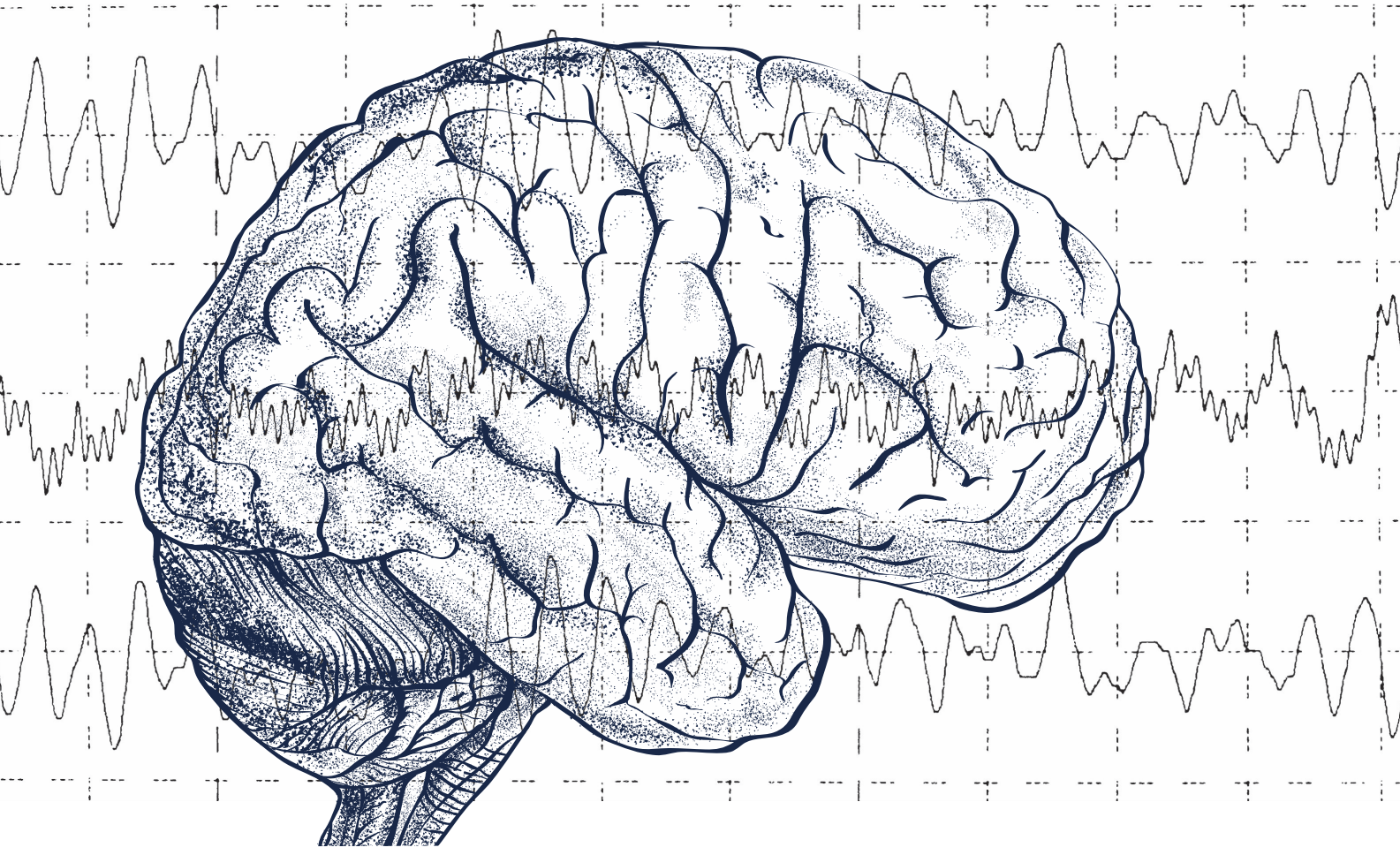
SLEEP

Official Publication of the Sleep Research Society

VOLUME 40, 2017 | ABSTRACT SUPPLEMENT



Sleep Research Society®

SLEEP 2017 BOSTON • JUNE 3-7

31st Anniversary Meeting of the Associated Professional Sleep Societies, LLC

OXFORD UNIVERSITY PRESS

SLEEP

JOURNAL OF SLEEP AND SLEEP DISORDERS RESEARCH

Volume 40 Supplement 1 | April 30, 2017 | Pages 1–514

Official publication of the Sleep Research Society.

Welcome to your preview of SLEEP 2017, the 31st Anniversary Meeting of the Associated Professional Sleep Societies, which will be held in Boston, Massachusetts on June 3-7, 2017.

