whether motor learning leads to regionally specific spindle increases in the motor network in the nap that follows learning, whether local spindle increases correlate with post-nap performance improvement and whether SZ patients differ from controls in spindle changes and memory improvement.

**Methods:** SZ patients (n=15) and demographically-matched healthy controls (HC, n=12) were trained on the finger tapping motor sequence task (MST) and their performance tested after a 90 minute nap opportunity. We acquired continuous EEG and magnetoencephalographic (MEG) data simultaneously during MST training, the nap and MST testing. We computed the motor evoked responses, time-locked to each finger tap, for each subject during MST training and derived the anatomical constrained current source estimates using the minimum norm estimation method on both the EEG and MEG data.

**Results:** Preliminary analysis of 4 HC and 4 SZ patients showed that the subjects had sufficient sleep time (63.9±22.7 min) during MEG. Both groups showed significant overall performance improvement on MST and did not differ in this regard. The SZ group exhibited reduced spindle density over the central and frontal electrodes. This spindle density deficit was also prominent at the MEG sensors. The source localization of the motor evoked responses revealed right lateralized activation of the primary and supplementary motor areas for both groups.

**Conclusion:** These preliminary findings demonstrate the use of MEG/EEG to localize cortical sources of motor performance. We are presently conducting analyses to test our hypothesis that motor learning leads to specific spindle increases in the motor network that correlate with sleep-dependent memory consolidation in HC but not in SZ patients.

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### 1127

**LEG MOVEMENT ACTIVITY DURING SLEEP IN ADULTS WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER**

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**Introduction:** Sleep disturbances are prevalent in Attention Deficit Hyperactivity Disorder (ADHD). The hypothesis that the typical daytime hyperactivity observed in these patients may correspond to an increased motor activity during the night, thus causing sleep disruption, has been studied in children, but rarely in adults. Here we present a first detailed analysis of the nocturnal motor activity in ADHD adults compared to healthy controls, including the time structure of leg movements (LM) during sleep.

**Methods:** Fifteen ADHD patients and eighteen control subjects underwent four in-lab polysomnographic sleep recordings. The periodic character of LM was evaluated using validated markers of “periodicity”, i.e. the periodicity index, inter-movement intervals and time distribution of LM during sleep, in addition to standard parameters, such as the periodic leg movement during sleep index (PLMSI) and periodic leg movement during sleep arousal index (PLMSAI). Sleep quality and the prevalence of insomnia symptoms were assessed with the Pittsburgh Sleep Quality Index (PSQI) and the Insomnia Severity Index ( ISI). None of the participants had restless legs syndrome.

**Results:** Objective sleep parameters from the baseline night did not differ between ADHD and control subjects, with the exception of a longer sleep latency (p=0.007) in the patient group, as well as a slightly higher PLMSI (p=0.044) and PLMS duration (p=0.023), only in REM sleep. The PSQI questionnaire indicated a poor sleep quality and the ISI the prevalence of subclinical insomnia symptoms in ADHD patients.

**Conclusion:** Leg movement activity during sleep in ADHD adult subjects was neither more frequent than in healthy controls nor did the nocturnal motor events show an increased periodicity. The reduced subjective sleep quality reported by ADHD adults was in contrast to the normal objective polysomnographic parameters, suggesting a sleep-state misperception in these individuals or more subtle sleep abnormalities not picked up by the traditional sleep staging.

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### 1128

**LATER BEDTIME IS ASSOCIATED WITH DECREMENTS IN PERCEIVED CONTROL OF OBSESSIONS AND COMPULSIONS**

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**Introduction:** Accumulating evidence links sleep timing disruptions with obsessive-compulsive symptoms and poor treatment response. One theory proposes that impaired response inhibition contributes to the persistence of obsessions and compulsions, and research has similarly suggested that poor inhibitory control may be one cognitive consequence of sleep/circadian disruption. We hypothesize that individuals with disrupted sleep timing may lack the ability to dismiss obsessive thoughts and compulsive behaviors, ultimately resulting in more severe and treatment-resistant symptoms.

**Methods:** Twenty individuals diagnosed with OCD and ten individuals endorsing subthreshold OCD symptoms participated in one week of sleep and OC symptom monitoring. Participants wore actigraphs and completed sleep diaries and daily ratings of perceived degree of control over obsessive thoughts and ritualized behaviors. Hierarchical Linear Modeling (HLM) was used to investigate the interplay of sleep timing and OCD symptoms over time.

**Results:** The relation between perceived control of obsessions and previous night’s bedtime was significant, $t(27) = -3.23, p < .01$, indicating that later previous night’s bedtime was associated with lower perceived control of obsessive thoughts, when controlling for previous day’s perceived control of obsessions. Similarly, the relation between perceived control of compulsions and previous night’s bedtime approached significance. Consistent with our directional hypothesis, neither perceived control over obsessions or compulsions significantly predicted changes in bedtime.

**Conclusion:** These findings are consistent with the inhibitory failure theory of OCD which suggests that deficits in the ability to dismiss intrusions result in clinically significant obsessions, and similarly, and impaired behavioral inhibition gives rise to compulsions. We propose that disrupted sleep timing may be one mechanism which confers risk for such inhibitory deficits. Considering OCD-focused psychotherapy relies on refraining from compulsions, inhibitory control deficits may explain why individuals with comorbid OCD and sleep/circadian disruption have more severe and treatment-refractory symptoms.