0787, 0817 St. Louis, E. K. .0003, 0355, 0695 Stocks, A. .0112 Stone, J. E. .0289, 0433
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609 Stickgold, R. .0113, 0278, 0278, 0311, 0311 Stitver, J. .1115 St Laurent, C. W. .0222 St Louis, E. K. .0277, 0277 St. Louis, E. K. .0787, 0817 St. Louis, E. K. .0003, 0355, 0695 Stocks, A. .0112
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609 Stickgold, R. .0113, 0278, 0278, 0311, 0311 Stitt, C. J. .0357 Stiver, J. .1115 St Laurent, C. W. .0222 St Louis, E. K. .0277, 0277 St. Louis, E. K. .0787, 0817 St. Louis, E. K. .0003, 0355, 0695 Stocks, A. .0112 Stone, J. E. .0289, 0433 Stone, K. .0388, 1145
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609 Stickgold, R. .0113, 0278, 0278, 0311, 0311 Stitt, C. J. .0357 Stiver, J. .1115 St Laurent, C. W. .0222 St Louis, E. K. .0277, 0277 St. Louis, E. K. .0003, 0355, 0695 Stocks, A. .0112 Stone, J. E. .0289, 0433 Stone, K. L. .0048, 0852, 0855
Stepnowski, C. .0568, 0568 Stepnowsky, C. .0696, 1017 Stepnowsky, C. J. .0683 Sterkel, A. L. .0781, 0781 Sternberg, A. L. .0196, 0261 Sterner, T. .1182 Sterni, L. .0944 Stevens, J. .0487, 0488 Stewart, E. .0234, 0248 Stewart, N. H. .0596 Stickel, A. .0607, 0607, 0609 Stickgold, R. .0113, 0278, 0278, 0311, 0311 Stitt, C. J. .0357 Stiver, J. .1115 St Laurent, C. W. .0222 St Louis, E. K. .0277, 0277 St. Louis, E. K. .0787, 0817 St. Louis, E. K. .0003, 0355, 0695 Stocks, A. .0112 Stone, J. E. .0289, 0433 Stone, K. .0388, 1145

Stothard, E. R
St. Pierre, M
Straud, C. L
Straus, L
Straus, L. D
strauss, S
Strayer, S. M
Strayer, S. M
Stretch, R
Strohl, K
Strohl, K. P
Strohl, K
Strollo, P
Strollo, P. J
Stubbers, K. M
Stucynski, J
Stultz, D. J
Stutaite, G
Suarez, M
Subramanian, R
Sucevic, J
Sudhakar, R
Sudnawa, K. K
Suh, I
Suh, S
Suh, S
Suhail, M
Suhan, L
Sullivan, J
Sullivan, K. J
Sullivan, L
Sullivan, S
Sumi, Y
Sumiya, M
Summers, C
Sun, H
Sun, T
Sun, Y
Sunaga, H
Sundar, K
Sundar, K. M
Sunwoo, JS
Sutherland, R
Suthers, B
Suthoff, E
Suzanne, D. S
Suzuki, M
Svatikova, A
Svetnik, V
Swanson, G0780
Swanson, L
Swanson, L. M
Swick, T. J
Sydejko, D. M
Syed, Z
Szakacs, Z
Szelestey, B
Szuperak, M

Т

Та, D	682
Tablizo, M0	902

Tabuteau, H
Tachibana, N
Tackett, J. V
Tahara, Y
Takahashi, N
Takai, N
Takamatsu, S
Talaat, K
Talavera, G. A
Talbot, C
Talker, I
Tallavajhula, S
Tan, P
Tan, X
Tanaka, T
Taneja, D
Tang, L
Tang, X
Tanigawa, T
Tanioka, K
Tao, Y
Tapia, I
1
Tapia, I. E
Tarasiuk, A
Taravath, S
Tarraf, W
Tashman, Y. S
Tate, L. L
Tauman, R
Taveras, E. M
Taweesedt, P
1ay, J
Tay, J
Taylor, A
Taylor, A. .0886 Taylor, B. .0388
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083
Taylor, A.
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, M. .0159
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0802
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, P. .0024
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, P. .0024 Taylor, S. C. .0974
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, P. .0024
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, P. .0024 Taylor, S. C. .0974
Taylor, A. .0886 Taylor, B. .0398 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327
Taylor, A. .0886 Taylor, B. .0398 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1151 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. H. .0523, 0541, 0542 Teigen, L. .0817
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Teigen, L. .0817 Teixeira, C. .0529
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. H. .0523, 0541, 0542 Teigen, L. .0817
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Teigen, L. .0817 Teixeira, C. .0529
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0882 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. H. .0817 Teixeira, C. .0529 Tempaku, P. F. .0022, 0577, 1126 ten Brink, M. .0256
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Teigen, L. .0817 Teixeira, C. .0529 Tempaku, P. F. .0022, 0577, 1126 ten Brink, M. .0256 Teng, J. .0143, 0519, 0519, 0824, 0824
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Teigen, L. .0817 Teixeira, C. .0529 Tempaku, P. F. .0022, 0577, 1126 ten Brink, M. .0256 Teng, J. .0143, 0519, 0519, 0824, 0824 Terui, Y. .0518
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. H. .0817 Teixeira, C. .0529 Tempaku, P. F. .0022, 0577, 1126 ten Brink, M. .0256 Teng, J. .0143, 0519, 0519, 0824, 0824 Terui, Y. .0518 Testa, K. J. .0028
Taylor, A. .0886 Taylor, B. .0398, 1063, 1073, 1186 Taylor, D. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, D. J. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, J. L. .1151 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Teigen, L. .0817 Teixeira, C. .0529 Tempaku, P. F. .0022, 0577, 1126 ten Brink, M. .0256 Teng, J. .0143, 0519, 0519, 0824, 0824 Terui, Y. .0518 Testa, K. J. .0128 Thacker, J. .0891
Taylor, A. .0886 Taylor, B. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. H. .0817 Teixeira, C. .0529 Tempaku, P. F. .0024, 0824 Terui, Y. .0143, 0519, 0519, 0824, 0824 Terui, Y. .0518 Testa, K. J. .0128 Thacker, J. .0891, 0891 Thackray, A. E. .0539
Taylor, A. .0886 Taylor, B. .0398 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, D. J. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, K. A. .0667, 0806 Taylor, N. .0882 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Teigen, L. .0523, 0541, 0542 Teigen, L. .0024, 0529 Tempaku, P. F. .0024, 0529 Tempaku, P. F. .0024, 0529 Tempaku, P. F. .00257 Teixeira, C. .0529 Tempaku, P. F. .0022, 0577, 1126 ten Brink, M. .0256 Teng, J. .0143, 0519, 0519, 0824, 0824 Text, K. J. .028 Thacker, J. .0891, 0891 Thacker, J. .03940
Taylor, A. .0886 Taylor, B. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. H. .0817 Teixeira, C. .0529 Tempaku, P. F. .0024, 0824 Terui, Y. .0143, 0519, 0519, 0824, 0824 Terui, Y. .0518 Testa, K. J. .0128 Thacker, J. .0891, 0891 Thackray, A. E. .0539
Taylor, A. .0886 Taylor, B. .0398 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, D. J. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, K. A. .0667, 0806 Taylor, N. .0882 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Teigen, L. .0523, 0541, 0542 Teigen, L. .0024, 0529 Tempaku, P. F. .0024, 0529 Tempaku, P. F. .0024, 0529 Tempaku, P. F. .00257 Teixeira, C. .0529 Tempaku, P. F. .0022, 0577, 1126 ten Brink, M. .0256 Teng, J. .0143, 0519, 0519, 0824, 0824 Text, K. J. .028 Thacker, J. .0891, 0891 Thacker, J. .03940
Taylor, A. .0886 Taylor, B. .0398 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1137 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0802 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. H. .0523, 0541, 0542 Teigen, L. .0022, 0577, 1126 ten Brink, M. .0256 Teng, J. .0143, 0519, 0519, 0824, 0824 Terui, Y. .0518 Testa, K. J. .028 Thacker, J. .028 Thacker, J. .0539 Thai, C. L. .0539 Thai, C. L. .0619, 0731, 0802 Thakkar, M. .0619, 0731, 0802
Taylor, A. .0886 Taylor, B. .0398 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1151 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0024 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. H. .0523, 0541, 0542 Teigen, L. .0523, 0541, 0542 Teigen, L. .0022, 0577, 1126 ten Brink, M. .0256 Teng, J. .0143, 0519, 0519, 0824, 0824 Terui, Y. .0518 Testa, K. J. .1028 Thacker, J. .028 Thacker, J. .0539 Thai, C. L. .0539 Thai, C. L. .0539 Thakar, M. .0619, 0731, 0802 Thakkar, M. .0619, 0731, 0802 <t< td=""></t<>
Taylor, A. .0886 Taylor, B. .0388 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. H. .0523, 0541, 0542 Tegeler, C. H. .0523, 0541, 0542 Tegeler, C. H. .0529 Tempaku, P. F. .0022, 0577, 1126 ten Brink, M. .0256 Teng, J. .0143, 0519, 0519, 0824, 0824 Terui, Y. .0518 Testa, K. J. .1028 Thacker, J. .0539 Thai, C. L. .0539 Thai, C. L. .0539 Thakar, M. .0619, 0731, 0802 Thakar, M. .0619, 0731, 0802
Taylor, A. .0886 Taylor, B. .0398 Taylor, D. .0398, 1063, 1073, 1186 Taylor, D. J. .0178, 0225, 0384, 0399, 0483, 0483, 1110 Taylor, E. .0079, 0263, 0314, 0315, 0430, 1076, 1077, 1083 Taylor, H. L. .1151 Taylor, J. L. .1151 Taylor, K. A. .0667, 0806 Taylor, N. .0159 Taylor, N. .0024 Taylor, S. C. .0974 Tazawa, R. .0205 Tchintcharauli, T. .0327 Tegeler, C. L. .0523, 0541, 0542 Tegeler, C. H. .0523, 0541, 0542 Teigen, L. .0523, 0541, 0542 Teigen, L. .0022, 0577, 1126 ten Brink, M. .0256 Teng, J. .0143, 0519, 0519, 0824, 0824 Terui, Y. .0518 Testa, K. J. .1028 Thacker, J. .028 Thacker, J. .0539 Thai, C. L. .0539 Thai, C. L. .0539 Thakar, M. .0619, 0731, 0802 Thakkar, M. .0619, 0731, 0802 <t< td=""></t<>

Thapa, S	
Thase, M	
Thatipelli, S	
Thayer, J. F	
Theorell-Haglöw, J.	
Théoret, R	
Theoret, R.	
Théoret, R.	
Thind, H	
Thoma, B. C	
Thomas, R. C.	
Thomas, R0348, 0	0592, 0661
Thomas, R. J	
Thompson, H	
Thompson, N	
Thompson, W.	
Thomson, C. A	
THORET, E	
Thorey, V	
Thorndike, F	
Thorndike, F. P.	
Thornton, T.	
Thorpy, M.	
Thorpy, M. J	
Thosar, S. S.	
Thundercliffe, J. A	
Thurston, R. C	
Thybo, J	
Tiemeyer, K	
Timm, P	
Timofeev, I	
Tin, H	
Tina, V	
Titone, M. K	
Tkacs, N. C	
Tobin, C	
Tobin, M	
Togeiro, S	
Toh, S	
Tojino, A. G	
Tokumaru, Y	
Tomasi, D	0166
Tomooka, K	
Tonascia, J	
Tong, C. H	
Tonnu, C. V	0470, 0470
Tononi, G	
Torabzadeh, E	0571, 0571
Tordoff, S. A.	0749
Toriola, A. T	
Torre, B. C	0589
Toth, S	0878
Touchette, E	0859
Townsend, R	0053
Töyräs, J	0593, 0593
Tracy, O. J	
Trainer, M. M	0030
Tran, K. M	
Tran, LK	0640
Tran, L	0829
Tran, ML	0195
	11110120
Trejo, J. I	

Trichet, T
Triller, A
Trivedi, R
Trotter, D
Trotti, L
Troyanskaya, M
Truzzi, G. M
TSAI, CW
Tsai, H
Tsai, HF
Tsai, SY
Tsai, S
Tsai, SM
Tsao, CY
Tschopp, K
Tschopp, S
Tselha, T
Tsimpanouli, ME
Tsuiki, S
Tsutsui, K
Tu, A
Tu, A. Y
Tu, CH
Tuan, LH
Tubbs, A
Tucker, A. J
Tucker, A. J. .0286 Tucker, M. .0104
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635,
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635,
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022 Tulk, J. .1036, 1037
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022 Tulk, J. .1036, 1037 Tumminia, M. .0258
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010 Turkiewicz, S. .0170
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010 Turkiewicz, S. .0170 Turkington, F. .0636, 0636
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010 Turkiewicz, S. .0170 Turkington, F. .0636, 0636 Turner, A. .0864
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010 Turkiewicz, S. .0170 Turkington, F. .0636, 0636 Turner, A. .0864 Turner, R. .0226, 0240
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010 Turkiewicz, S. .0170 Turkington, F. .0636, 0636 Turner, A. .0864 Turner, A. D. .0011, 0107, 0655, 0863, 1150, 1153
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010 Turkiewicz, S. .0170 Turkington, F. .0636, 0636 Turner, A. .0864 Turner, R. .0226, 0240 Turner, R. W. .0226 Tutek, J. .0236
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .1022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010 Turkington, F. .0636, 0636 Turner, A. .0864 Turner, R. .0226, 0240 Turner, R. W. .0226, 0240 Turner, R. W. .0236 Tutek, J. .0551, 0551 Tyan, J. L. .0211
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .0022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010 Turkiewicz, S. .0170 Turkington, F. .0636, 0636 Turner, A. .0864 Turner, R. W. .0226, 0240 Turner, R. W. .0226 Tutek, J. .0226, 0240 Turner, R. W. .0236 Tutek, J. .0211 Tyler, C. .0747
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .0022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010 Turkiewicz, S. .0170 Turkington, F. .0636, 0636 Turner, A. .0864 Turner, R. .0226, 0240 Turner, R. W. .0226 Tutek, J. .0551, 0551 Tyan, J. L. .0211 Tyler, C. .0747 Tyson, T. L. .0310
Tucker, A. J. .0286 Tucker, M. .0104 Tudor, J. C. .0028, 0028, 0346 Tufik, S. .0002, 0022, 0270, 0272, 0493, 0498, 0563, 0577, 0635, 0639, 0663, 0828, 0830, 0831, 0832, 0881, 1079, 1126 Tufik, S. V. .0022 Tulk, J. .1036, 1037 Tumminia, M. .0258 Tuovinen, N. .0010 Turkiewicz, S. .0170 Turkington, F. .0636, 0636 Turner, A. .0864 Turner, R. W. .0226, 0240 Turner, R. W. .0226 Tutek, J. .0226, 0240 Turner, R. W. .0236 Tutek, J. .0211 Tyler, C. .0747

