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Review

Sleep deprivation and neurobehavioral functioning in children

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ABSTRACT

Sleep deprivation can result in significant impairments in daytime neurobehavioral functioning in children. Neural substrates impacted by sleep deprivation include the prefrontal cortex, basal ganglia and amygdala and result in difficulties with executive functioning, reward anticipation and emotional reactivity respectively. In everyday life, such difficulties contribute to academic struggles, challenging behaviors and public health concerns of substance abuse and suicidality. In this article, we aim to review 1) core neural structures impacted by sleep deprivation; 2) neurobehavioral problems associated with sleep deprivation; 3) specific mechanisms that may explain the relationship between sleep disturbances and neurobehavioral dysfunction; and 4) sleep problems reported in common neurodevelopmental disorders including attention deficit hyperactivity disorder (ADHD) and autistic spectrum disorders (ASDs).

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1. Introduction

Children demonstrate difficult behaviors when sleep deprived that can be stressful and impact quality of life for the entire family. Connecting sleep problems with daytime behavioral challenges may not be intuitive to parents. Adults manifest different symptoms when sleep deprived such as daytime sleepiness, psychomotor slowing and impairments in cognitive processing and memory (for a review see O'Brien et al., 2003). In comparison, sleep deprivation in children is more likely to be associated with a range of emotional/behavioral disturbances, including problematic behaviors (Aronen et al., 2000), attention problems (Fallone et al., 2005; Sadeh et al., 2006), anxiety/depression (Forbes et al., 2008), and hyperactivity (Touchette et al., 2009).

In this review, we focus on neural mechanisms that may explain the link between sleep deprivation and behavioral dysfunction in children, highlighting research conducted within the last 10 years. We include a discussion on common sleep problems associated with neurodevelopmental disorders, attention deficit hyperactivity disorder (ADHD) and autistic spectrum disorders (ASDs), emphasizing the bidirectional relationship between core symptoms and sleep problems. In defining neurobehavioral functioning, we refer to behaviors such as difficulties with attention, impulsivity, hyperactivity, performance skill difficulties, and aggression in addition to executive functioning abilities. The term executive functioning encompasses skills necessary for attention, planning and execution of tasks, set shifting, analysis, self-regulation and self-monitoring in addition to working memory.

2. Neuroanatomical structures and sleep deprivation

Brain maturation is a complex process (for a review, see Giedd, 1999) that begins prenatally with neural proliferation and migration and synapse formation continuing till two years of age. Myelination is an important process that begins prenatally as well but continues into adolescence with different systems myelinating at different times. The determinants of neurodevelopment and behavior rely on complex neural circuits that connect neural substrates to serve a specific function. The development of these neural circuits is still a mystery and influenced by genetic, sociocultural, medical and environmental factors (Rice and Barone, 2000). The neuroanatomic substrates involved in neurobehavioral functioning span cortical, subcortical and brainstem regions and formulate complex networks which include the prefrontal cortex, amygdala and striatum. Executive functioning is highly localized to the prefrontal cortex. The amygdala is of great importance to emotional reactivity and affect and striatum to reward seeking behavior.

Neuroimaging techniques reveal complex patterns of neuroanatomical functioning during specific sleep stages. During NREM slow wave sleep, the brainstem, thalamus, basal ganglia, and prefrontal and temporal lobe regions all appear to undergo reduced activity (Nofzinger, 2005). In REM sleep, significant levels of activity are reported in the pontine tegmentum, thalamic nuclei, occipital cortex, mediobasal prefrontal lobes together with affect related regions including the amygdala, hippocampus, and anterior cingulate cortex (Nofzinger, 2005). The

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prefrontal cortex is relatively inactive all through sleep in contrast to its high activity during waking states (Binder et al., 1999). This inactivity is reflected by the high voltage and slow brain wave oscillations in NREM sleep in the frontal lobes, relative to other cortical regions. This finding suggests that there is disabled thalamocortical input and a lower level of metabolism in the frontal lobes during NREM sleep stages (Maquet et al., 1997). Several investigators have suggested that sleep is particularly important for restoring prefrontal cortical activity (Dahl, 1996a, 1996b; Horne, 1993; Muzur et al., 2002) however, this restorative processes remains poorly understand.

Neuroimaging studies show profound effects of one night's total sleep deprivation on the blood flow to prefrontal areas which correspond to the deteriorations in daytime prefrontal task performance (Drummond et al., 1999; Thomas et al., 2000). For instance, in one study (Drummond et al., 1999), thirteen adult volunteers were studied with fMRI during arithmetic performance after a normal night of sleep and following sleep deprivation. After a full night of sleep, activation localized to the bilateral prefrontal cortex, parietal lobes and pre-motor areas. Following sleep deprivation, activity in these regions decreased markedly, especially in the prefrontal cortex, as measured performance levels also dropped. Surprisingly, studies of groups of young adults failed to demonstrate deficits in executive function following sleep deprivation (Binks et al., 1999; Pace-Schott et al., 2009). College students reported impairments on the majority of the executive function tasks tested following one night's total sleep deprivation; however, participants sleep deprived 35 to 39 h showed few performance deficits on these tests when compared with non-sleep-deprived controls (Pace-Schott et al., 2009). One possible explanation for this discrepancy might be that younger people are able to adapt to chronic sleep deprivation better than older adults.

