(1hz) and spindles (9-15 Hz), play a key role in facilitating this consolidation. Our prior research reported age-associated declines in slow oscillations in Duchenne and Becker Muscular Dystrophy (DMD/BMD) (Simon et al., 2020). Here, we characterize age-associated changes in sleep spindle characteristics across development in this group.

Methods: Following our 2020 analysis, we retrospectively analyzed the clinical sleep studies of 28 DMD/BMD males (Age span: 4 to 20 years). We applied our spindle detection algorithm to six electrodes (F3, F4, C3, C4, 01, O2). We assessed spindle density, frequency, and amplitude based on age (child, early adolescent, and late adolescent).

Results: We conducted rmANOVAs to evaluate each spindle characteristics using within-factors (Stage and Electrode) and between-factor (Age). We found significantly more spindles with longer durations in N2 than N3; greater spindle density at frontal compared to occipital regions; and higher amplitudes at central compared to frontal sites. We found no age-associated changes in these spindle metrics. We did find an age-associated change in the frequency of spindles, with significantly greater average spindle amplitude increasing significantly with age.

Conclusion: In line with prior research, we found more spindles in N2 than N3 and greater spindle density at frontal compared to posterior electrodes. In contrast to our previous research demonstrating age-associated declines in slow oscillations, our current analyses show minimal age-associated changes in spindle characteristics from 4 to 20 years. Further analysis is required to assess for age-associated changes in spindle-slow oscillation coupling occur across development. Our findings have implications for functional changes in sleep-dependent cognition mechanisms across development in BMD/DMD.

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0192

EFFECTS OF EMERGING ALCOHOL USE ON DEVELOPMENTAL TRAJECTORIES OF FUNCTIONAL SLEEP MEASURES IN ADOLESCENTS.

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Introduction: Adolescence is characterized by developmental changes in sleep timing and architecture as well as alcohol use initiation. While the effects of acute and chronic alcohol use on sleep in adults are well-documented, much less is known in adolescents. We used longitudinal data from the National Consortium on Alcohol and Neurodevelopment in Adolescence (NCANDA) to examine how emerging alcohol use affected sleep architecture in adolescents.

Methods: Overnight polysomnographic recordings were made each year, for 4 years, in 94 adolescents (12–21 years at baseline, 43% female) from the NCANDA cohort. All participants were no or low (youth adjusted Cahalan score of zero) alcohol users at baseline. These data were used to examine developmental trajectories of sleep macro-architecture and sleep electroencephalographic (EEG) measures using linear mixed effect models (LMMs), considering age, sex, family history of alcohol use, body mass index, ethnicity, and alcohol use class (i.e., no-to-low, moderate or heavy) at each annual assessment.

Results: There were strong developmental changes in sleep macro-structure and EEG, most notably, a decrease in slow wave sleep percentage and slow wave (delta) EEG activity with advancing age (p=0.02). Compared to those who remained no-to-low drinkers, participants who became moderate/heavy drinkers during the follow-up period, had different sleep trajectories, especially those older at baseline at baseline, including higher slow wave activity (p = 0.04), higher REM sleep percentage (p = 0.03), poorer sleep efficiency (p=0.003), and longer latency to sustained sleep (p = 0.03). The effects of alcohol use depended on sex, with male heavy drinkers having more REM sleep than female heavy drinkers (p = 0.04). Overall, a positive family history of alcohol use was associated with less NREM sleep and shorter sleep duration.

Conclusion: Our results present novel findings showing that emerging alcohol use during adolescence exerts complex effects on sleep macro- and micro-structure, over and above normal developmental changes in sleep. These effects could, in part, be alcohol effects on brain maturation processes underlying sleep regulation.

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0193

MOVING WHILE SLEEPING: ON THE PARADOXICAL CO-OCCURRENCE OF MUSCLE ATONIA AND TWITCHING

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Introduction: During active (or REM) sleep, discreet, jerky movements called myoclonic twitches occur against a background of muscle atonia. The neural mechanisms that allow these two seemingly contradictory phenomena to co-occur remain unclear. One view holds that twitches are produced when a large descending motor signal overpowers the inhibition on spinal motor neurons. An alternative view is that atonia and twitching are coordinated by the same brainstem structures that produce and regulate active sleep. One such structure is the sublaterodorsal tegmental nucleus (SLD), which plays a key role in adult rats to the production of atonia, but whose contribution to twitching has not been examined. Here, we investigated the relationship of SLD neurons to atonia and twitching during a period in early development when twitching is abundant.

Methods: We recorded extracellular neural activity from the SLD in 8 12-day-old (P12) head-fixed rats as they cycled freely between sleep and wake. To assess the relationship between twitches and neural activity, we also recorded limb movements using high-speed video.

Results: Consistent with the adult literature, the majority of SLD neurons (35/56) were significantly more active during periods of sleep-related atonia. Interestingly, a subgroup of neurons (n=15) was inhibited at the onset of a burst of twitches, whereas another subgroup of neurons (n=26) was excited around the onset of a burst of twitches. Thus, together, the activity of 41 of 56 SLD neurons was modulated by twitches.

Conclusion: These results demonstrate that the activity of SLD neurons is associated with both atonia and twitching. This finding suggests that these two early-developing components of active sleep are coordinated within the brainstem. These initial findings open new avenues for further research into the neural mechanisms that coordinate the co-occurrence of twitching and atonia during active sleep.

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