U

Uchimura, N
Udler, M
Uehira, M
Uemura, S. I
Uhde, T. W
ULMER, C. S
Ulrich, A. B
Ulrich, C. M
Ulsa, C
Umasabor-Bubu, O. Q
Upender, R
Urbanek, J
Urbano, G

Urquhart, G. J	2
Ursache, A	5
Uygun, D. S	8

V

Vakulin, A
Valencia, J. A
Valentine, K
Vallabhaneni, V
Valli, B
Vallieres, A
Vallim, J. R
Valomon, A
Vana, K. D
Van Beers, P
Vance, A
van den Berg, M. J
Van Der Maren, S
van der Merwe, A
Vander Stoep, A
Vandi, S
Van Dongen, H 0025, 0043, 0121, 0125, 0274, 0297, 0300, 0301,
0308, 0332, 0730, 1038
Vanecek, R
van Egmond, L. T
van Lamsweerde, A. E
van Rijn, E
Vanuk, J. R0070, 0079, 0305, 0305, 0307, 0314, 1158, 1158, 1160
Van Wagoner, D
Varga, A
Varga, A. W
Vargas, I
Vargas, I
Vasquez, M
Vasquez, M
Vassai, S
Vaughan, E. C
Vaughn, B
Vaughn, B. V
Vaz Fragoso, C. A
Veatch, O. J
Veeravigrom, M
Vega Sanchez, M. E
Vega Sanchez, M
VegaSanchez, M
Velamuri, K
Veler, H
Velpari, S
Vemuri, P
Venner, A
Verceles, A
Vergez, A
Verkler, J
Verlaque, R
Vermeeren, A
Veronda, A. C
Vervloet, T
Vesnaver, D
Vetter, C
Vetter, M
Vgontzas, A0506, 1035, 1120, 1130

Vgontzas, A. N 0319, 0457, 0458, 0585, 0878, 0890, 0919, 0920,
0936, 1107
Vidigal, T. A
Vigoureux, T. F
Vijayan, S
Vila, B
Villalba, D
Villalobos, A. P
Villalobos, R
Villalobos Jr., R
Villarreal, B
Villarreal, I. A
Villaseñor, T
Vinces, K
Vinci, C. E
Vinckenbosch, F
Violanti, J
Virudachalam, S
Visintainer, P
Vispute, S
Vital-Lopez, F
Viteri, E
Vitiello, M. V
Vo, T
Volkow, N. D
Volpe, L
Volpp, K. G
Vondran, R
Vonk, P
Von Korff, M0469
Voss, M. W
Vujcic, B
Vujovic, N

W

Wada, H
Waddle, A. E
Wadhawan, A
Waeber, A
Wager, M. E
Wagg, A. S
Wahl, A. M
Wahlstrom, K. L
Wakefield, C
Wakschlag, L
Wali, S. O
Walia, H
Walia, H. K
Walker, B. L
Walker, J
Walker, N. A
Walker, S. A
Wallace, D
Wallace, D. M
Wallace, J
Wallace, M
Wallace, M. L
Walmboldt, F
Walsh, J. K
Walsh, K
Walsh, N
Walters, R

Walton, R
Wamsley, E. J
Wanaski, S. P
Wang, C
Wang, C
Wang, D
Wang, GJ
Wang, G
Wang, G
Wang, J
Wang, L
Wang, M
Wang, N
Wang, R
Wang, S. Y
Wang, S. S
Wang, TC
Wang, TY
Wang, W
Wang, W
Wang, X
Wang, X
Wang, Y
Wang, Y
Wang, Y
Wang, Y
Wang, Z
Wani, B
Wani, I
Wanigatunga, A. A
NU ' / O 010
Wanigatunga, S
Wanigatunga, S. K
Wanigatunga, S. K
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .070 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0694, 0705, 088
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0694, 0705, 088 Watanabe, N. .0633, 063
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0633, 063 Watanabe, N. .0633, 063 Watanabe, Y. .059
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0694, 0705, 088 Watanabe, N. .0633, 063
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0633, 063 Watanabe, N. .0633, 063 Watanabe, Y. .059 Waters, T. .0668 Watkins, S. L. .022
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0633, 063 Watanabe, N. .059 Waters, T. .068
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0694, 0705, 088 Watanabe, N. .0633, 063 Waters, T. .068 Watkins, S. L. .022 Watson, H. .085 Watson, K. T. .004
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0633, 063 Watanabe, N. .0633, 063 Watanabe, Y. .059 Waters, T. .068 Watkins, S. L. .022 Watson, H. .085
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0694, 0705, 088 Watanabe, N. .0633, 063 Waters, T. .068 Watkins, S. L. .022 Watson, H. .085 Watson, K. T. .004
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0694, 0705, 088 Watanabe, N. .0633, 063 Waters, T. .068 Watkins, S. L. .022 Watson, H. .085 Watson, N. .100
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0694, 0705, 088 Watanabe, N. .0633, 063 Watsers, T. .068 Watkins, S. L. .022 Watson, H. .085 Watson, N. .100 Waxman, J. .063
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0694, 0705, 088 Watanabe, N. .0633, 063 Watkins, S. L. .022 Watson, H. .085 Watson, K. T. .004 Watson, N. .100 Waxman, J. .063 Weathers, J. .111
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0694, 0705, 088 Watanabe, N. .0633, 063 Waters, T. .068 Watkins, S. L. .022 Watson, H. .085 Watson, N. .100 Waxman, J. .063 Weathers, J. .111 Weaver, M. .118
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0633, 063 Watanabe, N. .0633, 063 Waters, T. .068 Watkins, S. L. .022 Watson, H. .085 Watson, K. T. .004 Watson, N. .100 Waxman, J. .063 Weathers, J. .111 Weaver, M. .118 Weaver, M. D. .025
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .070 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0633, 063 Watanabe, N. .0633, 063 Waters, T. .068 Watkins, S. L. .022 Watson, H. .085 Watson, N. .100 Waxman, J. .063 Weathers, J. .111 Weaver, M. .118 Weaver, M. .025 Weaver, M. .0412, 041 Weaver-Rogers, S. .092
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .107 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0633, 063 Watanabe, N. .0633, 063 Watanabe, Y. .059 Waters, T. .068 Watkins, S. L. .022 Watson, H. .085 Watson, N. .100 Waxman, J. .063 Weathers, J. .111 Weaver, M. .118 Weaver, M. .0412, 041 Weaver, S. .0567, 0610, 104
Wanigatunga, S. K. .035 Ward, J. B. .1016, 101 Ward, S. V. .061 Ward, T. .091 Wardle, S. .070 Wardle-Pinkston, S. .0225, 0384, 0398, 1063, 118 Wasey, W. .064 Washington, D. L. .0467, 058 Wasilczuk, A. Z. .016 Wasserzug, O. .089 Watach, A. J. .0633, 063 Watanabe, N. .0633, 063 Waters, T. .068 Watkins, S. L. .022 Watson, H. .085 Watson, N. .100 Waxman, J. .063 Weathers, J. .111 Weaver, M. .118 Weaver, M. .025 Weaver, M. .0412, 041 Weaver-Rogers, S. .092

Wehling, R. R	
Wei, SY	
Wei, W	
Wei, X	
Wei, Y	
Wei, Z	
Weihs, K. L	
Weiler, U	
Weiner, H. L	28
Weingarten, J	12
Weingarten, J. A	
Weintraub, D	
Wei-Shu, L	
Weiss, C	51
Weiss, J. P	12
Weitzberg, E	21
Weizman, L	84
Weljie, A	
Wellman, A	93
Wellman, D. A	48
Wells, C. C	74
Wells, C	47
Wen, Y	16
Wendel, C	17
Werden, E	20
Werneburg, B	35
Werner, K	16
Wernette, E. M	84
Wertz, A. T	
Wescott, D. L	
Wesley, K. L	
Westover, M	
West Saxvig, I	
Wharton, L	
Wharton, R	
Wheatley, J. R	
Wheaton, A. G	32
Wheelis, M	
Whibley, D	
White, D. P	
White, K	
White, M	
White, M	
Whitehurst, L	19
Whitehurst, L. N	
Whitelaw, A	
Whitesell, P	
Whiteside, G. T	
Whitford, J	
Whiting, C	
Whitney, P	
Whittaker, D	
Whooley, M	
Whyte, J	
Wicker, E. W	
Wickwire, E	
Wickwire, E. M	
Wiedmer, A	
Wiemels, J	
Wiemken, A	
Wiemken, A. S	
Wierenga, C. E	
Wiers, C. E	

Wiley, A	
Wiley, J. F	
Wilkerson, A	
Wilkerson, A. K	
Willes, L	2
William, W1155, 115	
Williams, A. J	1
Williams, C. E	9
Williams, D. A	1
Williams, J. S	2
Williams, M. K	8
Williams, M	
Williams, N. J	7
Williams, N	
Williams, N. J.0152, 0153, 0232, 0372, 0374, 0863, 0864, 1150, 115	3
Williams, N	3
Williams, N	
Williams, S	8
Williamson, A. A	
0390, 0397, 0875, 0875, 0885, 0885, 0921, 0921, 0924	4,
0929, 0946, 095	2
Williamson, D	
Willis, D	
Wills, C. C	
0013, 0120, 0140, 0226, 0232, 0235, 0236, 0240, 0241, 0243, 0372	
0374, 0375, 0376, 0406, 0544, 0553, 0865, 1053, 1093, 1095, 110	
Wilson, M	
Winful, O. T	
Winiger, E	
Winkelman, J. W	
Winter, W	
Winters, A	
Wise, J. C	
Wise, M	
Wisor, J. P	
Withrow, D	
Witman, M. A	
Wittine, L	
Wohlgemuth, W	1
Wohlgemuth, W. K	
Wolfson, A	
Wolfson, A. R	
Wolk, D	
Wolkow, A. P	
Wong, B	0
WONG, E	2
Wong, H	2 4
Wong, H	2 4 9
Wong, H	2 4 9 9
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. K. .066 Wong, K. F. .0519, 051	2 9 9
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. K. .066 Wong, K. F. .0519, 051 Wong, L. R. .0078, 017	2 4 9 9 9 5
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. K. .066 Wong, K. F. .0519, 051 Wong, L. R. .0078, 017 Wong, P. M. .0250, 025	2 4 9 9 9 5 7
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. K. .066 Wong, K. F. .0519, 051 Wong, L. R. .0078, 017 Wong, P. M. .0250, 025 Wong, S. .033	2 4 9 9 5 7 3
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. K. .066 Wong, K. F. .0519, 051 Wong, L. R. .0078, 017 Wong, P. M. .0250, 025 Wong, S. .033 Woo, S. .097	2 4 9 9 5 7 3
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. K. .066 Wong, K. F. .0519, 051 Wong, L. R. .0078, 017 Wong, P. M. .0250, 025 Wong, S. .033 Woo, S. .097 Woodruff, J. .0636, 063	2 4 9 9 9 5 7 3 1 6
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. K. .066 Wong, K. F. .0519, 051 Wong, L. R. .0078, 017 Wong, P. M. .0250, 025 Wong, S. .033 Woo, S. .097 Woodruff, J. .0636, 063 Woods, A. D. .037	2 4 9 9 5 7 3 1 6 1
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. .066 Wong, K. F. .0519, 051 Wong, L. R. .0078, 017 Wong, P. M. .0250, 025 Wong, S. .033 Woo, S. .097 Woodruff, J. .0636, 063 Woods, A. D. .037 Woods, C. .000	2 4 9 9 9 5 7 3 1 6 1 5
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. .066 Wong, K. F. .0519, 051 Wong, L. R. .0078, 017 Wong, S. .0250, 025 Wong, S. .033 Woo, S. .097 Woodruff, J. .0636, 063 Woods, A. D. .037 Woodward, S. .107	2 4 9 9 9 5 7 3 1 6 1 5 4
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. .066 Wong, K. F. .0519, 051 Wong, L. R. .0078, 017 Wong, S. .0250, 025 Wong, S. .033 Woo, S. .097 Woodruff, J. .0636, 063 Woods, A. D. .037 Woodward, S. .107 Woodward, S. H. .106	2 4 9 9 5 7 3 1 6 1 5 4 8
Wong, H. .057 Wong, J. .0809, 1138, 114 Wong, K. .066 Wong, K. F. .0519, 051 Wong, L. R. .0078, 017 Wong, S. .0250, 025 Wong, S. .033 Woo, S. .097 Woodruff, J. .0636, 063 Woods, A. D. .037 Woodward, S. .107	2 4 9 9 5 7 3 1 6 1 5 4 8 1

Wright, A	
Wright, K.	
Wright, K. P.	
Wright Jr., K. P	
Wu, B	
Wu, C	
Wu, H	
Wu, H	
Wu, J	
Wu, JL	
Wu, L. J	
Wu, M. N	
Wu, S	
Wu, S	
Wu, T	
Wu, Y	
Wyatt, J. K	
Wyland, C.	

