sleep architecture and impairment of cognitive functions. Less is known, however, of how OSA severity impacts the local expression of neural oscillations during sleep relevant for cognition. Here, we demonstrate frequency-specific, deleterious impacts of OSA severity on spectral power in memory-relevant oscillatory frequencies within both non-rapid eye movement (NREM) and REM sleep, using high-density electroencephalography (hdEEG) to delineate findings with enhanced spatiospectral specificity.

Methods: 57 cognitively unimpaired older adults (61.25±6.24 years, 38 female) underwent overnight polysomnography with 256-channel hdEEG. EEG data were cleaned, segmented into concatenated NREM and REM epochs, and spectrally analyzed using the multitaper method. OSA-related measures, i.e., apnea-hypopnea index(AHI), respiratory disturbance index (RDI), and oxygen desaturation index (ODI) were obtained for REM and NREM sleep and were log-transformed to normality. Memory function was assessed using proportion of overnight change in recall performance on a sleep-dependent word paired-associates task (WPT, cube-root transformed to normality). Pearson's correlations with threshold-free cluster enhancement (TFCE, 5000 permutations) were run to identify topographical associations between OSA severity and absolute EEG spectral power. After averaging across spatially segregated clusters of significant associations, multiple regressions adjusting for age and sex were leveraged to model underlying OSA-local sleep relationships. To elucidate the functional significance of these associations, cluster averages of spectral power significantly predicted by OSA-related metrics were then used to predict WPT memory performance in regression models adjusting for sex and age.

Results: Within NREM epochs, TFCE-significant negative topographical correlations were seen between $\log(ODI)_{NREM}$ and slow sigma power (SSP,11-13Hz; global associations), $\log(ODI)_{NREM}$ and fast sigma power (FSP,13-16Hz; frontal and central-parietal clusters), $\log(AHI)_{NREM}$ and SSP (central-parietal cluster), $\log(RDI)_{NREM}$ and theta power (4.5-7.5Hz; frontal and parietal clusters), $\log(RDI)_{NREM}$ and alpha power (7.5-11Hz; frontal and central-parietal clusters), and between $\log(RDI)_{NREM}$ and SSP (central cluster). Analyzing REM epochs revealed clusters of TFCE-significant negative associations between $\log(RDI)_{REM}$ and theta power (frontal cluster), $\log(RDI)_{REM}$ and gamma (28-40Hz; parietal-occipital cluster), and log(RDI) and high gamma (40-55Hz; frontal and parietal clusters). Subsequent regression models using segregated cluster averages demonstrated significant influences of $\log(RDI)_{NREM}$ on central SSP (B=-0.162, p=0.033), of $\log(ODI)_{NREM}$ on global SSP (B=-0.188, p=0.026), frontal FSP (B=-0.081, p=0.034), and central-parietal FSP (B = -0.132, p=0.029), and of $\log(RDI)_{REM}$ on parietal gamma power(B=-0.016, p=0.018), frontal high gamma power (B=-0.015, p=0.002), and parietal high gamma power (B=-0.011, p=0.002). Finally, WPT performance was significantly predicted by cluster averages of central FSP (B=0.288, p=0.011), frontal FSP (B=0.385, p=0.033), global SSP (B=0.216, p=0.007), central-parietal SSP (B=0.230, p=0.008), and parietal gamma power (B=1.639, p=0.048), with parietal high gamma power (B = 2.853, p=0.064) exhibiting similar trends.

Conclusions: We demonstrate widespread, significant impacts of OSA severity on frontal and central-parietal expression of NREM and REM sleep, with deficits in NREM sigma activity and REM gamma activity disrupting sleep-dependent memory. Further research may examine if OSA treatment could reverse cognitive impairment in older adults with OSA, through its impact on local oscillatory activity.

