Besides, linear models were also employed to study the influence of type-of-day, training shift, alarms, log10 of light exposure and minutes of MVPA, on the sleep patterns.
Results: Higher morning or evening light exposure and minutes of MVPA were found in sync with training shift and training days, with significant interactions (all p<0.001). Alarm usage was also prevalent in the morning shift and in training days, with a significant interaction (p<0.001). Sleep onset delays were associated with attending the night shift (b=1.1±0.3h, p<0.001), free days (b=1.9±0.2h, p<0.001), night light exposure (standardized b=0.2±0.1h, p=0.007), and night MVPA (standardized b=0.3±0.1h, p<0.001), whereas standardized morning light exposure was associated with 0.3±0.1h onset advance (p<0.001). Sleep end delays were associated with attending the night shift (b=1.2±0.4h, p=0.003), free days (b=1.1±0.1h, p<0.001), and night light exposure (standardized b=0.2±0.1h, p=0.003). Using alarms advanced the sleep end in 1.8±0.1h (p<0.001). Sleep duration reductions were associated with free days (b=-0.7±0.2h, p<0.001), alarms (b=-1.7±0.2h, p<0.001), and night MVPA (standardized b=-0.2±0.1h, p=0.003), whereas 0.7±0.2h increase in duration was associated with standardized morning light exposure (p=0.043).
Conclusions: School shifts are a good model for studying the influence of social, environmental and behavioral factors on sleep in young people. Although social influences are stronger in sleep pattern regulation, behavioral interventions can aid in sleep health directly or through regulating exposure to environmental factors such as light.
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THE INTEGRATION OF SKIN AND CORE BODY TEMPERATURE IN THE EXPRESSION OF REM SLEEP AND THE ROLE OF THE HYPOTHALAMUS

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Rapid eye movement (REM) sleep is characterized by suppression of thermoregulatory defence and is preferentially expressed over non-REM (NREM) sleep during thermoneutral ambient temperature (Ta) warming. The lateral hypothalamus (LH) integrates diverse inputs related to temperature, energy balance and circadian time to modulate sleep-wake expression. Although hypocretin (Hcrt) neurons promote wakefulness, melanin-concentrating hormone (MCH) neurons increase REM sleep during thermoneutral Ta warming. However, how the LH controls NREM-REM sleep cycling or what types of temperature information are integrated to modulate REM sleep remain unknown. We hypothesize that skin (TSkin) and core body (TCore) temperature information are integrated in the hypothalamus where the MCH and Hcrt systems drive REM sleep and wakefulness, respectively. During thermoneutral warm Ta pulsing presented during the inactive (light) phase, we show that wild type (WT) mice significantly increased total REM sleep duration and bout number with reduced inter-REM intervals. However, MCH receptor1 knock-out (MCHR1KO) mice showed no effect in vigilance state, and Hcrt-KO mice showed increased wakefulness and decreased REM sleep without alterations in inter-REM intervals. All groups demonstrated an intact homeostatic regulation of REM sleep. These data suggest that both the MCH and Hcrt systems are required for the dynamic modulation of REM sleep as a function of Ta, but that neither system is necessary for REM sleep homeostasis. We then further investigated brain temperature (TBrain), TCore and TSkin parameters in a subgroup of mice to investigate which parameters may be integrated within the LH to modulate REM sleep. WT and MCHR1KO mice showed clear ultradian TCore cycling during the inactive sleep phase where REM sleep was preferentially nested during TCore nadirs. Hcrt-KO mice, in contrast, showed disrupted ultradian TCore cycling. Moreover, WT mice dynamically increased REM sleep expression across a wider distribution of the TCore cycle as a function of integrated TCore-TSkin, whereas MCHR1KO mice failed to modulate REM sleep timing or duration within TCore cycles. These findings suggest that WT mice integrate TSkin and TCore for dynamic REM sleep expression where the MCH system plays a critical role, whereas an intact Hcrt system impacts sleep-wake transitions and the structure of the ultradian rhythm.

THE TOPOGRAPHY OF THALAMIC STROKE HAS AN IMPACT ON ASSOCIATED SLOW WAVE SLEEP AND COGNITIVE CHANGES

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Background and Aim: The thalamus plays an important role in controlling sleep-wake and memory functions. However, the contribution of discrete thalamic regions to these functions remains unclear. Here we postulate that sleep and cognitive changes in patients with isolated thalamic stroke may depend upon the extent of the underlying lesion.
Patients and methods: We included 15 patients with acute isolated (>80% of the total lesion size) thalamic stroke: 8 patients with anteromedial thalamic stroke and 7 patients with lateral thalamic stroke. We assessed stroke characteristics and within the first 5 days post-stroke, nocturnal sleep with high-density electroencephalography (HD-EEG) and respiratory polygraphy. In the evening before the HD-EEG study, subjective sleepiness (Epworth Sleepiness Scale (ESS)), language (Bernese Word Finding Test), neglect (Bells test), verbal memory (Digit span test forward and backward, Hopkins Verbal Learning Test) and visual memory (Corsi block tapping test) were assessed.
Results: There were no differences in age, sex, stroke severity (National Stroke Severity Scale Score at admission and modified Rankin Scale Score at discharge) and cardiovascular risk-related comorbidities between patients with anteromedial and lateral thalamic stroke. Total sleep time (355.14±90.63 minutes and 306.42±91.48 minutes, p=0.383) and sleep efficiency (69.65±14.40 % versus 66.05±17.00 %, p=0.620) were also similar. However, patients with anteromedial thalamic stroke had shorter duration of NREM3 sleep (% of total sleep time: 15.31±7.54 % versus 28.42±7.60 %, p=0.011) and a higher ESS score (7.29±2.69 points versus 4.00±2.00 points, p=0.037). ESS score showed a negative correlation with the duration of NREM3 sleep (Spearman correlation: r=-0.90, p<0.01) which remained significant after controlling for topography of the thalamic lesion (partial Spearman correlation: r=-0.65, p=0.02).
Patients with anteromedial, but not those with lateral, thalamic stroke exhibited confabulations in the HVLT (total number of confabulations: 2.62±3.02 confabulations, p=0.020). The total number of confabulations in HVLT did not correlate with ESS score or with the duration of NREM3 sleep. There were no other cognitive differences between the two groups.
Conclusion: These findings support previous human and experimental observations suggesting a role of the anteromedial thalamus in the regulation of slow-wave sleep and cognition. Further analyses are planned to assess the potential correlation between slow-wave sleep changes, excessive daytime sleepiness and cognitive dysfunction in patients with thalamic stroke.

TO SLEEP, PERCHANCE TO BREATHE: INVESTIGATING THE IMPACT OF OBSTRUCTIVE SLEEP APNEA ON SLEEP NEUROPHYSIOLOGY AND SLEEP-DEPENDENT MEMORY ACROSS BRAIN STATES IN OLDER ADULTS

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Introduction: Obstructive sleep apnea (OSA) symptomatology in older adults is associated with multifarious health risks, including disruption of