X

Xanthopoulos, M	0888
Xanthopoulos, M. S.	
Xavier, S. D.	
Xi, M	
Xia, X	
Xia, Y	
Xiao, C	,
Xiao, F	
Xiao, Q	
Xiao, R	
Xiaojun, Z	
Xie, J	
Xie, J	
Xie, J	
XU, H	0559, 0565, 0601
Xu, J	
Xu, L	
Xu, L	,
Xu, Y	
Xu, Y	
Xu, Y	
Xu, Z	
Xue, J	
Xue, P	

Y

Yabes, J. G
Yadav, S
Yaffe, K0048, 0652, 1145, 1152
Yaggi, H
Yaggi, H. K
Yagi, T
Yalamanchi, K
Yamada, K1215
Yamada, S
Yamamoto, Y
Yamazaki, E. M
Yang, A
Yang, C

	60
Yang, F. N	
Yang, H	79
Yang, HW	32
Yang, K	35
Yang, N	
Yankowy, L	
Yano, E	
Yano, E. M	
Yao, DJ	
YAR, W	
Yarbrough, W. C	
Yardley, J	
Yassa, M. A	
Yawn, B	96
Yee, A. H	47
Yee, B	69
Yee, B. J	
YEH, E	
Yeh, M	
Yeh, PH	
Yesavage, J. A	132
YI, H	
Yin, G 0679, 06	
Yin, LY	
YIN, S	13
Yoon, IY	45
Yoshida, S	46
Yoshizawa, K	
You, D	
Younes, M	
Young-McCaughan, S	
Youngren, W	
Youngren, W. A	01
	CE
Youngstedt, S	64
Youngstedt, S	64 51
Youngstedt, S	64 51 37
Youngstedt, S	64 51 37
Youngstedt, S	64 51 37 20
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10	64 51 37 20 91
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11	64 51 37 20 91 35
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06	64 51 20 91 35 95
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, X. .03	64 51 20 91 35 95 18
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, X. .03 Yu, Y. A. .08	64 51 20 91 35 95 18 97
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, X. .03 Yu, Y. A. .0684, 06	64 51 37 20 91 35 95 18 97 54
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .066 Yu, L. .1135, 11 Yu, TY. .066 Yu, X. .03 Yu, Y. A. .0654, 06 Yuan, R. K. .0033, 0117, 0302, 03	64 51 20 91 35 95 18 97 54 02
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .066 Yu, L. .1135, 11 Yu, TY. .06 Yu, Y. .033 Yu, Y. .0654, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .07	64 51 37 20 91 35 95 18 97 54 02 97
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, Y. .0654, 06 Yuu, Y. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06	64 51 37 20 91 35 95 18 97 54 02 97 60
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, Y. A. .03 Yu, Y. A. .0654, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06	64 51 37 20 91 35 95 18 97 54 02 97 60 526
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, Y. .0654, 06 Yuu, Y. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06	64 51 37 20 91 35 95 18 97 54 02 97 60 526
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, Y. A. .03 Yu, Y. A. .0654, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06	64 51 37 20 691 35 695 18 697 654 02 997 660 626 82
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .066 Yu, L. .1135, 11 Yu, X. .066 Yu, Y. .0654, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .0654, 06 Yuan, X. .067 Yue, Y. .066 Yuen, H. M. .0882, 08	64 51 37 20 91 35 95 18 97 54 02 97 60 26 82 88
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, Y. A. .03 Yu, Y. A. .0654, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06 Yuen, H. M. .0882, 08 Yuenan, N. .07	64 51 37 20 91 35 95 18 97 60 26 26 82 86 16
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, Y. A. .03 Yu, Y. A. .0654, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06 Yuen, H. M. .0882, 08 Yuenan, N. .07 Yüksel, D. .0916, 09 Yun, S. .0021, 0021, 00	64 51 37 20 91 35 95 18 97 60 26 82 86 16 24
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, X. .03 Yu, Y. A. .0684, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06 Yuen, H. M. .0882, 08 Yuenan, N. .07 Yüksel, D. .0916, 09 Yun, S. .0021, 0021, 00 Yunker, W. .09	64 51 37 20 91 35 95 18 97 60 26 82 86 16 24 08
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, X. .03 Yu, Y. A. .0684, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06 Yuen, H. M. .0882, 08 Yuenan, N. .07 Yüksel, D. .0916, 09 Yun, S. .0021, 0021, 00 Yunker, W. .09 Yusuf, H. .0807, 08	64 51 37 20 91 35 95 18 97 60 26 82 86 26 82 86 16 24 908 807
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, Y. .066 Yu, Y. .06 Yu, Y. .06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .06 Yue, Y. .0654, 06 Yuan, X. .0033, 0117, 0302, 03 Yuan, N. .007 Yu-Cheng, L. .06 Yue, Y. .0654, 06 Yuen, N. .07 Yusel, D. .06 Yuen, S. .0021, 0021, 00 Yun, S. .0021, 0021, 00 Yunker, W. .09 Yusuf, H. .0807, 08 Zaccaria, J. .05	64 51 37 20 91 35 95 695 697 602 97 600 626 82 86 16 24 08 607 84
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, X. .03 Yu, Y. A. .0684, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .0654, 06 Yuen, Y. .0654, 06 Yuan, X. .0033, 0117, 0302, 03 Yuen, N. .0033, 0117, 0302, 03 Yuen, N. .07 Yu-Cheng, L. .06 Yuen, Y. .0614, 06 Yuen, Y. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06 Yuen, Y. .0614, 06 Yuen, Y. .0021, 0021, 00 Yun, S. .0021, 0021, 00 Yunker, W. .09 Yusuf, H. .0807, 08 Zaccaria, J. .05 ZAGANAS, I. .1120, 11	64 51 37 20 91 35 95 18 97 54 02 97 60 26 82 86 16 24 08 84 30
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, Y. .0654, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06 Yue, Y. .0654, 06 Yuen, N. .077 Yu-Cheng, L. .06 Yuen, N. .077 Yuksel, D. .0916, 09 Yun, S. .0021, 0021, 00 Yunker, W. .09 Yusuf, H. .0807, 08 Zaccaria, J. .05 ZAGANAS, I. .1120, 11 Zajichek, A. .0583, 05	64 51 37 20 91 35 95 18 97 60 26 82 86 16 24 86 124 86 124 86 124 83 83
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, X. .03 Yu, Y. A. .0684, 06 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06 Yuen, H. M. .0882, 08 Yuenan, N. .07 Yüksel, D. .0916, 09 Yun, S. .0021, 0021, 002 Yusuf, H. .0807, 08 Zaccaria, J. .05 ZAGANAS, I. .1120, 11 Zajichek, A. .0583, 05 Zammit, G. .0479, 0502, 0502, 05	64 51 37 20 91 35 95 18 97 60 26 26 27 60 26 26 26 26 26 26 26 26 26 26 26 26 26
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, X. .03 Yu, Y. A. .088 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .00 Yue, Y. .0654, 06 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06 Yue, Y. .0654, 06 Yuen, N. .07 Yu-Cheng, L. .0033, 0117, 0302, 03 Yuen, N. .07 Yuesel, D. .0916, 09 Yun, S. .0021, 0021, 002 Yun, S. .0021, 0021, 002 Yusuf, H. .0807, 08 Zaccaria, J. .05 ZAGANAS, I. .1120, 11 Zajichek, A. .0583, 05 Zammit, G. .0479, 0502, 0502, 05 Zamora, T. .06 <td>64 51 37 20 91 35 95 18 97 64 02 97 60 26 82 86 026 82 86 026 82 86 026 83 83 30 83 30 83</td>	64 51 37 20 91 35 95 18 97 64 02 97 60 26 82 86 026 82 86 026 82 86 026 83 83 30 83 30 83
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, Y. .0654, 06 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .00654, 06 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .007 Yu-Cheng, L. .06 Yuen, Y. .0654, 06 Yuen, S. .0023, 0117, 0302, 03 Yuan, X. .007 Yu-Cheng, L. .006 Yue, Y. .065 Yuenan, N. .07 Yüksel, D. .0916, 09 Yun, S. .0021, 0021, 002 Yusuf, H. .0807, 08 Zaccaria, J. .05 ZAGANAS, I. .1120, 11 Zajichek, A. .0583, 05 Zammit, G. .0479, 0502, 0502, 05 Zamora, T. .06	64 51 37 20 91 35 95 60 26 26 27 60 26 26 26 26 26 26 26 26 26 26 26 26 26
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, X. .03 Yu, Y. A. .088 Yuan, R. K. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .00 Yue, Y. .0654, 06 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .07 Yu-Cheng, L. .06 Yue, Y. .0654, 06 Yuen, N. .07 Yu-Cheng, L. .0033, 0117, 0302, 03 Yuen, N. .07 Yuesel, D. .0916, 09 Yun, S. .0021, 0021, 002 Yun, S. .0021, 0021, 002 Yusuf, H. .0807, 08 Zaccaria, J. .05 ZAGANAS, I. .1120, 11 Zajichek, A. .0583, 05 Zammit, G. .0479, 0502, 0502, 05 Zamora, T. .06 <td>64 51 37 20 91 35 95 60 26 26 27 60 26 26 26 26 26 26 26 26 26 26 26 26 26</td>	64 51 37 20 91 35 95 60 26 26 27 60 26 26 26 26 26 26 26 26 26 26 26 26 26
Youngstedt, S. .0863, 08 Youngstedt, S. D. .0401, 0550, 08 Yu, F. .11 Yu, G. .10 Yu, J. L. .06 Yu, L. .1135, 11 Yu, TY. .06 Yu, Y. .0654, 06 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .00654, 06 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .0033, 0117, 0302, 03 Yuan, X. .007 Yu-Cheng, L. .06 Yuen, Y. .0654, 06 Yuen, S. .0023, 0117, 0302, 03 Yuan, X. .007 Yu-Cheng, L. .006 Yue, Y. .065 Yuenan, N. .07 Yüksel, D. .0916, 09 Yun, S. .0021, 0021, 002 Yusuf, H. .0807, 08 Zaccaria, J. .05 ZAGANAS, I. .1120, 11 Zajichek, A. .0583, 05 Zammit, G. .0479, 0502, 0502, 05 Zamora, T. .06	64 51 37 20 91 35 95 18 97 60 26 27 60 26 26 27 60 26 26 27 60 26 26 26 26 26 26 26 26 26 26 26 26 26

Zarrouf, L. R
Zee, P
Zee, P. C
Zeepvat, J
Zeidler, M
Zeidler, M. R
Zeineddine, S
Zeitzer, J
Zeitzer, J. M
Zelazny, J
Zelazny, J. H
Zemel, B
Zemel, B. S
Zemski-Berry, K. A
Zendels, P
Zeng, D
Zetterberg, H0422
Zhang, C
ZHANG, C
Zhang, J
Zhang, J
Zhang, J
Zhang, L
Zhang, N
Zhang, R
ZHANG, X
Zhang, X
Zhang, Y
Zhang, Y
Zhang, Z
Zhang, Z. Y
Zhao, J
Zhao, L
Zhao, R

Zhao, W
Zhaoyang, R
Zheng, L
Zhivotovsky, S
Zhong, C
Zhou, B
Zhou, E
Zhou, E. S
Zhou, J
Zhou, J
Zhou, J
Zhou, K
Zhou, M
Zhou, Z
Zhu, J
Zhu, L
Zhu, R
Zhu, R
Zhuang, S
Zidan, M. H
Zielinski, M. R
Zimmerman, M
Zinchuk, A
Zinke, P
Zipunnikov, V
Zitting, KM
Zizi, F
Zizi, F
Zou, J
ZOU, J
ZOU, J
Zrebiec, J
Zucconi, M
Zurlinden, T
Zummuch, 1

Keyword Index

Α

5xFAD mice
abuse
Academic Achievement
Academic Performance
Acceptance and Commitment Therapy (ACT)
Access to Care
Acoustic Neuromodulation
actigraphic sleep
actigraphy 0057, 0131, 0151, 0183, 0192, 0204, 0210, 0215, 0231,
0258, 0343, 0380, 0388, 0398, 0402, 0421, 0741, 0777, 0813, 0819,
0820, 0858, 0939, 0954, 0974, 0976, 1018, 1043, 1051, 1098, 1110,
1142, 1144, 1199 actigraphy-measured sleep
actigraphy-measured sleep parameters
0127, 0138, 0139, 0177, 0196, 0250, 0318, 0333, 0337, 0342, 0360,
0390, 0852, 0855, 0862, 0915, 0917, 1005, 1029, 1033, 1036, 1068,
1091, 1120, 1130, 1145, 1196, 1199
activities
Activity
activity tracking
acupuncture
Acute Coronary Syndrome
Acute Sleep Deprivation
Adaptive Psychophysics
addiction
adenotonsillar increase
Adenotonsillectomy
ADHD0327, 0757, 0957, 0967, 0983, 1092, 1109, 1111
adherence 0568, 0632, 0636, 0646, 0652, 0654, 0662, 0666, 0668,
0683, 0717, 0861, 0886, 0888, 1106
adiponectin-hypoadiponectinemia
adipone tissue
adolescence
adolescente
0328, 0331, 0332, 0337, 0339, 0360, 0381, 0390, 0394, 0876, 0888,
0915, 0917, 0921, 0924, 0927, 0940, 0945, 0952, 0957, 0963, 0996
Adolescents
adult
0452, 0500, 0561, 0633, 0634, 0673, 0693, 0740, 0752, 0753, 0763,
0772, 0786, 0822, 0823, 0850, 0852, 0854, 0861, 0926, 1023, 1045,
1147, 1207
Adults
Adverse Event
affect
African American
afternoon sleep
age
Ageing
aging
. 0063, 0091, 0119, 0122, 0251, 0346, 0347, 0349, 0350, 0351, 0354,
0829, 0863, 0866, 1137
AHI
air pollution
Air Purifier
airline pilots
alcohol
alcohol craving
alcohol use disorder

