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Late Breaking Abstracts

LBA 1

Reduced Slow Wave Activity in Unmedicated Adolescents with Major Depressive Disorder

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Introduction: Disrupted sleep is often a core complaint in individuals with major depressive disorder (MDD). In adolescents with MDD, studies have found changes in slow wave activity (SWA). Findings from previous studies, however, have been inconclusive due to samples with broad age ranges, different medication status and variable severity of depression. The aim of the present study was to examine sleep neurophysiology in an unmedicated sample of adolescents with and without MDD using high-density sleep electroencephalogram (EEG).

Methods: Sixty-five adolescents with and without depression between the age of 14 and 17 years (mean 15.15 years, SD = 1.1; 25 females; 35 with MDD) participated in the present study. Based on a clinical interview participants were screened for MDD. Participants followed a sleep-wake schedule for three days prior to the sleep EEG night. High-density sleep EEG (58 EEG derivations) was recorded in participants' homes. An ANOVA with factors age, sex and group was used to determine statistical differences between the groups and SWA was calculated as power in the 0.6-4.6 Hz range.

Results: We found a significant reduction of SWA in adolescents suffering from depression as compared to those without depression. Thirty-three derivations distributed over widespread brain regions showed statistically significant differences. Effect sizes were large, with eta-squared values for significant electrodes ranging between 0.11 to 0.28.

Conclusions: The diminution of SWA in adolescents with depression was topographically more widespread and effect sizes were larger compared to former studies. This might be explained by the recruitment of an unmedicated sample, the narrow age range and the moderate to severe depression, which may help reduce variability and increases statistical power. Our results add to the existing literature showing a reduction of SWA in depression and further our understanding of the role of sleep in adolescent depression.

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LBA 2

Partial Sleep Deprivation Associated with Increased Perivascular Space Volume Fraction

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Introduction: Studies indicate that late-stage sleep is crucial for the effective functioning of metabolic waste clearance processes. Perivascular spaces (PVS) are fluid-filled cavities surrounding blood vessels that participate in this process. Here, we studied whether a single night of partial sleep deprivation (PSD) would cause detectable changes in the PVS volume fraction (PVS vf).

Methods: MRI data from 81 young and older adults were collected after a night of normal sleep and after PSD (~4 hours) from the Stockholm Sleepy Brain study. PVS were automatically segmented and quantified from vesselness maps derived from T1w images. Paired t-tests were used to determine significant changes to PVS vf within white matter regions between the PSD and normal sleep conditions. Subjects were analyzed as both a complete cohort, and stratified by age group and sex. All results were corrected via Bonferroni multiple comparison correction ($p<.0015$).

Results: PVS vf was significantly larger after PSD compared to normal sleep in the left paracentral ($p=0.0466$), middle temporal ($p=0.02$), parsopercularis ($p=0.04$), and postcentral ($p=0.04$) regions; however, these tests did not survive multiple comparison correction. When analyzed by age group, the young group had a significant change in the left hemisphere paracentral region ($p=0.001$). Additionally, in the young female group, the left hemisphere superior parietal region trended toward significance ($p=0.0019$).

Conclusion: Our findings illustrate that one night of PSD could reflect glymphatic dysfunction observed via increased PVS vf, particularly in young adults and women. The finding that PSD appears to uniquely affect PVS in the left hemisphere suggests a unilateral effect of PSD on the glymphatic system. Next steps include examining if these structural changes are permanent or reversable, and analyzing polysomnography data to identify relationships between PVS vf and total sleep time and sleep efficiency.

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LBA 3

Different Simultaneous Sleep States in the Hippocampus and Neocortex of Human Subjects

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Introduction: Although sleep is defined as a homogenous brain state, a study from our lab in rodents that has been twice replicated reported that different areas of the brain could show different sleep states simultaneously. In this study, we explored whether different simultaneous states exist between the hippocampus and neocortex during sleep in humans.

Methods: We used scalp electroencephalography (EEG) and intracranial recordings (iEEG) from human subjects undergoing treatment for intractable epilepsy. Applying standard scoring criteria to each signal separately, the posterior hippocampus and scalp electrode (Cz) were sleep scored manually in 30 s epochs. Results: We found that the hippocampus and cortex spend different amounts of time in sleep. The cortex spends a significantly greater amount of time in Wake and REM than the hippocampus (n = 8 subjects). Accordingly, different simultaneous state epochs were just as common as same simultaneous state epochs. Furthermore, non-simultaneous bouts could last up to 25 mins. Analysis of power spectral density (PSD) for simultaneous and non-simultaneous states showed that the PSD profile for the brain region of interest in these non-simultaneous epochs closely matched the designated state's PSD when that state was simultaneously present in both structures. Non-simultaneous states analyzed included hippocampal N2 sleep during cortical waking, hippocampal N2 sleep during cortical REM, and hippocampal REM during cortical N2 sleep. Results indicate that whole subcortical sleep bouts can be missed when sleep scoring is based on scalp electrodes alone.

Implication: The existence of non-simultaneous sleep states in subcortical structures could mean that past studies of sleep function and homeostatic control may have missed covert sleep state occurrences. Future studies may consider the possibility that sleep could be locally homeostatically regulated depending on the functional requirement of the sleeping structure.

LBA 4

Incidence and Progression of Coronary Calcium Scores in Patients with Symptomatic Obstructive Sleep Apnea: the ELSA-Brasil study

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Introduction: Recent evidence suggests that symptomatic obstructive sleep apnea (OSA) patients are susceptible to cardiovascular events. The development of atherosclerosis is a plausible mechanism but the impact of OSA on atherosclerosis is unclear. Here, we assess the role of OSA and related clinical features on prevalence, incidence, and progression of coronary artery calcium (CAC) scores in a community-based cohort.

Methods: Consecutive participants from ELSA-Brasil underwent sleep assessments including questionnaires, actigraphy and home sleep studies. All participants had CAC scores measured using a 64-slice multi-detector computed tomography. Prevalent subclinical atherosclerosis was defined as CAC score>0 at baseline. Incident subclinical atherosclerosis was defined as baseline CAC=0 followed by CAC>0 at a 5-year follow-up visit. CAC progression was defined as baseline CAC>0 followed by an increase in scores at follow-up. The association between OSA (apnea-hypopnea index ≥15 events/h) and prevalent, incident and progression of subclinical atherosclerosis was assessed using logistic regression, adjusted by relevant cardiovascular risk factors. Stratified analyses based on excessive daytime sleepiness (EDS) were performed.

Results: We analyzed 1,956 participants with available CAC scores at baseline (age: 49±8 years; 57.9% women; 28.2% with OSA). We found higher prevalence of subclinical atherosclerosis is OSA patients (31.3%) compared to those without OSA (19.0%; p<0.001). In longitudinal analyses (n=1,247, mean follow-up: 5.1±0.9 years), we found a significant association between OSA and

incidence of subclinical atherosclerosis among those reporting EDS after adjusting for covariates (OR=1.97; 95% CI: 1.09–3.55; p=0.024). Furthermore, analysis of CAC progression (n=319) demonstrated that both OSA (β=1.08; 95% CI: 0.03-2.14; p=0.043) and OSA with EDS (β=1.66; 95%CI: 0.23-3.04; p=0.019) were significantly associated with atherosclerosis progression.

Conclusion: OSA, particularly with EDS, predicts the incidence and progression of subclinical atherosclerosis, a validated marker of future cardiovascular events. These results provided biological plausibility for the higher cardiovascular risk observed in excessively sleepy patients.

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