Conclusion: Our results demonstrate that noradrenaline and acetylcholine inhibit VLPO galanin neurons directly and indirectly. Both noradrenaline and acetylcholine increase GABAergic afferent inputs to VLPO galanin neurons by activating local GABAergic neurons. We propose that during wakefulness this feedforward inhibition provides additional inhibition of VLPO galanin sleep-promoting neurons.
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067

CAUSAL COMMUNICATION ACROSS THE ELECTRODE MANIFOLD DURING SLOW OSCILLATIONS

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Introduction: The slow oscillation (0.5-1Hz, SO) is the most studied sleep waveform and reflects sleep homeostasis and is crucial for memory consolidation. It is not clear how SO causally affects brain networks. We used the effective connectivity technique to investigate causal information flow across the electrode manifold during the SO.
Methods: Night sleep EEG signals of 59 adult participants were recorded and visually scored into five sleep stages. We used three EEG channels for each region including frontal, central, parietal, and occipital. SOs were detected automatically and signals from one second before to one second after the SO's troughs were used for estimating effective connectivity in SO and non-SO windows. Windowing technique and generalized partial directed coherence were employed to estimate causal information flow (CIF) between selected brain regions. The Linear mixed-effect (LME) method was used to model the peaks of the CIF based on different predictors including SO channel, source and sink of CIF, and distance between each of SO channel, source and sink regions.
Results: The results of CIF estimation showed two peaks of CIF about 250ms before and after the SO's trough, but no difference between CIF in SO's trough and non-SO windows. We found no effect of source and sink regions, and their distance on CIF (p-value > 0.05). However, distance between SO channel to source and sink region (p-value < 0.05) significantly predicted CIF. The coefficients of the LME model showed a direct effect of distance between SO channel to sink region and opposite effect of distance between SO channel to source region on CIF peaks.
Conclusion: The results showed there were significant changes of brain regions causal communication during SOs and these changes were affected by the distance of SO channel to sink and source region of CIF. Channels that are closer to the SO send more information and regions farther from the SO channel receive more information. Based on the results, we hypothesize that the SO brain networks are optimized to facilitate communication between regions that are far apart.
Support (if any):

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MORNING CARDIOVASCULAR FUNCTION IN CHRONIC CANNABIS USERS AND HEALTHY CONTROLS

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Introduction: In the US cannabis is recreationally legal in 15 states and medically legal in 34 states. Preclinical studies suggest beneficial effects of cannabis on the cardiovascular system (e.g., vasorelaxation). Yet, acute cases of hospitalization after cannabis consumption indicate potential adverse cardiac effects. Vascular endothelial function is a marker of cardiovascular disease and is measured as a change in resting brachial artery diameter (flow-mediated dilation, FMD) during reactive hyperemia. Both resting diameter (positively) and FMD response (negatively) are associated with cardiovascular risk. Resting diameter likely depends on long-term structural changes, and FMD response mostly depends on nitric oxide. Reactive hyperemia is more complex and depends on numerous variables, including adenosine and prostaglandins. FMD is attenuated in the morning when the frequency of adverse cardiovascular events peaks. To begin to understand the effects of chronic cannabis use on the cardiovascular system, in this pilot study, we compared morning measurements of vascular endothelial function, blood pressure, and heart rate between chronic cannabis users and controls while controlling for prior nighttime sleep opportunities.
Methods: Participants, cannabis non-users (n=5) and users (n=4), 44% female, age 25.4 ± 3.6 years - no demographic differences between groups, kept a consistent 2-week sleep schedule at home followed by an 8h sleep opportunity at their habitual time in the laboratory. Upon-wakening, we measured resting blood pressure, heart rate, baseline diameter, hyperemic response, and FMD. Statistical differences between groups were calculated using a two-tailed t-test.
Results: Systolic and diastolic blood pressures (p=0.13 and 0.26 respectively), heart rate (p=0.97), and FMD response (p=0.99) did not differ between groups. However, chronic cannabis users had a significantly higher baseline brachial artery diameter (mean difference: 1.04 mm ± 0.26, p=0.005), and lower hyperemic response (mean difference: -7944 iu/s ± 2538, p=0.02) compared to non-users.
Conclusion: These preliminary findings suggest that chronic cannabis consumption may be associated with adverse structural and functional changes in the vasculature of otherwise healthy young adults. Based on these initial observations, cannabis may act on the cardiovascular system via non-nitric oxide mechanisms. However, it is necessary to increase our sample size to test the robustness of these findings.
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CHRONIC PAIN IN VETERANS WITH TBI IS ASSOCIATED WITH DECREASED EEG SLOW WAVE COHERENCE DURING NREM SLEEP

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Introduction: Chronic pain and sleep disturbances are intricately linked to one another, especially in individuals with a history of traumatic brain injury (TBI) who are at greater risk for both symptoms. Although prior studies have analyzed differences in sleep electroencephalogram (EEG) in these clinical populations, the association between sleep EEG slow wave coherence and pain complaints is not fully examined or known. Our novel slow wave coherence approach may provide new insights into the relationship between TBI, chronic pain, and sleep
Methods: Ninety-six veterans were recruited and enrolled under a VA IRB-approved protocol. Participants completed a semi-structured clinical interview to determine their history of TBI, Symptom Impact Questionnaire Revised (SIQR), a measure of chronic pain complaints, and underwent an attended overnight in-lab polysomnogram (PSG). We developed a novel computational signal processing algorithm to identify and quantify EEG slow waves within 100 ms bins across the 6 standard PSG EEG channels. When a slow wave was simultaneously observed in 4 or more of the 6 leads, slow wave coherence was