alertness.	
Alertness Maximization.	
alertness/motivation.	
algorithm	
Allergies.	
Alpha-synuclein.	
Alternating Leg Muscle Activation	
Alzheimer	
Alzheimer's disease	,0415,0419,0438,1150
Alzheimer's Disease.	
Alzheimer's Disease (AD)	
Ambulatory blood pressure	
ambulatory EEG.	
ambulatory sleep monitoring.	
amplitudes	
Anesthesia	
anger	
animal model	
. 0001, 0008, 0023, 0072, 0074, 0126, 0141,	0142, 0148, 0155, 0224,
	0418
Animals	0432
Anterior insula.	
Anti Streptolysin O (ASO); Anti DNAse B (A	
Anticipation	
antidepressant.	
anxiety	
anxiety/depression	
0188, 0570, 0833, 0939, 0945, 0948, 0967	, 1037, 1064, 1092, 1101
APAP	
Apnea.	
apnea hypopnea index	
apnea hypopnea index (AHI)	
apnea hypopnea index (AHI)	0570, 0574, 0587, 0600,
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835	0570, 0574, 0587, 0600, , 0893, 0898, 1010, 1069
apnea hypopnea index (AHI)	0570, 0574, 0587, 0600, , 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835	0570, 0574, 0587, 0600, , 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression Apnea-Hyponea Index	0570, 0574, 0587, 0600, , 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression Apnea-Hyponea Index Apnea-Hypopnea Index	0570, 0574, 0587, 0600, , 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression Apnea-Hyponea Index Apnea-Hypopnea Index	0570, 0574, 0587, 0600, , 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression Apnea-Hypopnea Index Appetite hormones Applications	0570, 0574, 0587, 0600, , 0893, 0898, 1010, 1069 0591 0730 0732 0317 1212
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression Apnea-Hyponea Index Apnea-Hypopnea Index Appetite hormones Applications applied research	0570, 0574, 0587, 0600, ,0893, 0898, 1010, 1069 0591 0730 0732 0317 1212 0219
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069 0591 0730 0732 0317 1212 0219 0621
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069 0730 0730 0732 0317 1212 0219 0621 0672
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES.	0570, 0574, 0587, 0600, ,0893, 0898, 1010, 1069 0730 0732 0317 1212 0219 0621 0672
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil.	0570, 0574, 0587, 0600, ,0893, 0898, 1010, 1069 0730 0732 0732 0317 1212 0219 0672 0143, 0889
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. Applications. ARES. Armodafinil. arousal. Arousals. Artificial carbonated bathing. Artificial Intelligence.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. Artificial carbonated bathing. Artificial light at night.	0570, 0574, 0587, 0600, , 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. Artificial carbonated bathing. Artificial light at night. ASD.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial light at night. ASD. Asian population.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069 0591 0730 0732 0317 0219 0621 0672 0143, 0889 0143, 0889 0164 0518 0518 0383 0383 0757, 1002 0627
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. Artificial carbonated bathing. Artificial Intelligence. artificial light at night. ASD. Asian population. ASL.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069 0591 0730 0732 0317 0219 0621 0672 0143, 0889 0144, 0792, 1201 0383 0442, 0792, 1201 0383 0757, 1002 0627 0060
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial light at night. ASD. Asian population.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069 0591 0730 0732 0317 0219 0621 0672 0143, 0889 0144, 0792, 1201 0383 0442, 0792, 1201 0383 0757, 1002 0627 0060
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. Artificial carbonated bathing. Artificial Intelligence. artificial light at night. ASD. Asian population. ASL.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial Intelligence. artificial light at night. ASD. Asian population. Assessment.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial Intelligence. artificial light at night. ASD. Asian population. ASL. Assessment. atherosclerosis. Athlete.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial light at night. ASD. Asian population. ASL. Assessment. atherosclerosis. Athlete athletes.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial light at night. ASD. Assessment. atherosclerosis. Athlete. Athletes. Athletic performance.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial light at night. ASD. Assessment. atherosclerosis. Athlete. Athlete. Atonia.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. Artificial carbonated bathing. Artificial light at night. ASD. Assessment. atherosclerosis. Athlete. Athlete. Atopica dermatitis. Arousal. Artificial carbonated bathing. Artificial light at night. ASL. Assessment. Athlete. Athlete. Atonia. Atopic dermatitis.	0570, 0574, 0587, 0600, ,0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial light at night. ASD. Asian population. Assessment. atherosclerosis. Athlete. Athlete. Atonia. atopic dermatitis. atrial fibrillation.	0570, 0574, 0587, 0600, ,0893, 0898, 1010, 1069 0591 0730 0732 0317 1212 0219 0621 0672 0143, 0889 0164 0130, 0137 0518 0442, 0792, 1201 0383 0757, 1002 0627 0660 0399, 0742 0890 0206, 0226, 0228, 0240 0204 0066 0987 0589
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial light at night. ASD. Asian population. Assessment. atherosclerosis. Athlete. Athlete. Atonia. atopic dermatitis. attial fibrillation. Attention.	0570, 0574, 0587, 0600, 0893, 0898, 1010, 1069 0591 0730 0732 0317 1212 0219 0621 0672 0143, 0889 0164 0130, 0137 0518 0442, 0792, 1201 0383 0757, 1002 0627 0660 0399, 0742 0890 0236 .0206, 0226, 0228, 0240 0204 0066 0987 0589 .0166, 0254, 0287, 0312
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial Intelligence. artificial light at night. ASD. Asian population. ASL. Assessment. atherosclerosis. Athlete. Athlete. Athlete. Athletic performance. Attention. Attention. Attention. Attention. Attention Deficit Hyperactivity Disorder.	0570, 0574, 0587, 0600, ,0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial Intelligence. artificial light at night. ASD. Asian population. ASL. Assessment. atherosclerosis. Athlete. Athlete. Athlete. Athletic performance. Attention. Attention. Attention. Attention Deficit Hyperactivity Disorder. Attentional Bias.	0570, 0574, 0587, 0600, ,0893, 0898, 1010, 1069
apnea hypopnea index (AHI) . 0059, 0170, 0348, 0435, 0436, 0561, 0562, 0608, 0633, 0702, 0703, 0835 Apnea Progression. Apnea-Hypopnea Index. Apnea-Hypopnea Index. Appetite hormones. Applications. applied research. ARES. Armodafinil. arousal. Arousals. arterial stiffness. Artificial carbonated bathing. Artificial Intelligence. artificial light at night. ASD. Asian population. ASL. Assessment. atherosclerosis. Athlete. Athlete. Athlete. Athletic performance. Attention. Attention. Attention. Attention. Attention Deficit Hyperactivity Disorder.	0570, 0574, 0587, 0600, ,0893, 0898, 1010, 1069

Augmentation
augmentation pressure
Auricular Point Acupressure
Autism
Autism Spectrum Disorder
AutoBIPAP therapy
Autoimmune Neurology
Automatic sleep scoring
autonomic
autonomic-central coupling
awareness of DME company

B

back pain		
ballistocardiography		
bariatric surgery		
baroreflex sensitivity		
Basal Forebrain		
base of tongue		
Bedsharing		
Bedtime Procrastination		
bedtime resistance		
behavior		
behavioral activity rhythm		
behavioral alertness		
Behavioral Pleiotropy		
behavioral problems		
behavioral sleep problems		
behavioral treatment		
bench to bedside		
benzodiazepine		
big data 0201, 0386, 0440, 0443, 0444, 0452, 0820, 0850, 1008,		
1043, 1145, 1152, 1164, 1180, 1200, 1205, 1214		
binaural auditory beats		
Bioavailability0745		
Biofeedback		
Bioinformatics		
biomarker		
Biomarkers		
birds		
Blood Pressure		
Blood Pressure Monitoring		
Blue Light		
Board-certification		
Body Mass Index		
Body Mass Index (BMI)		
body temperature		
Bone		
Brain		
brain networks		
Brainstem		
brainstem circuits		
breast cancer		
breathing disorders		
Bright light		
Bruxism		
С		
Caffeine		

Caffeine	
cancer	

0025, 0381, 0599, 0990, 0994, 1006, 1019, 1023, 1030, 1031, 1036, 1037, 1041, 1045, 1053

Cancer Survivors
cancer symptoms
cannabinoid
Cannabis
CAPS
cardiac
cardiac autonomic activity
Cardiometabolic Disease
cardiometabolic health
cardiometabolic outcomes 0035, 0138, 0139, 0342, 0585, 0641,
0693, 0772, 0864, 0891, 0936, 1034
Cardiometabolic Risk
cardiomyocytes
Cardiovascular
Cardiovascular Disease
Cardiovascular Health
cardiovascular outcomes
cardiovascular physiology0556
cardiovascular risk factor
caregiver
caregivers
caregiving
Caretaker
Cataplexy
catathrenia
CBT
CBT-I
CD1-1
0470, 0475, 0476, 0483, 0489, 0499, 0501, 0506, 0507, 0512, 0522,
0524, 0526, 0530, 0531, 0534, 0540, 0655, 0924, 1030, 1031, 1186
CBTi
CCHS
Central Sleep Apnea
cerebral blood flow
Cerebral Perfusion
Cerebral small vessel disease
Cereset Research
Cerner
Chewing
Cheyne Stokes Respirations
Child life therapy
Childhood Maltreatment
childhood Maltreatment
children 0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0956, 0956
children 0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0956, 0956
children 0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0956, 0958 Children sleep
children 0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0956, 0958 Children sleep
children 0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0956, 0958 Children sleep
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0956, 0958 Children sleep0933 Children's Sleep0971 Chinese children0909 Chinese medicine0516
children 0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0956, 0958 Children sleep
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 Children sleep0933 Children's Sleep0971 Chinese children0909 Chinese medicine0516 cholesterol0663 choose sleep1169
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 Children sleep0933 Children's Sleep0971 Chinese children0909 Chinese medicine0516 cholesterol0663 choose sleep1169 chronic intermittent hypoxia0237, 0424
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 Children sleep0933 Children's Sleep0971 Chinese children0909 Chinese medicine0516 cholesterol0663 choose sleep1169 chronic intermittent hypoxia0237, 0424 chronic neck pain
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 Children sleep0933 Children's Sleep0971 Chinese children0909 Chinese medicine0516 cholesterol0663 choose sleep1169 chronic intermittent hypoxia0237, 0424 chronic neck pain1215 chronic obstructive pulmonary disease0547, 0696, 1021
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 0956, 0958 Children sleep0933 Children's Sleep0971 Chinese children0909 Chinese medicine0516 cholesterol0663 choose sleep1169 chronic intermittent hypoxia0237, 0424 chronic neck pain1215 chronic obstructive pulmonary disease0547, 0696, 1021 Chronic Pain0077, 0259, 0491, 0517, 0530
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 0956, 0958 Children sleep0933 Children's Sleep0971 Chinese children0900 Chinese medicine0516 cholesterol0663 choose sleep1169 chronic intermittent hypoxia0237, 0424 chronic neck pain1215 chronic obstructive pulmonary disease0547, 0696, 1021 Chronic Pain0077, 0259, 0491, 0517, 0530 chronic sleep and respiratory disorders
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 Ohildren sleep0933 Children's Sleep0971 Chinese children0909 Chinese medicine0516 cholesterol0663 choose sleep1169 chronic intermittent hypoxia0237, 0424 chronic neck pain
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 Children sleep0933 Children's Sleep0971 Chinese children0909 Chinese medicine0913 Children's Sleep0971 Chinese children0909 Chinese medicine
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 Ohildren sleep0933 Children's Sleep0971 Chinese children0909 Chinese medicine0516 cholesterol0663 chorose sleep1169 chronic intermittent hypoxia0237, 0424 chronic neck pain1215 chronic obstructive pulmonary disease0547, 0696, 1021 Chronic Pain0077, 0259, 0491, 0517, 0530 chronic sleep and respiratory disorders
children 0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0956, 0958 Children sleep
children0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0956, 0958 Children sleep0933 Children's Sleep0971 Chinese children0909 Chinese medicine0516 cholesterol0663 choose sleep1169 chronic intermittent hypoxia0237, 0424 chronic neck pain1215 chronic obstructive pulmonary disease0547, 0696, 1021 Chronic Sleep and respiratory disorders0957, 0491, 0517, 0530 chronic sleep restriction0077, 0259, 0491, 0517, 0530 chronic sleep restriction0044, 0202, 0245, 0263, 0283, 0336, 0414, 0782, 0783 circadian0018, 0019, 0117, 0364, 0396, 0406, 0421, 1004, 1096,
children 0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 0956, 0958 Children sleep
children 0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0956, 0958 Children sleep
children 0086, 0134, 0312, 0321, 0356, 0877, 0913, 0932, 0956, 0958 0956, 0958 Children sleep

