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LOCAL SLOW WAVE SLEEP AND POST-STROKE BRAIN REPAIR

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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.
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RELATIONSHIP BETWEEN SLEEP METRICS WITH FREE-LIVING GLUCOSE CONCENTRATIONS AND GLYCEMIC VARIABILITY IN NON-DIABETIC ADULTS

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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/m^2) 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≤r≤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.
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MILD UPPER AIRWAY OBSTRUCTION LEADS TO INCREASED ENERGY INTAKE AND GROWTH RETARDATION THAT PERSISTS AFTER THE OBSTRUCTION REMOVAL

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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