This abstract supplement unites the journal *SLEEP*, and the science of SLEEP 2017. All abstracts presented at SLEEP 2017 are included in this special issue. This year, 1,209 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 clinical sleep science and then assigned to one of 18 subcategories. Each abstract has a unique four-digit number to facilitate identification and location both within this issue and at SLEEP 2017. The four-digit number in the abstract supplement matches the four-digit code published in the SLEEP 2017 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. We look forward to sharing in the success of this pivotal event and hope you consider joining the American Academy of Sleep Medicine and Sleep Research Society in Boston, Massachusetts in June.

Ronald Szymusiak, PhD
Editor-in-Chief

Table of Contents

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

0040

CIRCADIAN PHASE PREFERENCE, SLEEP PATTERNS, AND MENSTRUAL CYCLE LENGTH IN FIRST-YEAR UNIVERSITY STUDENTS: PRELIMINARY RESULTS

Van Reen E[1], Ernst A[3], Axelrod K[4], Sharkey KM[1], Kiesner J[5], Carskadon MA[1,2]

[1]Alpert Medical School of Brown University, Providence, RI, 2Sleep for Science Laboratory of Brown University, Providence, RI, [3]E.P. Bradley Hospital, Providence, RI, [4]Brown University, Providence, RI, [5]Università di Padova, Padova, ITALY, [6]Alpert Medical School of Brown University, Providence, RI, [7]University of South Australia, Adelaide, AUSTRALIA

Introduction: Circadian disruption and short sleep are associated with fertility problems in women. Menstrual cycle lengths shorter than 25 days or longer than 30 days are more likely to be anovulatory (infertile menstrual cycle). We examined whether 1) sleep patterns and 2) the congruence between circadian phase preference and sleep patterns are associated with menstrual cycle length in first-year university students.

Methods: Women (n= 206, mean age=18.6; SD= 0.5 y) completed on-line sleep diaries for 9 weeks. Each diary included sleep times and menstrual bleeding (yes/no). Menstrual cycle length (MCL) was the interval from the first day of menstrual bleeding to the next first day of menstrual bleeding. Sleep pattern variables derived for each woman across each menstrual cycle included: mean and standard deviation of reported bedtime (BT), wake time (WT), and total sleep time (TST). Circadian phase preference was determined from the Horne Östberg questionnaire (MEQ) completed in week 9 using 5 standard categories. A sleep timing vs. circadian phase preference "mismatch" score was calculated using the absolute difference between WT categories based on quintile split and MEQ categories (possible scores ranged from 0–4). Linear mixed-effect models were used to examine 1) the effects of sleep patterns (i.e., mean and SD of WT, BT, TST) on MCL (278 cycles) and 2) the effects of phase preference vs. WT mismatch on MCL in a subset of 188 women (256 cycles). Each woman contributed between 1–3 menstrual cycles.

Results: Overall, average MCL was 27 days (sd=7.1). A significant association between greater mismatch and shorter menstrual cycle length was found (b=-1.4, se=.57, p=.02). No significant associations were observed between sleep patterns and menstrual cycle length; however, a trend was seen for WT (b=.5, se=.48, p=.09) and BT (b=.75,.45, p=.1) in both of which later timing was associated with shorter MCL.

Conclusion: Sleep patterns alone were not significantly associated with MCL; however, shorter MCL was associated with a poor match between circadian phase preference and wake timing. The mismatch between circadian phase preference and sleep timing highlights a potential mechanism for fertility issues in shift workers.

Support (If Any): NIMH MH079179 (to MAC).

0041

PREDICTING TRAIT-LIKE VARIABILITY IN THE COEFFICIENTS OF THE TWO PROCESS MODEL OF PERFORMANCE

Payne-Rogers C, Kenyon M, Jones J

CurAegis Technologies, Inc., Rochester, NY

Introduction: Biomathematical models of fatigue are often used to predict alertness degradation as a function of prior sleep-wake history. There are large inter-individual differences in the magnitude of an individual's degradation in response to sleep loss which, if unaccounted for, can lead to significant inaccuracies in model performance. It has been shown that these differences are stable within the individual across variations in sleep history. Current methods of individualization require periodic performance measurement feedback from the user. Here we have developed a method for estimating these stable, trait-like inter-individual differences based solely upon a priori, ambulatory data.

Methods: Seven days of biometric and motion sensor data were collected from 16 subjects under restricted, ambulatory conditions prior to controlled, in-lab collection of performance data across a 28-hour sleep deprivation period. A parameterized two process model of fatigue was utilized to simulate performance degradation under sleep loss. The model coefficients were optimized such that they accounted for inter-individual differences in performance degradation. A proprietary, machine-learning-based algorithm was trained, using 12 of the ambulatory datasets, to predict the optimized coefficients for each individual. The effectiveness of the algorithm was tested on the remaining four datasets.