circadian misalignment
circadian preference
circadian rhythm 0006, 0007, 0020, 0032, 0037, 0050, 0059, 0062,
0068, 0170, 0183, 0197, 0201, 0203, 0212, 0214, 0216, 0217, 0339,
0370, 0380, 0381, 0383, 0433, 0471, 0777, 0784, 0785, 0786, 0823,
0985, 0994, 1012, 1145
circadian rhythm sleep-wake disorder
circadian rhythms
Circuit Mapping
CLEANING
clinical trial 0008, 0146, 0198, 0468, 0472, 0500, 0502, 0519, 0606,
0673, 0702, 0703, 0711, 0740, 0752, 0753, 0763, 0780, 0794, 0824,
0963, 1104, 1158
Closed-loop
cloud sleep scoring system
Cloud-based
cluster analysis
CMRO2
co-sleeping
Cognition 0106, 0120, 0123, 0125, 0299, 0319, 0338, 0537, 0639,
0983, 1026, 1026, 1119, 1137
Cognitions
Cognitive Aging
Cognitive anxiety
cognitive behavioral therapy
Cognitive Behavioral Therapy for Insomnia
Cognitive Decline
Cognitive Dysfunction
cognitive enhancement
Cognitive fatigue
Cognitive Flexibility
Cognitive Function
cognitive functioning
cognitive functioning
cognitive perceptions
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123Colorectal adenoma1009Colorectal cancer0044
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123Colorectal adenoma1009Colorectal cancer0044combination therapy0662
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123Colorectal adenoma1009Colorectal cancer0044combination therapy0662Community-Based0621, 1189
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123Colorectal adenoma0099Colorectal cancer0044combination therapy0662Community-Based0621, 1189community-dwelling older men0392, 0393, 0818
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123Colorectal adenoma1009Colorectal cancer0044combination therapy0662Community-Based0621, 1189community; Polio; Polio survivors;.0623
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123Colorectal adenoma1009Colorectal cancer0044combination therapy0662Community-Based0621, 1189community; Polio; Polio survivors;.0623Comorbid Insomnia0077
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123Colorectal adenoma1009Colorectal cancer0044combination therapy0662Community-Based0621, 1189community; Polio; Polio survivors;.0623Comorbid Insomnia0077Comorbid Insomnia0077
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123Colorectal adenoma1009Colorectal cancer0044combination therapy0662Community-Based0621, 1189community; Polio; Polio survivors;.0623Comorbid Insomnia0077
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123Colorectal adenoma1009Colorectal cancer0044combination therapy0662Community-Based0621, 1189community; Polio; Polio survivors;.0623Comorbid Insomnia0077Comorbid Insomnia0077
cognitive perceptions0627Cognitive Performance0056, 0097, 1083Cognitive-Behavioral Therapy for Insomnia0525college0258college students0123Colorectal adenoma1009Colorectal cancer0044combination therapy0662Community-Based0621, 1189community-dwelling older men0392, 0393, 0818Community; Polio; Polio survivors;.0623Comorbid Insomnia0077Comorbid Insomnia & Sleep Apnea (COMISA)0107Comorbid Insomnia and Sleep Apnea0655comorbidity0596, 0620, 0729, 0822, 0823, 0852, 1012, 1019, 1033
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0097 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Community; Polio; Polio survivors; .0623 Comorbid Insomnia. .0077 Comorbid Insomnia and Sleep Apnea. .0655 comorbid Insomnia and Sleep Apnea. .0655 comorbidity. .0596, 0620, 0729, 0822, 0823, 0852, 1012, 1019, 1033 compliance.
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0097 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Community; Polio; Polio survivors; .0623 Comorbid Insomnia. .0077 Comorbid Insomnia & Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 comorbidity. .0596, 0620, 0729, 0822, 0823, 0852, 1012, 1019, 1033 compliance.
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0097 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Community; Polio; Polio survivors; .0623 Comorbid Insomnia. .0077 Comorbid Insomnia & Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 comorbidity. .0596, 0620, 0729, 0822, 0823, 0852, 1012, 1019, 1033 compliance.
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0099 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Comorbid Insomnia. .0077 Comorbid Insomnia & Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 comorbidity. .0596, 0620, 0729, 0822, 0823, 0852, 1012, 1019, 1033 compliance.
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0099 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Comorbid Insomnia. .0077 Comorbid Insomnia & Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 comorbidity. .0596, 0620, 0729, 0822, 0823, 0852, 1012, 1019, 1033 compliance.
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0099 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Comorbid Insomnia. .0077 Comorbid Insomnia & Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 compliance.
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0099 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Comorbid Insomnia. .0077 Comorbid Insomnia & Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 comorbidity. .0596, 0620, 0729, 0822, 0823, 0852, 1012, 1019, 1033 compliance.
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0099 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Comorbid Insomnia. .0077 Comorbid Insomnia & Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 compliance.
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0099 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Comorbid Insomnia. .0077 Comorbid Insomnia & Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 comorbidity. .0596, 0620, 0729, 0822, 0823, 0852, 1012, 1019, 1033 compliance.
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0099 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Community; Polio; Polio survivors; .0623 Comorbid Insomnia. .0077 Comorbid Insomnia & Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 computational modeling. .0124 Computational modeling. .0124 Computerized Cognitive Testing. .1119 Concussion. .0209 conditions. .0798 confusional arousal. .0808 Connexin. .36 0158 Consolidation. .0099 constrained sleep opportunities. .0326
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0099 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Community: Polio; Polio survivors; .0623 Comorbid Insomnia. .0077 Comorbid Insomnia and Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 computational modeling. .0124 Computerized Cognitive Testing. .1119 Concussion. .0299 confusional arousal. .0808 Connexin. .36 0158 Consolidation. .0099 constrained sleep opportunities. .0326 Consumer. .1198
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0099 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Community: Polio; Polio survivors; .0623 Comorbid Insomnia. .0077 Comorbid Insomnia and Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 computational modeling. .0124 Computerized Cognitive Testing. .1119 Concussion. .0299 confusional arousal. .0808 Connexin. .36 0158 Consolidation. .0099 constrained sleep opportunities. .0326 Consumer. .1198 consumer sleep technology. .1192, 1199
cognitive perceptions. .0627 Cognitive Performance. .0056, 0097, 1083 Cognitive-Behavioral Therapy for Insomnia. .0525 college. .0258 college students. .0123 Colorectal adenoma. .0099 Colorectal cancer. .0044 combination therapy. .0662 Community-Based. .0621, 1189 community-dwelling older men. .0392, 0393, 0818 Community: Polio; Polio survivors; .0623 Comorbid Insomnia. .0077 Comorbid Insomnia and Sleep Apnea (COMISA). .0107 Comorbid Insomnia and Sleep Apnea. .0655 computational modeling. .0124 Computerized Cognitive Testing. .1119 Concussion. .0299 confusional arousal. .0808 Connexin. .36 0158 Consolidation. .0099 constrained sleep opportunities. .0326 Consumer. .1198

Continuous positive airway pressure (CPAP) therapy119	0
cooperation	1
СОРД	8
core temperature	5
Cornelia de Lange	2
Coronary artery disease; Atherosclerosis	1
Coronary computed tomography angiography	1
Cortical EEG	8
Cortical Thickness	3
cost	3
Countermeasure	5
CPAP 0348, 0357, 0568, 0590, 0625, 0629, 0632, 0636, 0646, 0647	7,
0651, 0654, 0655, 0663, 0669, 0677, 0686, 0689, 0692, 0706, 0902	2,
0986, 113	1
CPAP adherence	5
CPAP and DME company070	7
CPAP compliance	
craniofacial	2
Critical illness	9
Cross-sectional survey	
CSForexin	7
cumulative impairment	0
cyclic alternating pattern	
cystic fibrosis	4

D

daily affect
daily blue light
daily morning blue light therapy
daily rhythm
Daily Rhythms
daylight exposure
daylight savings time
Daytime Functioning
Daytime Impairment
daytime nap
daytime naps
daytime sleepiness
deamidation
Decision making
declarative learning
declarative memory
Deep Learning
delayed phase
Delirium
delivery system
delta power
dementia 0341, 0416, 0422, 0425, 0487, 0488, 0543, 1120, 1130
1131, 1131, 1152, 1156, 1157, 1185
dementia severity
Dementia with Lewy bodies
demographics
Denture
Depression 0037, 0070, 0185, 0212, 0258, 0394, 0407, 0453, 0508
0533, 0533, 0540, 0712, 0733, 0736, 0738, 0771, 0775, 0819, 0935
0958, 0973, 0992, 1019, 1063, 1084, 1086, 1087, 1088, 1090, 1091
1094, 1095, 1097, 1098, 1099, 1100, 1100, 1117, 1140
Descriptive model
development
developmental epidemiology
Device
Device Usage

Devices
Diabetes
Diagnosis
Diagnostic strategy
Diagnosuc strategy
diet
dietary nitrate supplement
difficulty in falling asleep
diffusion tensor imaging
digital CBT-I
Digital SCreen
digital therapeutic
Direct referral
Disagnosis and treatment
discrimination
DISE
Disfacilitation
Disparities
disrupted nighttime sleep
disrupted sleep
disruption
Diversity
Domain
Donepezil
dopamine
Dopamine Agonists
Dopamine receptor D2
Down syndrome
dream
Dreaming
Dreams
Driving
dropout
*
Drosophila
Dual Orexin Receptor Antagonist
Durability
Duration
Dynamic
dynamic functional connectivity
dysfunction
Dysmenorrhea
dystrophy

E

Early childhood
education
EEG0001, 0004, 0051, 0093, 0094, 0100, 0101, 0113, 0148, 0169,
0207, 0256, 0280, 0282, 0304, 0319, 0324, 0327, 0340, 0344, 0348,
0422, 0426, 0435, 0440, 0446, 0449, 0452, 0546, 0616, 0669, 0850,
0989, 1008, 1072, 1075, 1136, 1147, 1192, 1195, 1210, 1211
EEG monitoring
EEG Reference
EEG-fMRI
effectiveness
efficacy
effort
EHLERS DANLOS1042
elderly population
electronic health record
emergency worker
Emotion

emotion perception
emotional and behavioral problem
Emotional Distress
Emotional Memory
Emotional memory consolidation
Emotional Reactivity
employee health
employee wellness
encoding
End-to-End
Endophenotyping0448
endoscopic sinus surgery0727
endotype
endotypes
Energy expenditure
Enteric disease
environmental exposure
Epidemiology
Epworth Sleepiness Scale (ESS)14, 0024, 0080, 0227
0355, 0580, 0603, 0702, 0703, 0706, 0711, 0739, 0751, 0756, 0759
esports
ESS
ethnicity
Eveningness
excessive daytime sleepiness
executive function
Executive Functioning
Exercise
exercise test
Exhalant
Exosomes
experiment
Eye-blink parameters

F

Face Perception	49
face-name	17
Factors	57
factors influencing care	24
Falling asleep	
False Memory	
false negative	
fatigue	
~	
. 0055, 0184, 0218, 0233, 0236, 0244, 0279, 0286, 0298, 0481, 056	54,
0748, 0771, 1101, 111	18
Fatigue-management Tool	34
fear conditioning and extinction	65
feasibility	95
Female	60
Female Collegiate Athletes	31
females	69
Ferric Carboxymaltose	01
FFT-analyses	37
Fiber photometry	
fibroblast growth factor	
fibromyalgia	
Firefighters	
first responder	
First Responders	
Fitbit	
Fitness 032	

flow limitation
fluid overload
fMRI0010, 0064, 0079, 0111, 0455
Food
Food Effect
food insecurity
food intake
Forced Desynchrony
Forecasting Model
forgetting
Forward Model
Framing Bias
Free
free flap reconstruction surgery
functional connectivity
functional mri

G

GABA0067
Gabapentin Enacarbil
gender0187, 0362, 0412, 0508, 0595, 0760, 0826, 0845, 0945, 1064
Gender differences
general population
generalisation
generalized anxiety disorder
Genetic syndrome
genetics
geography
gestational
gestational diabetes
glucose
glycemic control
Glycomics
goal settings
graph theory
Gravity imagery
Gray Matter Volume
green space
Greenspace
growth hormone
GWAS0016

Η

Habitual sleep
Haiti
1082
Halucinogens
hdEEG
Head and Neck Cancer
head band
head elevation
headache
health
Health Behavior
health behaviors
health disparities0183, 0358, 0359, 0360, 0361, 0365, 0366, 0367,
0368, 0369, 0370, 0378, 0457, 0464, 0608, 0617, 0784, 0862, 0919,
0988, 0995, 1168, 1178
health literacy
health outcomes
health promotion
Health Services

healthcare delivery
Healthcare Professionals
Healthfacts
Healthy Adults
healthy diet indicator
Heart Failure
heart rate
Heart Rate Variability
Heavy Vehicle Driver
height
Hepcidin
High Altitude
high blood pressure
High Density EEG
High School Students
high-fat diet
Hippocampus
Hispanic/Latino
Hispanics
histamine
HIV
Homeysteine
home sleep apnea test 0436, 0576, 0579, 0587, 0590, 0600, 0605,
0610, 0619, 1010, 1163, 1170, 1177, 1177, 1182, 1202, 1207
Home sleep apnea testing
Homeostasis
Homeostasis
hormones
Hospital sleep consolidation
hot flashes
HRV
Hydration
hyperarousal
hyperglycemia
hypersomnia.
0026, 0045, 0355, 0427, 0428, 0466, 0742, 0746, 0748, 0754, 0755,
0020, 0043, 0333, 0427, 0428, 0400, 0742, 0740, 0740, 0754, 0753, 0756, 0758, 0760, 0767, 0769, 0771, 0936, 0953, 0953, 1107
hypersomnolence
Hypertension
Hypnagogic Foot Tremor
hypnotic dependence
hypnotics
hypocretin
Hypoglossal nerve simulator
hypoglossal nerve stimulation 0628, 0637, 0645, 0665, 0674, 0676,
0691
Hypopnea
hypothalamus
hypothyroidism
hypoxemia
Hypoxia
Hypoxic Burden

I

ICT
idiopathic hypersomnia
image
immune system
impairment
Impulsivity
incident reduced sleep efficiency

incidental encoding	
Inconsistent	
independent component analysis	
Individual Differences	
Infancy	
•	
Infant	
Infant sleep0161, 0318, 0329, 0946, 09	
inflammasome	
Inflammation	044, 0292, 0419, 0585, 1112
Inflammatory Markers.	
injuries	
injury	
inpatient care	
insomnia 0004, 0007, 0021, 0021, 00	
0194, 0233, 0240, 0273, 0275, 0279, 03	
0456, 0457, 0458, 0459, 0461, 0462, 04	63, 0464, 0465, 0466, 0467,
0471, 0472, 0473, 0474, 0476, 0477, 04	78, 0479, 0480, 0481, 0482,
0483, 0484, 0485, 0486, 0487, 0488, 04	
0495, 0496, 0497, 0499, 0502, 0503, 05	
0510, 0511, 0512, 0514, 0515, 0516, 05	
0525, 0526, 0528, 0529, 0531, 0532, 05	
0540, 0541, 0542, 0543, 0544, 0546, 05	
0552, 0554, 0615, 0645, 0736, 0810, 08	
0847, 0860, 0868, 0892, 0918, 0919, 09	20, 0921, 0923, 0923, 0924,
0925, 0926, 0929, 1017, 1030, 1031, 10	37, 1051, 1059, 1073, 1087,
1092, 1108, 1109, 1110, 1111, 1116, 1	
insomnia disorder.	
Insomnia Severity.	
insomnia symptoms.	
insufficient sleep0	
insufficient sleep duration	
insulin resistance	
insulin sensitivity	
integrated model.	
Intellectual giftedness	
interoceptive-sensitivity.	
Interrogation.	
8	
intervention	,
Interviewing	
intraoperative ultrasound	
Intrathecal Baclofen Therapy	
intrinsic factors	
ЮТ	
iRBD	
Israel.	
IV iron	
· · · · · · · · · · · · · · · · · · ·	

J

Jet lag sleep	8
Jet travel	8
Job Demands	3
Juvenile Justice	9

K

kidney transplantation	99
Kleine-Levin syndrome	26
klotho	22

L

Language	989
Large dataset	9441

Latent Class Analyses0607, 0609Latinx children0320Lavandula angustifolia0498
leadership
Learning
leep-related breathing disorders
lemborexant0473, 0474, 0477, 0478, 0479, 0480, 0481, 0484, 0486
lifestyle
lifestyle behaviors
Light
Light exposure
light sensitivity
lipid metabolism
•
liquid consumption
LIWC
Long-haul aviation
Longitudinal changes
Loose teeth
low back pain
Low frequency electrical stimulator
low income
lucid
Lucid dreaming
lung function

