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0217

IS THE TIMING OF THE ENDOGENOUS CIRCADIAN RHYTHM OF NEUROBEHAVIORAL FUNCTIONING INHERENTLY TASK-DEPENDENT?

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Introduction: Changes in waking neurobehavioral functioning (NF) over time are governed by homeostatic and circadian processes. It has been reported that peak circadian timing varies inherently between tasks, such that the optimal timing for NF would be task-dependent. Here we investigated this idea with a simulated shift work study protocol followed by a 24h constant routine (CR) protocol to experimentally and statistically separate the circadian from the homeostatic process.
Methods: N=13 healthy adults (ages 25.5±3.2y; 9 men) completed a 7-day/6-night in-laboratory study. They were randomized to a 3-day simulated day shift condition (n=7) with nighttime sleep (22:00–06:00) or a 3-day simulated night shift condition (n=6) with daytime sleep (10:00–18:00). They then underwent a 24h CR protocol, during which they stayed awake under constant behavioral and environmental conditions and blood was collected at 1–3h intervals for the assessment of dim light melatonin onset (DLMO). During scheduled wakefulness, subjects completed three functionally distinct NF tasks at ~2h intervals: the Karolinska Sleepiness Scale (KSS), Digit Symbol Substitution Test (DSST), and Psychomotor Vigilance Test (PVT). Data from these tasks taken during the CR protocol were analyzed with non-linear mixed-effects regression to separate endogenous circadian effects from the homeostatic process.
Results: Following simulated night shift, compared to simulated day shift, on average there was a modest, 1.4h (±0.8h SE) delay in DLMO ($F_{1,11}=3.68$, $p=0.082$). As such, the simulated night shift condition produced a 10.6h shift in alignment of the homeostatic process relative to the circadian process. Regardless of prior shift condition, the peak of the circadian rhythm effect on NF occurred post-DLMO by 16.8h (±1.0h) for KSS, 15.9h (±1.4h) for DSST, and 18.6h (±1.0h) for PVT, which was not significantly different ($F_{2,9}=1.55$, $p=0.26$).
Conclusion: As proof of principle, we studied three distinct NF assays, and found only small, non-significant differences between them in the timing of underlying circadian rhythmicity. While a larger sample could have yielded statistical significance in our comparison of circadian peak times, the small magnitude of the observed difference does not support the idea of inherent task-dependent differences in the timing of the endogenous circadian rhythm's influence on NF.
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0218

N2 AND WAKEFULNESS DRIVE SUBJECTIVE SLEEP SATISFACTION IN ADULTS

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Introduction: The measurable aspects of brain function derivable from polysomnography (PSG) that are correlated with sleep satisfaction are poorly understood. Previously, a weak association of PSG measures with subjectively rated sleep depth and restfulness was shown. Using recent developments in automated sleep scoring, which remove the within- and between-rater error associated with human scoring, we revisit whether whole night PSG measurements are associated with sleep satisfaction. Additionally, we investigate if PSG data collected closer to wake time explains the subjective sleep experience better than whole night PSG.
Methods: Random Forest machine learning was used to investigate the relationship between PSG data from the Sleep Heart Health Study (N=3,165, middle-aged and older adults) and self-reported sleep satisfaction (restfulness, depth). PSG were rescored using a novel automated algorithm that generates both a sleep stage for each 15-s epoch as well as a stage matching probability. Data were also parsed into 20 minute-fragments based on time relative to wake.
Results: Models explained 30% of subjective sleep depth and 27% of subjective sleep restfulness, with a similar top four predictors: minutes of N2 and wake after sleep onset (WASO), sleep efficiency, and age, capturing 28% (restfulness) and 26% (depth) of the relative model variance. With increasing subjective sleep quality, there was a progressive increase in N2 and decrease in WASO of similar magnitude, without systematic changes in N1, N3 or REM. In comparing those with the best and worst subjective experience of sleep, there is a range of approximately 30 minutes more N2, 30 minutes less WASO, an improvement of sleep efficiency of 7-8%, and an age span of 3-5 years. Random Forest models derived from PSG fragments closer to the offset of sleep did not provide better explanatory power compared to the whole-night data set.
Conclusion: Approximately one-third of the variance in two measures of self-reported sleep experience can be explained by whole-night PSG variables, notably an increase in N2 and decrease in wake that led to improved sleep efficiency. Interventions that specifically target these may be suitable for improving the self-reported sleep experience.
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0219

INTERPLAY OF SCHOOL DAYS AND FREE DAYS WITH SLEEP MIDPOINT ON THE ASSOCIATION OF VISCERAL ADIPOSITY WITH BLOOD PRESSURE IN ADOLESCENTS

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Introduction: The circadian timing of sleep, including its variability, has emerged as an important contributor to obesity and cardiovascular health, such as elevated blood pressure. Adolescence is a particularly vulnerable period for circadian misalignment, which may express differently if youth are in school or on free-days. We examined whether deviations in sleep midpoint increase the impact of visceral adiposity on elevated blood pressure in adolescents as a function of being entrained to school or not.

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