participants with later average bedtimes also had more variability in their bedtime ($p < 0.001$).
Conclusion: Variability in nocturnal sleep was not associated with parent reported behavioral difficulties, which is contrary to recent findings. It is unclear if sleep duration and bedtime variability are associated with day-level changes in children's behavior. Additional work that emphasizes aspects of sleep beyond sleep duration is needed to advance our understanding of preschool-age children's sleep.
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0189

HIGH LEVELS OF SLEEP DISTURBANCE ACROSS EARLY CHILDHOOD INCREASES CARDIOMETABOLIC DISEASE RISK INDEX IN EARLY ADOLESCENCE: LONGITUDINAL SLEEP ANALYSIS USING THE HOME STUDY

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Introduction: Sleep is a predictor of cardiometabolic disease (CMD) risk, and new evidence links early childhood sleep to later CMD risk. This study examines the impact of early childhood sleep duration, bedtime timing, and sleep disturbance on a CMD risk score in early adolescence.
Methods: Within the Health Outcomes and Measures of Environment (HOME) Study, a prospective pregnancy and birth cohort study, we assessed sleep patterns among 346 children using the Children's Sleep Habits Questionnaire from ages 2 to 8 years. We calculated cardiometabolic risk scores ate age 12 for 183 of these children from visceral adiposity area, blood pressure, fasting serum triglyceride, high density lipoprotein, leptin, and adiponectin concentrations. We used a group-based semi-parametric mixture model to identify distinct trajectories in sleep duration, bedtime timing, and sleep disturbance for the entire sample. We then examined the associations between sleep trajectories and CMD risk score using general linear models for children with a CMD risk score, using both an unadjusted model (no covariates) and an adjusted model (adjusting for child pubertal stage, child sex, duration of breastfeeding, household income, and maternal education).
Results: Three sleep trajectories emerged for bedtime timing (late timing, medium timing, and early timing) and for sleep disturbance (high, medium, and low), and two for sleep duration (high and low). In the unadjusted model, we found significant differences in CMD risk scores by trajectories of sleep disturbance. Children in the 'high' trajectory had higher CMD risk scores (Least Square Mean=1.51; 95% CI: 0.39, 2.64) than those in the 'low' trajectory (Least Square Mean =-0.51; 95% CI: -1.16, 0.15; p=.002) and 'medium' trajectory (Least Square Mean=-0.15; 95% CI: -1.14, 0.85; p=.03). These findings only approached significance after adjusting for covariates. No significant differences in CMD risk were observed for bedtime timing or total sleep time trajectories in the unadjusted or adjusted models.
Conclusion: In this cohort, parent-reported sleep disturbance in early childhood was associated with more adverse cardiometabolic profiles in early adolescence. Our findings suggest that trials to reduce CMD risk via sleep interventions – which have been conducted in adolescents and adults – may be implemented too late.
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0190

ASSOCIATION OF SLEEP SPINDLE ACTIVITY WITH COGNITION IN YOUTH FROM THE GENERAL POPULATION

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Introduction: Sleep spindle activity has been increasingly studied as an underlying mechanism of cognition. In youth, it appears the relationship between spindle activity and cognition depends upon the spindle metric and cognitive domain examined. Prior research has been conducted primarily in highly selective experimental studies of typically developing youth. We aimed to clarify the relationship between spindle activity and lower and higher order cognitive functions in children and adolescents from the general population.
Methods: We studied 639 children aged 5-12y (median 9y) and 418 adolescents aged 12-23y (median 16y) from a population-based cohort. All subjects underwent a 9-hour, in-lab polysomnography. We calculated sleep spindle density (SSD), the total number of spindles per minute of stage 2 of non-rapid eye movement sleep, and peak spindle frequency (PSF) in the 10-16 Hz range at central, frontal and fronto-occipital derivations. Wechsler intelligence testing assessed verbal and non-verbal intelligence quotients (IQ), processing speed (coding) and working memory (digit span backward [DSB]). Multivariable-adjusted linear regression models with age, sex, race/ethnicity, body mass index, apnea/hypopnea index, and insomnia symptoms as covariates examined the association between SSD and PSF with cognitive outcomes.
Results: At ages 5-12, central SSD was positively associated with verbal IQ ($p=0.04$), non-verbal IQ ($p=0.03$), coding ($p=0.01$) and DSB ($p<0.01$); additionally, frontal SSD was positively associated with coding and DSB (both $p<0.01$) and fronto-occipital SSD with DSB ($p<0.01$). Also, central ($p<0.01$) and frontal ($p=0.01$) PSF was positively associated with DSB. At ages 12-23, fronto-occipital SSD was positively associated with non-verbal IQ ($p=0.02$), while no other statistically significant associations were observed for SSD or PSF with cognitive outcomes (all $p\geq0.08$).
Conclusion: Spindle density is a strong correlate of general ability (both verbal and non-verbal IQ) in childhood, and it remains for non-verbal IQ in adolescence. Both increased spindle density and peak frequency are associated with better working memory in childhood, yet not in adolescence. These developmental differences may be due to cortical (e.g., synaptic pruning) and thalamocortical (e.g., increased myelination) maturational changes occurring during adolescence.
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0191

CHARACTERISTICS OF SLEEP SPINDLES ACROSS DEVELOPMENT IN MALES WITH DUCHENNE/BECKER MUSCULAR DYSTROPHY DISORDER

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Introduction: Sleep supports cognition, in particular, the consolidation of memories. Sleeping brain rhythms, such as slow oscillations

(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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