Results: The maximum percent error in the test set of each of the estimated coefficients was 3.12, 48.55, 33.29, and 18.44, respectively. Conversely, the percent error of a group-aggregate coefficient compared across the optimized coefficients of all subjects was found to be 7.84 ± 7.56, 209.72 ± 193.78, 163.22 ± 162.04, and 124.33 ± 107.04, respectively.

Conclusion: The coefficient estimation algorithm developed is novel, in that it is capable of approximating an individual's trait-like vulnerability in the coefficients of a parameterized two process model of fatigue using only a priori, ambulatory data and has shown significant potential in improving model performance.

Support (If Any): CurAegis Technologies, Inc.

0042

PERFORMANCE OF ADHESIVE WIRELESS PATCH SENSOR FOR SCREENING OF SLEEP ARCHITECTURE IN NORMAL AND APNEA

Selvaraj N

Vital Connect Inc, Campbell, CA

Introduction: Sleep is a renowned marker of health. One in three adults endure sleep disorders with out diagnosis due to lack of effective sleep screening technology. The study presents clinical validation of VitalPatch®, a wireless adhesive medical device for screening of sleep architecture in normal and apnea compared to the Polysomnography (PSG).

Methods: 45 volunteers (male/female: 24/21; 42 ± 13 years) were recruited for an overnight PSG study, and attached to 22-channel PSG and a VitalPatch sensor on chest. Simultaneous PSG and patch data were acquired wirelessly during overnight. PSG recordings were scored to obtain 5-stage sleep architecture and apnea-hypopnea index (AHI) per AASM guidelines. Based on PSG's AHI values, the study population was grouped into normal (28), mild apnea (10) and moderate apnea (7). The statistical differences in sleep patterns among 3 groups were assessed using PSG sleep metrics. 3-class hypnograms with wake, non-rapid eye movement (NREM) and REM stages were further derived using VitalPatch recordings and calculated their respective sleep metrics. Performance analyses of VitalPatch's sleep assessment were carried out compared to the PSG.

Results: PSG revealed significant decrease in total REM time (P=0.032) and increase in latency to REM (P=0.005) in apnea than normal. Latency to NREM and wake after sleep onset (WASO) were increased in apnea (P=0.063 and P=0.072, respectively). The accuracy and Cohen's kappa of sleep stage prediction using VitalPatch compared to the PSG were (82.4 ± 8.2, 79.2 ± 4.3 and 72.2 ± 10.0 in %) and (0.57 ± 0.16, 0.54 ± 0.09 and 0.39 ± 0.25), respectively in

normal, mild apnea and moderate apnea groups. VitalPatch's total sleep time, total NREM time and total REM time had highest correlation (R) of 0.93, 0.86 and 0.72 with PSG respectively in normal and relatively lower in apnea groups. WASO was highly correlated to PSG in moderate apnea (0.67) compared to normal (0.47), as the number awakenings and wake duration after sleep onset were higher in apnea than normal.
Conclusion: The study validates good performance of adhesive wireless VitalPatch sensor for sleep staging in normal and apnea compared to the PSG. The unobtrusive disposable patch sensor can be valuable for widespread clinical screening of sleep architecture.
Support (If Any): None.

0043

INVESTIGATION OF THE DEVELOPMENTAL ORIGIN OF FOREBRAIN GABAERGIC NEURONS INVOLVED IN SLEEP-WAKE CONTROL USING A FATE-MAPPING APPROACH

Brown RE[1,2], Yang C[2], Winston S[2], Anderson-Chernisof M[2], McNally JM[2], McKenna JT[2]
[1]VA BHS/Harvard Medical school, Brockton, MA, [2]VA Boston Healthcare System and Harvard Medical School, West Roxbury, MA