Sleep deprivation also impacts neural circuitry underlying regulation of emotions, impulsivity and reward seeking behavior. Sleep deprived adult volunteers viewing emotional images have increased activation of the amygdala on functional neuroimaging yet weaker connection between the prefrontal cortex and the amygdala (van der Helm et al., 2010). This scenario allows for uncontrolled, increased emotional response. Likewise, neurocognitive functions that involve the striatum and basal ganglia such as risk avoidance and responsiveness to rewards are also impacted by sleep deprivation. For instance, sleep deprived adults take greater risks and are less concerned about consequences of their behavior (Dijk, 2011). Such findings have also been noted in adolescents aged 11–13 years using functional magnetic resonance imaging (fMRI) and a guessing task with monetary rewards (Holm et al., 2009). During reward anticipation, less activation in the caudate nucleus (part of the ventral striatum) was associated with reduced sleep time, later sleep onset time, and lower self-reported sleep quality. During reward outcome, less caudate activation was seen with later sleep onset time, earlier sleep offset time, and lower sleep quality. The study suggests that sleep deprivation could contribute to low reactivity in reward-related brain areas in adolescents and may lead to compensatory increases in reward-driven behavior. Such findings have significant public health implications when one considers that reward seeking behaviors are associated with depressive symptoms, sensation seeking, and substance abuse in adolescents (Forbes et al., 2010; Nelson et al., 2005).

3. Experimental sleep deprivation and neurobehavioral functioning in otherwise healthy children

Partial sleep deprivation and problems initiating and maintaining sleep impact the lives of about 25–40% of normally developing children and adolescents (Meltzer and Mindell, 2006; Owens, 2007). The normal duration of sleep varies by age (Iglowstein et al., 2003), culture, and environment (Hense et al., 2011). Sleep duration is influenced by habitual bedtime, rise time, circadian phase, and external mandatory schedules (e.g., school days versus weekend days). Subjective sleep duration has slowly declined over the last 50 years with the evolution of a more intense and demanding pace of life (Bixler, 2009; Krueger and Friedman, 2009). In a national poll of American children, 34% of toddlers, 32% of preschoolers and 27% of school aged children were reported to sleep fewer hours than what the parent/caregiver thought they needed (National Sleep Foundation, 2004). More objective evidence confirms these concerns. Children ages 4–10 years slept on average 8 h per night compared to the recommended 10 h for this age range during a recent study using wrist actigraphy (Spruyt et al., 2011).

School performance is often used as a marker of both neurobehavioral and cognitive functioning in children. Only small overall effect sizes are reported between sleep and academic function suggesting that more sleep does not necessarily result in better school performance (Astill et al., 2012; Dewald et al., 2010). Effect sizes were larger for studies including younger participants than for studies that included older participants (Dewald et al., 2010). Overall, shorter duration of sleep is associated with more behavior problems ($r = 0.9$, 95% CI 0.07–0.11) (Astill et al., 2012).

Few experimental studies have been conducted with children that include sleep restriction. Sadeh et al. (2003) studied 77 children in the 4th–6th grades and randomized them to 1 h of sleep extension or one hour sleep restriction. The sleep restriction group slept on average 41 min shorter and the sleep extension group slept about 35 min longer compared to their baseline. Notably, the sleep extension group reported improvements on executive functioning tasks such as the digit span task (measuring memory function) and the continuous performance task (measuring attention and visual discrimination) compared to the sleep restriction group. Similarly, in the Fallone and Carskadan study, subjects aged 6–12 years were randomized to either an “optimized sleep group” in which they slept no more than 10 h or a “restricted sleep group” (sleep restricted to 8 h or 6.5 h if a child was in 3rd grade or above) for a three week period (Fallone et al., 2005). The order of conditions was then counterbalanced. Teachers were surveyed on children's behavior characteristics including hyperactivity, impulsivity, excessive talkativeness, internalizing symptoms (sad affect, complaints of physical problems or emotional lability), as well as oppositional and aggressive behaviors. Of these measures, the sleep restriction group had significantly more academic and attention problems but did not score higher on hyperactive–impulsive, internalizing or oppositional–aggressive scales as hypothesized. Authors explained this by showing higher sleepiness scores in the sleep restricted group and suggested that only children with attention deficit disorders may be more likely to demonstrate paradoxical hyperactivity with sleep deprivation.

4. Poor sleep contributing to future neurobehavior problems in children

A recent area of research interest has been the role of early sleep problems in the development of future affective and neurobehavioral problems. Touchette et al. found that patterns of persistent short sleep duration or tendency for short sleep duration in childhood (2.5–6 years) predict high hyperactivity and impulsivity scores and poor performance on neurodevelopmental tasks at time of school entry (ages 5–6 years) (Touchette et al., 2007). This group went on to investigate risk factors associated with short nighttime sleep duration and hyperactivity between ages 1.5 and 5 years using a large database of 2057 children (Touchette et al., 2009). Investigators found that risk of short nocturnal sleep time in kids with high hyperactivity scores (OR 5.1, 95% CI [3.2–7.9]) was higher than risk of finding high hyperactivity scores in short sleepers (OR 4.2, 95% CI [2.7–6.6]). While causality cannot be truly discerned from this study design, results suggest that it is more plausible that hyperactivity may interfere with nighttime sleep duration rather than short sleep resulting in higher hyperactivity scores. Risk factors for having short sleep duration and high hyperactivity scores included being male, having insufficient household