Μ

Machine Learning0062, 0438, 0594, 1188, 1188, 1186,
1196, 1196, 1206, 1208, 1211, 1211
macronutrient oxidation
Maladaptive thouhgts about sleep
Male Fertility
mandibular advancement device
Mandibular movement
mannual scoring
marijuana
Marital Relationship
marksmanship0215
Maschine learning
maternal care0272
Maternal Child Health
maternal sleep
mathematical modeling
Mathematics
meal times
measurement
Mechanical ventilation
media
Media use
Medical Student
medical students
Medicare
medication
medications
mediterranean diet
Melatonin0030, 0039, 0045, 0050, 0433, 0471, 0545, 0779, 0994
melatonin rhythm
memory0080, 0086, 0087, 0089, 0089, 0093, 0094, 0096, 0098,
0100, 0101, 0108, 0109, 0113, 0115, 0118, 0120, 0121, 0198, 0332,
0334, 0337, 0391, 0449, 0502, 0731, 0835, 1153
Memory consolidation

memory deficits
Menopause
menstrual cycle
Menstruation
mental disorders
mental health
meta-analysis
•
metabolic outcomes
metabolic syndrome
metabolism
metabolomics
methadone treatment
methamphetamine
method
Mice
Microbial Composition
Microbiome
Microglia
Middle Childhood0371
middle-school
Midpoint
migraine
mild cognitive impairment
mild OSA
Mild Traumatic Brain Injury
military 0024, 0046, 0188, 0189, 0199, 0215, 0219, 0242, 0273,
0284, 0298, 0385, 0399, 0462, 0620, 0810, 1103, 1155
Milk
Mindfulness
0824, 0824
Minority and Low SES
Misinformation Effect0116
Misinformation Effect
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507
Misinformation Effect0116Misperception1115mobile0206, 0507Mobile Application1213
Misinformation Effect0116Misperception1115mobile0206, 0507Mobile Application1213mobile health0964
Misinformation Effect0116Misperception1115mobile0206, 0507Mobile Application1213mobile health0964modeling0218
Misinformation Effect0116Misperception1115mobile0206, 0507Mobile Application1213mobile health0964modeling0218modification effect0713
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mortality. .0048, 0457, 0599
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0420
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0420 MSLT. .0150
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mortality. .0048, 0457, 0599 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0420 MSLT. .0150 mTBI. .0209
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mottality. .0048, 0457, 0599 Motor activity. .1135, 1141 Motor activity. .1135, 1141 Motor Memory Consolidation. .0104 MRI. .0420 MSLT. .0150 mTBI. .0209 multidimensional sleep. .0345
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0420 MSLT. .0150 mTBI. .0209 multidimensional sleep. .0345 multiple object tracking. .0254
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0420 MSLT. .0150 mTBI. .0209 multidimensional sleep. .0345 multiple object tracking. .0254 Multiple Sclerosis. .1122, 1128, 1142, 1142
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mortality. .0048, 0457, 0599 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0420 MSLT. .0150 mTBI. .0209 multidimensional sleep. .0345 multiple object tracking. .0254 Multiple Sleep Latency Test. .0741
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mortality. .0048, 0457, 0599 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0420 MSLT. .0150 mTBI. .0209 multidimensional sleep. .0345 multiple object tracking. .0254 Multiple Sleep Latency Test. .0741 Multiple Sleep Latency Test. .0741
Misinformation Effect. .0116 Misperception. .1115 mobile. .0206, 0507 Mobile Application. .1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0222 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0420 MSLT. .0150 mTBI. .0209 multidimensional sleep. .0345 multiple object tracking. .0254 Multiple Sleep Latency Test. .0741 Multiple Sleep Latency Test (MSLT)
Misinformation Effect. 0116 Misperception. 1115 mobile. 0206, 0507 Mobile Application. 1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0209 multidimensional sleep. .0345 multiple object tracking. .0254 Multiple Sleep Latency Test. .0741 Multiple Sleep Latency Test (MSLT) .0280, 0603, 0738, 0758, 0759
Misinformation Effect. 0116 Misperception. 1115 mobile. 0206, 0507 Mobile Application. 1213 mobile health. 0964 modeling. 0218 modification effect. 0713 Mood. 0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. 0292 mood disturbance. 0304 morning cardiovascular function. 0461 Morningness. 0320 Mortality. .0048, 0457, 0599 Mother. 0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0420 MSLT. .0150 mTBI. .0209 multidimensional sleep. .0345 multiple Sleep Latency Test. .0741 Multiple Sleep Latency Test (MSLT)
Misinformation Effect. 0116 Misperception. 1115 mobile. 0206, 0507 Mobile Application. 1213 mobile health. .0964 modeling. .0218 modification effect. .0713 Mood. .0112, 0192, 0250, 0252, 0291, 1089 Mood and cognition. .0292 mood disturbance. .0304 morning cardiovascular function. .0461 Morningness. .0320 Mother. .0323 motivation. .0287, 0336 Motor activity. .1135, 1141 Motor Learning. .0316 Motor Memory Consolidation. .0104 MRI. .0209 multidimensional sleep. .0345 multiple object tracking. .0254 Multiple Sleep Latency Test. .0741 Multiple Sleep Latency Test (MSLT) .0280, 0603, 0738, 0758, 0759

-	
	Ν.
11	
-	•

nap	
nap physiology	0334
napping	1135
narcolepsy	
0008, 0072, 0141, 0142, 0155, 0641, 0739, 0740, 0741, 0)744,
0746, 0749, 0751, 0752, 0753, 0754, 0755, 0756, 0758, 0	
0760, 0761, 0762, 0763, 0764, 0765, 0766, 0767, 0768, 0	
0770, 0772, 0773, 0774, 0941, 0950, 0951, 0961,	
Narcolepsy, cataplexy, post-streptococcal	
nasal airway stent.	
Navy	
neck circumference	
neighborhood.	
neighborhood environment	
nerve stimulation	
Nest-Building.	
Neural Activation.	
Neural Efficiency	
Neurobehavioral performance	
neurobehavioural	
Neurocognition	
neurocognitive function.	
neurodegeneration	
Neurodegenerative	
Neurodegenerative Disorders	
Neurodevelopmental	1161
Neurodevelopmental Disorder.	0968
neurodevelopmental disorders.	1002
neurodevelopmental dysabilities	1132
Neuroimaging $0081, 0082, 0263, 0315, 0331, 0331, 0082, 0263, 0315, 0331, 0082, 0263, 0315, 0331, 0082, 0263, 0315, 0331, 0331, 0331, 0331, 0331, 0331, 0331, 0331, 0331, 0331, 0331, 0331, 0331, 0331, 0331, 0331, 0321,$	0331
Neuroimaging	
Neurological outcomes of stroke.	1149
Neurological outcomes of stroke	1149 0643
Neurological outcomes of stroke	1149 0643 0429
Neurological outcomes of stroke	1149 0643 0429 0521
Neurological outcomes of stroke	1149 0643 0429 0521 0027
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070,
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017 1126
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017 1126 0167
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017 1126 0167 0675
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017 1126 0167 0675 0750
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017 1126 0167 0675 0750 1153
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017 1126 0167 0675 0750 1153
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017 1126 0167 0675 0750 1153 0413
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017 1126 0167 0750 0153 0413 0443
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017 1126 0167 0750 0153 0413 0443 0723 1121
Neurological outcomes of stroke	1149 0643 0429 0521 0027 0970 0409 0395 0970 1063 0806 0239 1070, 1194 0438 0043 0309 1017 1126 0167 0750 0153 0413 0443 0723 1121

NREM	
NREM sleep instability	
NREM vs REM	
nurse	409
Nurses	384
nursing	229
nutrition	375

0

Obesity 0405, 0555, 0583, 0583, 0713, 0873, 0884, 0884, 0899,
0971, 1047, 1049 Obesity Hypoventilation Syndrome
obesity-associated sleep hypoventilation syndrome
objective improvement
Objective Sleep duration, actigraphy
Objective Sleep Time
Obstructive Sleep Apnea 0011, 0022, 0053, 0059, 0148, 0170, 0237,
0373, 0377, 0419, 0424, 0439, 0450, 0551, 0551, 0557, 0559, 0560,
0573, 0577, 0419, 0424, 0439, 0430, 0551, 0551, 0557, 0559, 0500, 0561, 0562, 0565, 0568, 0568, 0569, 0571, 0571, 0572, 0574, 0577,
0578, 0580, 0582, 0585, 0586, 0588, 0589, 0571, 0571, 0572, 0574, 0577, 0578, 0580, 0582, 0585, 0586, 0588, 0589, 0590, 0591, 0594, 0596,
0578, 0580, 0562, 0580, 0580, 0580, 0580, 0597, 0570, 0571, 0574, 0570, 0598, 0600, 0601, 0604, 0606, 0608, 0611, 0613, 0614, 0617, 0620,
0624, 0625, 0626, 0628, 0629, 0631, 0634, 0635, 0637, 0638, 0641,
0645, 0647, 0650, 0651, 0651, 0652, 0653, 0654, 0654, 0657, 0659,
0662, 0664, 0665, 0666, 0667, 0668, 0669, 0671, 0673, 0673, 0674,
0677, 0679, 0679, 0680, 0682, 0683, 0684, 0685, 0686, 0688, 0689,
0690, 0693, 0694, 0695, 0696, 0698, 0705, 0709, 0712, 0713, 0721,
0721, 0722, 0722, 0724, 0729, 0733, 0735, 0735, 0736, 0737, 0749,
0751, 0767, 0804, 0816, 0827, 0835, 0838, 0838, 0853, 0855, 0860,
0873, 0875, 0875, 0876, 0878, 0879, 0880, 0882, 0882, 0884, 0884,
0885, 0885, 0891, 0891, 0893, 0894, 0896, 0898, 0898, 0900, 0901,
0901, 0902, 0904, 0905, 0906, 0907, 0908, 0929, 0968, 1010, 1014,
1033, 1056, 1057, 1060, 1065, 1073, 1094, 1106, 1106, 1128, 1136,
1136, 1138, 1143, 1150, 1155, 1155, 1162, 1170, 1176, 1177, 1177,
1178, 1188, 1188, 1197, 1202, 1206, 1207
Obstructive sleep apnea (OSA)
Obstructive sleep apnea risk
Obstructive sleep apnea syndrome (OSAS)
Obstructive sleep apnea, energy intake
obstructive sleep apnea
obstructuve sleep apnea
obstructive sleep apnea
occupational
Oculometrics
Odds Ratio Product
Older adult
Older Adults.
0036, 0083, 0469, 0475, 0543, 0828, 0836, 0839, 0843, 0844, 0858,
1185
older women
Olfaction Disorders
olfactory dysfunction
online CBTI
Open-label
operational
opiates
Opiods
Opioid Use Disorders
opioids
Optogenetics
oral and oropharynx cancers
oral appliance

Oral appliance therapy
OSA phenotypes
osteoarthritis pain
Ostructive Sleep Apnea
outcome
ovarian cancer
Overlap syndrome
Overnight Memory Recall
Oximetry Screening for OSA1149
oxygen consumption
oxygen desaturation
Oxygen saturation
oxygen supplementation
Oxytocin

P

P bodies
P-waves
Pain
Pandemrix
PAP
PAP Adherence
PAP therapy
PAP therapy adherence
PAP use
parasomnia0801, 0806, 0807, 0807, 0808, 0810, 0815, 0816, 1104
Parent cognition
Parent Sleep
Parent-child's sleep health
parenthood
parenting
Parenting behaviors
Parenting Style
Parkinson disease
Parkinson's
Parkinson's disease
Paroxysmal Atrial Fibrillation
patent foramen ovale
pathogenesis
patient experience
patient reported outcomes
patient safety
Patient satisfaction
patient survey
patient-centered care
Patient-centered outcome research (PCOR)1190
patient-centered outcomes
patient-centered voice of patient
patient-reported outcome
patient-reported outcomes
pediatric 0045, 0087, 0100, 0207, 0319, 0322, 0340, 0397, 0853
0875, 0878, 0882, 0883, 0885, 0886, 0889, 0891, 0893, 0894, 0895
0896, 0898, 0901, 0902, 0904, 0905, 0906, 0907, 0912, 0919, 0920
0926, 0929, 0939, 0941, 0944, 0946, 0947, 0949, 0950, 0951, 0952
0960, 0961, 0962, 0967, 0973, 0975, 0978, 0982, 0984, 0985, 0986
0988, 1003, 1165, 1179, 118
pediatric cancer

Pediatric obstructive sleep apnea. .0903 Pediatric Residents. .1176 Pediatrics. .0897, 0923, 0923, 0953, 0977, 0990 peer intervention. .0717
perfectionism
perimenopause. .0480 Perinatal. .0323 perinatal period. .0841
period
Periodic limb movements. .0805 Personality. .0202
pervasive technology
PET
Pharmacokinetic
pharmacology
0679
phenotypes
photometry
photoperiod
phylogeny
physical activity
Physical actvitiy
piezo
pillow
Pittsburgh Sleep Quality Index
placekeeping
polygraphy0726
polysomnogram
1197
Pontine Reticular Formation. .0067 Poor Sleep Quality. .0519, 0824, 0824
Population based study
population sleep health. .0408 Positional OSA. .0567, 1048
positional sleep apnea
Positive Airway Pressure
Positive Airway Pressure (PAP)
0604, 0633, 0634, 0652, 0664, 0667, 0682, 0683, 0687, 0689, 0694, 0705, 0861, 0883, 0885, 0886, 0888, 0896
positive airway pressure therapy
Post-intensive care
post-traumatic stress disorder (PTSD) 0024, 0065, 0118, 0467, 0582, 0651, 0801, 0817, 1003, 1064, 1065,
1066, 1068, 1069, 1072, 1073, 1074, 1075, 1077, 1078, 1154, 1194 Postmenopause
postpartum
postpartum depression. .0329, 0535 postpartum women. .0851
Postsynaptic Inhibition

posttrauma nightmares	
Power spectral analysis.	1127
Pre-Bedtime Activity, Light.	0922
Pre-sleep arousal	.0145, 0165
Predictive Analytics.	
predictive model.	
pregnancy	0859, 0870
Prenatal.	0930
Preoptic area.	.0076, 0154
preschool	.0222, 0972
preschooler.	0955
preschoolers.	0333
Prescriptions.	0806
presenteeism.	0453
pretreatment motivation.	0536
Prevalence	
Preventative Sleep Health	1148
primary care.	
primary caregiver.	
prior wake.	
Procedural Memory.	
processing speed.	0321
Productivity.	
professional basketball.	
Proinflammatory Markers.	
Propensity Score Subclassification.	
psg	
PSQI.	
Psychological health.	
Psychological Intervention.	
psychometrics.	
Psychomotor vigilance task	
Psychomotor Vigilance Test	
Psychomotor Vigilante Task.	
psychosocial well-being.	
PTSD.	
. 0038, 0081, 0800, 0812, 1063, 1067, 1070, 1071, 1076,	
	1081, 1083
puberty	
public health.	
0032, 0135, 0184, 0187, 0193, 0344, 0361, 0386, 0390,	
0940, 0977, 0978, 1007, 1034, 1047, 1049, 1097, 1103,	
	1200.1214
pulmonary hypertension	0718, 0718
pulmonary hypertension	0718,0718
pulmonary hypertension	0718, 0718 0137 0889
pulmonary hypertension. .0558, pulse pressure.	0718, 0718 0137 0889 0264
pulmonary hypertension	0718, 0718 0137 0264 1100