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TOTAL SLEEP DEPRIVATION LEADS TO CHANGES IN NEUROMUSCULAR JUNCTION OF SOLEUS MUSCLE IN MALE WISTAR RATS

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Introduction: Brain is the biggest beneficiary of sleep. Skeletal muscle atonia is a prominent feature of rapid eye movement (REM) sleep. Neuromuscular junction (NMJ) exhibits high morphological and functional plasticity (Arnold et al., 2014).Functional importance of skeletal muscle atonia is not known. Sleep deprivation may influence NMJ morphology at presynaptic and postsynaptic terminals levels. In the present study, we have looked into the ultrastructural changes in the rat soleus muscle NMJ after sleep , sleep deprivation and recovery sleep.

Materials and Methods: Total18 rats were divided into three groups. Group I rats had normal sleep wake cycle, Group II rats were subjected to 24 h sleep deprivation (SD) by running wheel method and Group III rats had 24h recovery sleep . At the end of the study, soleus muscle was collected for electron microscopic and neurochemical study. Morphometry was performed by modifying protocol mentioned in Spendiff et al.,2020 and neurotransmitter was measured by ELISA. The study was conducted as per the guidelines of the Institutional Animal Ethics Committee (960/IAEC/16).

Results: Electron microscopic observation revealed a significant increase in mitochondrial density in 24h SD ($p<0.01$) as compared to control in presynaptic terminal. Further, in post synapse we found similar increase in SD ($p<0.0002$).Within group comparison of intact versus altered mitochondria exhibited a significant increase in the diameter of altered mitochondria in all the experimental groups ($p\leq0.0001$). Moreover, within group comparison of area of intact versus altered mitochondria showed significant increase in area of altered mitochondria only in Control ($p=0.01$) and 24h SD ($p=0.02$). These aforementioned parameters did not show any significant change in recovery sleep. When we looked into the synaptic vesicle density we found significant increase in 24h SD as compared to control ($p=0.002$), but not in recovery sleep at presynaptic terminal. However, no significant difference was found in vesicle distribution within the active zone of presynaptic nerve terminal and postsynaptic muscle membrane of 24hr SD, control and recovery sleep groups. Interestingly, the number of junctional folds per synapse profile was significantly increased in the 24h SD group as compared to control, ($p=0.002$). Also, there was a significant difference between 24hr SD and recovery group ($p=0.002$). Concentration of acetylcholine in 24 h sleep deprivation was decreased ($p=0.02$). In addition, but acetylcholine esterase activity was significantly increased ($p=0.02$).The acetylcholine esterase activity did not come back to control level even after 24 of recovery sleep. Pearson correlation between acetylcholine and vesicular density showed found a negative correlation ($r=0.07$, $p<0.05$) in 24h sleep deprivation. These findings suggest that there are significant changes in neuromuscular junction of soleus muscle after 24hr sleep deprivation.

Conclusions: The results of the present study show that 24 h SD produces significant changes in neuromuscular junction morphology of soleus muscle. The changes were not observed after recovery sleep. We conclude that sleep plays a key role in NMJ homeostasis.

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Behavior, Cognition and Dreaming

AN EXPLORATION OF EARLY SLEEP DEVELOPMENT IN PRESCHOOL CHILDREN WITH AND WITHOUT A FAMILIAL HISTORY OF ADHD

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Introduction: Children with poor sleep are reported to show more problems with attention and do less well in school. Poor sleep is widely reported for children and adults with attention deficit hyperactivity disorder (ADHD), however the link between how this relation develops is relatively unknown. In our study, we investigated sleep and attention in infants and young children with and without a familial history of ADHD. By exploring the early development of sleep and attention we hope to gain a better understanding of how early sleep can impact brain and behaviour development.

Materials and Methods: We used both questionnaire and lab based methods to address our study aims. In Study 1, questionnaires on temperament markers of attention control and sleep quality and quantity were completed by parents of children under 6 years with (n=72) and without (n=139) a familial history of ADHD. In Study 2, actigraphs were worn by a subgroup of infants aged 10-20 months with and without a familial history of ADHD, to measure sleep-wake activity, and eye-tracking