Introduction: GABAergic neurons located in the basal forebrain (BF) play important roles in promoting wakefulness and cortical activation. The developmental origin of these neurons is unknown. Here we use a fate-mapping approach to investigate BF GABAergic neurons derived from the medial ganglionic eminence (MGE) which express the transcription factor, Lim homeobox 6 (Lhx6).
Methods: Previously validated mice expressing Cre Recombinase (Cre) under the control of the Lhx6 promoter region were purchased from Jackson Laboratories (Bar Harbor, ME). A cross with a Cre-reporter strain expressing the red fluorescent protein, tdTomato, allowed us to investigate the location of MGE-derived neurons. Immunostaining was used to identify parvalbumin neurons. Adeno-associated viral vectors expressing excitatory receptors (hM3Dq) activated exclusively by the designer drug, clozapine-N-oxide (CNO), were injected bilaterally into BF to test the effect on sleep-wake states.
Results: Neurons specified by Lhx6 were widely distributed throughout the BF. They included BF parvalbumin-containing projection neurons involved in promotion of wakefulness and cortical gamma-band oscillations. 575/1071 PV neurons counted in the BF of one Lhx6-Cre-tdTomato mouse contained tdTomato. However, only 13.7 % of tdTomato neurons were PV+, suggesting that Lhx6 also specifies other types of BF GABA neurons. Chemogenetic activation of Lhx6-derived BF neurons strongly increased wakefulness and gamma band power for >1 hr after i.p. injection of CNO (0.3 mg/kg) at ZT2. CNO treated mice had 83 ± 3 % wakefulness whereas saline-treated mice had only 30 ± 1 % (n=2) in the period 20–80 min following injection. Saline-injected mice also had increased wakefulness but only in the 20 min immediately following the injection.
Conclusion: Wakefulness-promoting BF GABAergic neurons are derived from MGE progenitor cells expressing Lhx6. Cortical interneurons implicated in schizophrenia and other neurodevelopmental disorders are derived from the same pathway suggesting a potential involvement of BF Lhx6-derived neurons in the sleep-wake disturbances observed in these disorders.
Support (If Any): This work was supported by the US Veterans Administration, VA CDA 1IK2BX002130 (JMM) and NIH: NINDS R21 NS093000 (REB) & NIMH R03 MH107650 (CY). JTM received partial salary compensation and funding from Merck MISP, but has no conflict of interest with this work.

0044

DIFFERENTIAL EFFECTS OF PARADOXICAL SLEEP DEPRIVATION ON ADOLESCENT AND ADULT MICE

Tuan L[1], Lee L[1]
[1]Graduate Institute of Anatomy and Cell Biology, National Taiwan University, Taipei, TAIWAN, [2]Graduate Institute of Anatomy and Cell Biology, National Taiwan University, Taipei, TAIWAN

Introduction: Sleep insufficiency has become a serious health issue. In the modern society, most of the people, including the adolescence, do not obtain enough sleep. Since adolescence is a critical period for brain development, the consequences of insufficient sleep during adolescence should be concerned. The current study emphasized the effects of 72-hour paradoxical sleep deprivation (SD) on behavioral and morphological aspects in adolescent mice and used adult mice for comparison.
Methods: In this study, we examined the acute effects of 72-hour paradoxical SD. 5 weeks old and 10–12 weeks old male C57/BL6 mice were used. The two time points were chosen to represent the periods of adolescence and adulthood, respectively. SD for 72 hours were conducted using modified multiple platform method. For mice kept in the home cage and on big platforms, sleep time was not limited and used as controls. Mice of SD and control groups were examined in behavioral, neurochemical and histological aspects.
Results: Our results showed that the short-term spatial memory, examined by Y-maze spontaneous alternation test, was affected by 72-hour paradoxical SD in adolescent mice but not in adult mice. The complexity of granule cells in dentate gyrus (DG) was reduced after SD in adolescent but minimal changes were observed in adult animals. There was an increase of spine density in DG granule cell after 72-hour paradoxical SD in adolescent and not in adult SD animals. Hippocampal neurogenesis in the DG was reduced by SD in both adolescent and adult groups.
Conclusion: Results from this study revealed that SD negatively impacted the cognitive function by impairing the spatial working memory in adolescent but not in adult mice. Moreover, the analyses of dendritic complexity and spine density of granule cells in the hippocampal DG also indicated age-related morphological alterations after 72-hour sleep deprivation. Our results indicated the adolescent mice are relatively more sensitive to SD than the adult mice. It is the first study, to our knowledge, that compared the differential effects of SD on adolescent and adult mice.
Support (If Any): None.

0045

A ROLE FOR EARLY LIFE REMS IN COGNITIVE DEVELOPMENT IN RATS

Shaffery J[1], Morris S[1], Blackwell A[1], Knowles A[1], Tillman M[1]
[1]University of Mississippi Medical Center, Jackson, MS, [2]University of Mississippi Medical Center, Jackson, MS, [3]University of Mississippi Medical Center, Jackson, MS, [4]University of Mississippi Medical Center, Jackson, MS, [5]University of Mississippi Medical Center, Jackson, MS

Introduction: In the young, rapid eye movement sleep (REMS) is initially more highly represented in daily sleep/wake cycles than later in life and is thought to facilitate brain maturation. We have shown that early life REMS disturbances (i.e., ERD) have relatively long-lasting, negative effects on hippocampal synaptic plasticity, including reductions in expression of several glutamate signaling proteins and in long- term potentiation (LTP) stability. Our previous results led us