Q

QT variability	.0571
QT/QTc	
quality improvement.	.1173
quality of life	.0990
Quality of Sleep.	.0129
quantitative EEG	.0350
Questionnaire	, 1191

R

race/ethnic disparities
race/ethnicity
0193, 0357, 0359, 0361, 0365, 0366, 0369, 0391, 0401, 0495, 0562,
0574, 0863, 0864, 1007, 1016, 1077, 1097, 1143, 1150

Racial Health Disparities
Radiotherapy
random forest
Randomized Controlled Trial
randomized trial
Rapid Eye Movement
rapid eye movement (REM) sleep
rapid eye movement sleep
Rapid Eye Movement Sleep Behavior Disorder
RBD
reaction speed
Reaction Time
readiness to change
Recertification
recovery
recovery sleep
referral
refrence channels
Relaxation
Reliability
REM
REM density
REM sleep0012, 0048, 0049, 0067, 0071, 0090, 0124, 0159, 0937
REM sleep behavior disorder (RBD) 0418, 0787, 0815, 0817
REM sleep fragmentation
REM sleep without atonia
REM-related parasomnia
REM/NREM
remote
Remote Monitoring
Remote Patient Monitoring
repetitive negative thinking 0234 0248
repetitive negative thinking
resident
resident
resident
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730
resident0205Residents0218, 0261, 0395residual apnea0704Residual Excessive Sleepiness0635resilience0189, 0314, 0315, 0377, 0775resistin- hyperresistinemia0720resolution0999resolution of inflammation0275respiratory0051Respiratory Depression0730Respiratory infections0631
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resiluin. .0702 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0215 Respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0215 Respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0031, 0980 Rested. .1084
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .1084 Resting cerebral blood flow. .0866
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .1084 Resting cerebral blood flow. .0866 resting state. .0010
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .1084 Resting cerebral blood flow. .0866 resting state. .0010 Resting State Functional Connectivity. .0166
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .1084 Resting cerebral blood flow. .0866 resting State .0010 Resting State Functional Connectivity. .0166 resting-state fMRI. .0724
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .0031, 0980 Rested. .0010 Resting cerebral blood flow. .0866 resting State .0010 Resting State Functional Connectivity. .0166 resting-state fMRI. .0724 restless legs. .0010
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .1084 Resting cerebral blood flow. .0866 resting State .0010 Resting State Functional Connectivity. .0166 resting-state fMRI. .0724
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .1084 Resting cerebral blood flow. .0866 resting State .0010 Resting State Functional Connectivity. .0166 resting-state fMRI. .0724 restless legs. .0010 restless legs. .0010 restless legs. .0010 restless legs. .0010
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory Infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .1084 Resting cerebral blood flow. .0866 resting state. .0010 Resting State Functional Connectivity. .0166 resting state fMRI. .0724 restless legs. .0010 restless legs syndrome. .0615, 0790, 0791, 0795, 0802, 0855, 0928, 1001 restless legs syndrome (RLS) 0794, 0796, 0797, 0798, 0809, 0868
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory Infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .0031, 0980 Rested. .00108 resting state. .0010 resting state. .0010 resting state. .0010 resting state Functional Connectivity. .0166 resting State Functional Connectivity. .0166 resting state fMRI. .0724 restless legs syndrome. .0615, 0790, 0791, 0795, 0802, 0855, 0928,
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory Infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .1084 Resting cerebral blood flow. .0866 resting state. .0010 Resting State Functional Connectivity. .0166 resting state fMRI. .0724 restless legs. .0010 restless legs syndrome. .0615, 0790, 0791, 0795, 0802, 0855, 0928, 1001 restless legs syndrome (RLS) 0794, 0796, 0797, 0798, 0809, 0868
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory Infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .0031, 0980 Rested. .00108 resting state. .0010 resting state. .0010 resting state. .0010 resting state Functional Connectivity. .0166 resting State Functional Connectivity. .0166 resting state fMRI. .0724 restless legs syndrome. .0615, 0790, 0791, 0795, 0802, 0855, 0928,
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin-hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .0010 Resting crebral blood flow. .0866 resting state. .0010 Resting State Functional Connectivity. .0166 resting state fMRI. .0724 restless legs. .0010 restless legs syndrome. .0615, 0790, 0791, 0795, 0802, 0855, 0928, .0010 restless legs syndrome (RLS) 0794, 0796, 0797, 0798, 0809, 0868 Restless sleep disorder. .0928, 0943 Restriction. .0338
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution. .0999 resolution. .0051 Respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .1084 Resting cerebral blood flow. .0866 resting state .0010 Resting State Functional Connectivity. .0166 resting state Functional Connectivity. .0166 resting state fMRI. .0724 restless legs syndrome. .0615, 0790, 0791, 0795, 0802, 0855, 0928,
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .1084 Resting cerebral blood flow. .0866 resting State .0010 Resting State Functional Connectivity. .0166 resting State Functional Connectivity. .0166 resting state fMRI. .0724 restless legs syndrome. .0615, 0790, 0791, 0795, 0802, 0855, 0928,
resident. .0205 Residents. .0218, 0261, 0395 residual apnea. .0704 Residual Excessive Sleepiness. .0635 resilience. .0189, 0314, 0315, 0377, 0775 resistin- hyperresistinemia. .0720 resolution. .0999 resolution of inflammation. .0275 respiratory. .0051 Respiratory Depression. .0730 Respiratory infections. .0631 rest. .0108 Rest Activity Pattern. .0417 rest-activity rhythm. .0031, 0980 Rested. .0010 Resting cerebral blood flow. .0866 resting state. .0010 Resting State Functional Connectivity. .0166 resting State Functional Connectivity. .0166 resting state slegs. .0010 restless legs syndrome. .0615, 0790, 0791, 0795, 0802, 0855, 0928,

Risk factors
Risk of Suicide
Risk Taking Behavior
risk-seeking
RLS
RSWA
Rumination
ruminations
Rural communities

S

C +	0766
safety	
SARCOPENIA	
schedules	
Schema.	
school attendance.	
school performance	
school start time	
school suspensions.	
scoliosis	
scoring consistency	
scratch	
Screeing.	
screening	
SDC	
Self-immersed processing	
Self-Management.	
self-medication.	
self-regulation	
Self-report	
Self-Report Instrument.	
self-reported sleep	
Sensor technology	
sensorimotor.	
Serum Lipids	
Sex	
sex differences	
Shared Decision-Making.	
shift work 0006, 0007, 0025, 0064, 0203, 0223, 0225, 0253, 0	
0284, 0382, 0775, 0778, 0779,	0781
shift-work	
Shiftwork	
Short sleep	
Short-haul aviation.	
sigma band	
Simple snorer	
simulation	0229
Sleep	
0040, 0095, 0104, 0114, 0167, 0185, 0195, 0222, 0260, 0354, 0	
0382, 0396, 0401, 0413, 0423, 0493, 0498, 0518, 0548, 0563, 0	
0829, 0831, 0832, 0871, 0874, 0881, 0916, 0922, 0954, 0956, 0	
0998, 1020, 1022, 1025, 1039, 1040, 1044, 1050, 1062, 1062, 1	1113,
1114, 1123, 1129, 1135, 1141, 1146, 1209,	1213
Sleep and Performance	0255
Sleep anxiety	0073
Sleep Apnea.	
0240, 0448, 0451, 0510, 0564, 0566, 0592, 0593, 0593, 0599, 0)602,
0615, 0616, 0619, 0656, 0661, 0681, 0697, 0697, 0699, 0701, 0	
0714, 0717, 0726, 0728, 0840, 0870, 0877, 0910, 1009,	
sleep behavior.	0262
Sleep Behaviors.	0404

sleep debt
1
sleep deficiency
Sleep Deprivation 0034, 0041, 0042, 0076, 0102, 0121, 0125, 0133,
0143, 0173, 0173, 0265, 0266, 0268, 0270, 0272, 0274, 0281, 0285,
0294, 0294, 0297, 0301, 0305, 0306, 0307, 0308, 0311, 0311, 0312,
0294, 0294, 0297, 0301, 0303, 0300, 0307, 0308, 0311, 0311, 0312,
0314, 0315, 0316, 0915, 1100, 1100
Sleep depth
sleep diary
Sleep Difficulty
sleep disorder
Sleep Disorderd Breathing
sleep disordered breathing 0340, 0581, 0644, 0719, 0890, 0897,
0911, 0914
Sleep disordered breathing; Respiratory mechanism;
sleep disorders0328, 0445, 0504, 1027, 1078, 1112, 1126, 1183
-
Sleep disparities
Sleep disparity
sleep disruption
sleep disturbance 0064, 0298, 0429, 0848, 0851, 0866, 0955, 0987,
•
0997, 1032, 1093
Sleep Disturbances
Sleep Duration 0082, 0140, 0168, 0176, 0221, 0226, 0234, 0235,
•
0236, 0260, 0267, 0277, 0277, 0372, 0379, 0405, 0417, 0420, 0565,
0783, 0846, 0863, 0864, 0865, 0909, 0932, 0995, 1015, 1024, 1035,
1046, 1058, 1076, 1093, 1102, 1117
sleep education 0197, 0246, 0391, 0490, 0531, 0749, 0815, 0826,
0952, 0982, 1060, 1167, 1168, 1169, 1171, 1179, 1181, 1184, 1214
Sleep efficiency, Apnoea Hypopnoea Index
Sleep Environment
Sleep epidemiology
sleep extension
Sleep fragmentation
Sleep habits
sleep health 0177, 0251, 0363, 0408, 0854, 0856, 0857, 1082, 1089,
1166, 1187, 1189
1166, 1187, 1189
1166, 1187, 1189 sleep health promotion
1166, 1187, 1189 sleep health promotion.
1166, 1187, 1189 sleep health promotion.
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Intertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep Mobile. .1212
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep Mobile. .1212 sleep myths. .0408
1166, 1187, 1189 sleep health promotion.
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep Mobile. .1212 sleep noset latency. .0408 sleep parameters. .0599 Sleep Physiology. .01111 sleep problem. .0965
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep myths. .0408 sleep onset latency. .0408 sleep praameters. .1004 Sleep problem. .0965 sleep problems. .1006, 1191
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep myths. .0408 sleep onset latency. .0408 sleep problem. .0965 sleep problems. .1006, 1191 Sleep Quality. .0013,
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep Mobile. .1212 sleep onset latency. .0408 sleep parameters. .1004 Sleep problem. .0965 sleep problems. .1006, 1191 Sleep Quality. .0013, .0145, 0165, 0176, 0179, 0239, 0363, 0384, 0818, 0837, 0841, 0842,
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep Mobile. .1212 sleep onset latency. .0408 sleep parameters. .1004 Sleep problem. .0965 sleep problems. .1006, 1191 Sleep Quality. .0013, .0145, 0165, 0176, 0179, 0239, 0363, 0384, 0818, 0837, 0841, 0842,
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep Mobile. .1212 sleep noset latency. .0408 sleep parameters. .1004 Sleep problem. .0965 sleep problem. .0965 sleep problems. .1006, 1191 Sleep Quality. .0013, .0145, 0165, 0176, 0179, 0239, 0363, 0384, 0818, 0837, 0841, 0842, .0865, 0965, 0989, 1041, 1056, 1071, 1079, 1115, 1121
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep Inertina. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep Mobile. .1212 sleep myths. .0408 sleep onset latency. .0040 Sleep parameters. .1059 Sleep Physiology. .0111 sleep problem. .0965 sleep problems. .1006, 1191 Sleep Quality. .0013, .0145, 0165, 0176, 0179, 0239, 0363, 0384, 0818, 0837, 0841, 0842, . .0865, 0965, 0989, 1041, 1056, 1071, 1079, 1115, 1121 Sleep quantity. .1086
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep Mobile. .1212 sleep myths. .0408 sleep onset latency. .0040 Sleep Physiology. .0111 sleep problems. .1004 Sleep problems. .0065 sleep problems. .0065 sleep problems. .0013, .0145, 0165, 0176, 0179, 0239, 0363, 0384, 0818, 0837, 0841, 0842, .0865, 0965, 0989, 1041, 1056, 1071, 1079, 1115, 1121 Sleep reactivity. .0465
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep Inertina. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep Mobile. .1212 sleep myths. .0408 sleep onset latency. .0040 Sleep parameters. .1059 Sleep Physiology. .0111 sleep problem. .0965 sleep problems. .1006, 1191 Sleep Quality. .0013, .0145, 0165, 0176, 0179, 0239, 0363, 0384, 0818, 0837, 0841, 0842, . .0865, 0965, 0989, 1041, 1056, 1071, 1079, 1115, 1121 Sleep quantity. .1086
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep Mobile. .1212 sleep myths. .0408 sleep onset latency. .0040 Sleep problems. .1004 Sleep problems. .0065 sleep problems. .0965 sleep problems. .00179, 0239, 0363, 0384, 0818, 0837, 0841, 0842, 0865, 0965, 0989, 1041, 1056, 1071, 1079, 1115, 1121 Sleep quantity. .0465 sleep regularity. .0465
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep Mobile. .1212 sleep myths. .0408 sleep onset latency. .0040 Sleep problem. .0965 sleep problems. .1004 Sleep problems. .00171, 0111 sleep problems. .0065 sleep problems. .0065 sleep problems. .0065 sleep quality. .0013, 0145, 0165, 0176, 0179, 0239, 0363, 0384, 0818, 0837, 0841, 0842, 0865, 0965, 0989, 1041, 1056, 1071, 1079, 1115, 1121 Sleep reactivity. .0465 sleep regularity. .0182, 1007 Sleep regulation. .0859
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep Mobile. .1212 sleep myths. .0408 sleep onset latency. .0040 Sleep problem. .0965 sleep problem. .0965 sleep problems. .1006, 1191 Sleep Quality. .0111 sleep quality. .0013, .0145, 0165, 0176, 0179, 0239, 0363, 0384, 0818, 0837, 0841, 0842, .0865, 0965, 0989, 1041, 1056, 1071, 1079, 1115, 1121 Sleep reactivity. .0465 sleep regularity. .0182, 1007 Sleep regulation. .0859 sleep respiration sounds. .0573
1166, 1187, 1189 sleep health promotion.
1166, 1187, 1189 sleep health promotion. .1168 Sleep homeostasis. .0270 Sleep Hygiene. .0180, 0217, 0544, 0848, 0981, 1166 Sleep in Neurodevelopmental Disorders. .0069 Sleep Inconsistency. .0166 Sleep Inertia. .0078, 0175 sleep Inertia. .0078, 0175 sleep intervention. .0931 sleep loss. .0028, 0171, 0224, 0909 sleep measurement. .0178 Sleep medicine. .0538 Sleep Mobile. .1212 sleep myths. .0408 sleep onset latency. .0040 Sleep problem. .0965 sleep problem. .0965 sleep problems. .1006, 1191 Sleep Quality. .0111 sleep quality. .0013, .0145, 0165, 0176, 0179, 0239, 0363, 0384, 0818, 0837, 0841, 0842, .0865, 0965, 0989, 1041, 1056, 1071, 1079, 1115, 1121 Sleep reactivity. .0465 sleep regularity. .0182, 1007 Sleep regulation. .0859 sleep respiration sounds. .0573
1166, 1187, 1189 sleep health promotion.

Sleep risk groups	
sleep risk groups,	09
sleep screener	77
sleep specialist	40
Sleep Spindle	37
Sleep Spindles	72
sleep stability	50
sleep stage dynamics	
Sleep stage prediction	
sleep staging	
sleep structure	
sleep study process	
sleep technologists	
sleep therapy	
sleep timing	
sleep tracker	
Sleep Traits	
Sleep Valuation	
sleep variability	
sleep wake disorders1174, 11	
Sleep wake scoring	
Sleep, Actigraphy, Metabolic Syndrome, Aging	
sleep-dependent consolidation	
sleep-disordered breathing	
sleep-eating	99
Sleep-Preparatory	11
Sleep-related behaviors	21
Sleep-related cognition	
Sleep-related motor events	
sleep-wake patterns	
Sleepiness	
sleepwalking	
Slow wave	52
Slow wave. .03 Slow wave activity. .0061, 00	52 83
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10	52 83 80
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11	52 83 80 33
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03	52 83 80 33 35
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066	52 83 80 33 35 95
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11	52 83 80 33 35 95 61
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .056	52 83 80 33 35 95 61 60
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000	52 83 80 33 35 95 61 60 05
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .100	52 83 80 33 35 61 60 005 000
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .100 Social Determinants of Health. .10	52 83 80 33 35 61 60 005 56
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .100 Social Jet Lag. .02	52 83 80 33 35 61 60 05 00 56 31
Slow wave. .03 Slow wave activity. .0061, 000 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 033 smartphone application. .066 Smith-Magenis Syndrome. .1004, 114 smoking. .055 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022	52 83 880 33 35 695 61 600 956 31 89
Slow wave. .03 Slow wave activity. .0061, 000 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 033 smartphone application. .066 Smith-Magenis Syndrome. .1004, 114 smoking. .050 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022 social judgment. .022	52 83 80 33 35 695 61 60 005 000 56 31 89 52
Slow wave. .03 Slow wave activity. .0061, 000 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 033 smartphone application. .066 Smith-Magenis Syndrome. .1004, 114 smoking. .050 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022 social Perception. .055	52 83 80 33 35 61 60 05 00 56 31 89 52 49
Slow wave. .03 Slow wave activity. .0061, 000 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 033 smartphone application. .066 Smith-Magenis Syndrome. .1004, 114 smoking. .050 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022 social judgment. .022	52 83 80 33 35 61 60 05 00 56 31 89 52 49
Slow wave. .03 Slow wave activity. .0061, 000 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 033 smartphone application. .066 Smith-Magenis Syndrome. .1004, 114 smoking. .050 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022 social Perception. .055	52 83 80 33 35 61 60 05 61 60 05 61 60 56 31 89 52 49
Slow wave. .03 Slow wave activity. .0061, 000 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 033 smartphone application. .066 Smith-Magenis Syndrome. .1004, 114 smoking. .056 Social. .000 Social Media. .100 Social Jet Lag. .02 social judgment. .02 Social Perception. .057 Social processes. .02	52 83 80 33 35 61 60 56 31 89 52 49 19 52 49 95
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022 social processes. .022 Social relationships. .019 Social Rhythms. .019	52 83 80 33 35 61 60 55 61 60 55 61 60 55 89 52 89 52 89 52 85
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .000 Social Jet Lag. .02 social jetlag. .0191, 02 social perception. .055 social processes. .02 Social Rhythms. .01 Social Rhythms. .01 social support. .03	52 83 80 33 35 95 61 60 05 00 56 31 89 52 49 95 85 77
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .056 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022 social processes. .02 Social Reception. .057 social support. .032 social support. .033	52 83 80 33 35 61 60 50 61 60 50 61 60 50 61 60 50 61 60 55 61 60 55 61 60 55 62 63 1 89 55 63 1 89 55 64 95 64 60 56 65 67 56 77 77 74 95 77 74 95 77 74 95 75 75 75 75 75 75 75 75 75 75 75 75 75
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .100 Social Jet Lag. .022 social judgment. .02 Social Perception. .055 social relationships. .019 Social Support. .032 Social Rhythms. .013 social Support. .033 social Support. .032 Social Cognitive. .022 Social Support. .033 social Support. .033 social Support. .032 Social Rhythms. .017 Social Support. .022 Social Social Support. .024 Social Support. .032 Social Support. .032 Social Support. .032	52 83 80 33 35 61 60 56 31 89 52 49 95 85 77 47
Slow wave. .03 Slow wave activity. .0061, 00 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 10 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .100 Social Jet Lag. .022 social judgment. .02 Social Perception. .055 social relationships. .019 Social support. .032 Social Rhythms. .013 social support. .033 social Fat. .0743, 0745, 07 Soft Palate Fat. .054	52 83 80 33 35 61 60 50 61 60 50 61 60 50 52 49 52 49 55 85 77 47 69
Slow wave. .03 Slow wave activity. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022 social Perception. .055 social relationships. .019 Social Processes. .022 Social relationships. .019 Social support. .033 social support. .033 social-cognitive. .022 Sodium Oxybate. .0743, 0745, 07 Soft Palate Fat. .054 software. .088	52 83 80 33 35 61 60 05 61 60 05 61 60 05 61 89 52 49 52 49 53 85 77 47 69 14 77 47 69 14 75 77 76 77 76 77 76 77 77 77 77
Slow wave. .03 Slow wave activity. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022 social processes. .02 Social relationships. .019 Social relationships. .019 Social support. .032 Social-cognitive. .022 Social-cognitive. .022 Social relationships. .019 Social support. .022 Sodium Oxybate. .0743, 0745, 07 Soft Pal	52 83 80 33 35 61 60 05 61 60 05 61 89 52 49 52 49 52 49 52 49 52 49 52 49 53 52 49 53 52 49 53 53 53 54 55 55 56 56 56 56 56 56 56 56
Slow wave. .03 Slow wave activity. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022 social processes. .02 Social relationships. .019 Social relationships. .019 Social support. .032 Social relationships. .019 Social suport. .032 Sodium	52 83 80 33 35 61 60 56 31 89 52 49 95 85 77 47 47 69 47 34 78
Slow wave. .03 Slow wave activity. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .000 Social Jet Lag. .022 social jetlag. .0191, 022 social processes. .02 Social relationships. .019 Social Rhythms. .019 social support. .033 software. .0743, 0745, 07 software. .08 Sound masking. .07 spatial performance task. .02	52 833 335 61 605 605 605 605 605 52 495 852 477 477 477 694 134 787 378 3
Slow wave03Slow wave activity0015, 0090, 0108, 0838, 0869, 100Slow Waves11slow-wave activity0321, 033smartphone application066Smith-Magenis Syndrome1004, 110smoking055Social000Social Media100Social Jet Lag022social jetlag0191, 022social processes02Social Perception055social relationships015Social Rhythms01743, 0745, 07Soft Palate Fat057software08Sound masking077spacifight02social performance task02specialized pro-resolving mediators02specialized pro-resolving mediators02specialized pro-resolving mediators02	52 83 33 35 61 600 56 319 522 499 552 477 477 477 477 477 477 477 477 527 477 527
Slow wave	52 83 33 35 61 600 56 319 529 529 610 560 561 605 561 605 561 605 561 605 562 495 577 477 694 477 694 477 694 477 694 477 505 605
Slow wave. .03 Slow wave activity. .0015, 0090, 0108, 0838, 0869, 100 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .111 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .000 Social Determinants of Health. .100 Social jet Lag. .022 social jetlag. .0191, 022 social jetlag. .0191, 022 social processes. .02 Social Perception. .055 social processes. .02 Social Rhythms. .017 social support. .033 social-cognitive. .022 Software. .0743, 0745, 07 Soft Palate Fat. .055 software. .022 spaciflight. .022 spatial performance task. .022 spectral analysis. .057 Spectrum. .000	52 83 335 61 600 56 319 529 610 560 560 561 319 529 852 477 477 477 477 547 477 54
Slow wave. .03 Slow wave activity. .0061, 000 slow wave sleep. .0015, 0090, 0108, 0838, 0869, 100 Slow Waves. .11 slow-wave activity. .0321, 03 smartphone application. .066 Smith-Magenis Syndrome. .1004, 11 smoking. .055 Social. .000 Social Media. .100 Social Jet Lag. .022 social jetlag. .0191, 022 social processes. .02 Social Perception. .055 social support. .03 social support. .03 social support. .03 social support. .03 software. .0743, 0745, 07 software. .08 Sound masking. .07 specialized pro-resolving mediators. .02 Spectral analysis. .05	52 83 335 61 605 315 526 315 526 315 526 315 527 5

Spinal cord injury or disease
Spindle
Spindles
Split night polysomnography
Spontaneous
sports
standardization
startle reactivity
Stem Cells
Stimulant
stimulus control
STOP BANG
STOP-BANG questionnaire
street-based female sex workers
stress0006, 0149, 0162, 0178, 0181, 0194,
0212, 0225, 0229, 0252, 0256, 0265, 0265, 0276, 0316, 0338, 0345,
0365, 0378, 0407, 0454, 0455, 0456, 0465, 0554, 0988, 1047, 1049,
1101, 1203
stress generation hypothesis
stress reactivity
Stressors
stroke
structural vulnerability
Students
Subjective Cognitive Decline
subjective health
subjective Sleep Quality
Substance Use
Subtyping
Suicide
Sun exposure
surgery
Surgical Malfunction
surgical outcome
sustained attention
Swimmers
synaptic plasticity
synaptic plasticity
System Consolidation
5ystem Consontation

Т

tapering
taste dysfunction
Tauopathy
ТВМ
technology
Technology Use
Teenage
Telehealth
telemedicine
telephone CBT-I
telomere length
temperament
Text message reminders
Thalamocortical network
thermoplastic
thermoregulation
Theta power
tics
Time on task
Time Restricted Feeding

time use
Timing
tnf reverse signaling
Topical nasal steroid
Total Sleep Deprivation
total sleep time
tourette's disorder
Tracheobronchomalacia
trajectory
transcranial magnitic stimulation
Transcriptomics
transgender
Transgender women of color
translation
translational science0001, 0127, 0162, 0220, 0277, 0545, 1029
transoral robotic surgery
trauma associated sleep disorder
trauma related nightmare
traumatic brain injury 0046, 0169, 0227, 0416, 1118, 1124, 1129,
1133
travel
Trazodone
Treatment
treatment adherence
treatment-emergent
Triprolodine
Trisomy
Troubleshooting Clinic
Tumor necrosis factor alpha
Tunable LED
Type1 diabetes
Type 2 diabetes

U

U.S. military
U.S. veterans
1069, 1074, 1106, 1143, 1156, 1157, 1180, 1194
ultra-long range
Ultrasound
Uncontrolled hypertension
under diagnoses
underserved populations
1179, 1181
University
UPLC-MS/MS
Upper Airway Anatomical Structures
Upper Airway Anatomy
upper airway mechanical property
upper airway muscle
upper airway muscle fatigue
upper airway resistance syndrome
upper airway stimulation 0628, 0650, 0665, 0674, 0676, 0681, 0690,
0691, 0706
upper airway stimulation (UAS)
UPSIT
Uptake
user journey from big data
\mathbf{V}

W

Waking
watchpat
Wearable
wearable technology
Wearables
Web Based Training
web-based intervention
weight loss
Weighted Blanket
Well-being0776, 0837

women0015, 0020, 0342, 0343, 0362, 0366, 0399, 0467, 0470,
0499, 0534, 0595, 0819, 0830, 0831, 0853, 0868, 0948, 1011, 1016,
1036, 1045, 1090
women's health
women's sleep
women/children
Work Stress
workers
workforce
working memory
workload
workplace yoga program0778
Worry
wrist actigraphy 0037, 0123, 0130, 0380, 0468, 0927, 0985, 1011,
1196

X

xgboost				
---------	--	--	--	--

Y

young adult	
young adults	
Young drivers	
Youth	

Z

zolpidem.							0094
-----------	--	--	--	--	--	--	------