

The Unrested Adolescent Brain

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ABSTRACT—*Mounting scientific evidence illustrates the detrimental effects of sleep loss on the brain and behavioral functioning. Given the ongoing development of the brain from the onset of puberty into the early 20s, these behavioral and neural effects are particularly concerning for adolescents. In this review, I summarize what we know about the relation among sleep disruption, changes in sleep biology across puberty, and the developing adolescent brain. Although examinations of sleep and the developing brain have been promising, significant gaps in knowledge remain regarding populations sampled and methodological challenges. The field will also benefit from better communication among sleep researchers and developmental neuroscientists to translate the results of studies on the brain into meaningful outcomes.*

KEYWORDS—*adolescence; brain development; mesolimbic circuitry; regulatory systems*

Sleep is a basic need. Without sleep, cognitive, learning, and emotional regulation skills are disturbed. Mounting evidence suggests this is particularly true during adolescence, a developmental period characterized by substantial change in the brain regions that support cognition, learning, and emotion. In this article, I briefly introduce the concept of normative brain development and the emerging data illustrating neural susceptibility to sleep disruption during adolescence (which I define as starting at the onset of puberty and continuing into the early 20s). When available, I provide the age range for the studies I cite.

Three reasons motivate us to investigate how sleep disruption affects the developing human brain. First, understanding sleep disruption from a neurobiological perspective may help explain

maladaptive behavioral changes sometimes observed as children transition into adolescence. Second, improving sleep may be a way to treat or prevent mental health disorders that emerge during adolescence. The onset of anxiety, depression, and other internalizing disorders increases in early to mid-adolescence and coincides with sleep problems (Alfano & Gamble, 2009). Third, developmental neuroscience studies on sleep may contribute to scholarly conversations regarding sleep recommendations for adolescent health policies.

In this article, I broadly review the relation between sleep disruption and the human brain across two psychological areas (cognitive control and reward sensitivity) and three neural domains (neural activation, white matter development, and connectivity). Based on neuroimaging studies, I describe the neural correlates of cognition and affect that vary as a function of sleep factors during the second decade of life.

Sleep is a period of reduced activity during which the individual exhibits decreased responsiveness to external stimuli. The primary outcomes examined in most of the studies on sleep I review are *sleep duration* and *sleep quality*. Sleep duration generally refers to number of hours of sleep, but because the exact number of hours of sleep is difficult to measure objectively, a more precise definition is how long an individual is in bed. Sleep quality refers to the extent to which sleep was restful and undisturbed, typically based on the average number of awakenings. Sleep is a multidimensional construct examined through various methods, including actigraph watches (which monitor rest and active cycles), polysomnography (which records biophysiological changes during sleep, which may include blood-oxygen level, heart rate, breathing, and brain waves), and self-report (which is done through daily diaries and questionnaires). In this review, I also address *sleep disruption*, which is a combination of sleep duration and quality. Other measures of sleep have been examined (though to a lesser extent), including sleep spindles (a burst of brain activity) and aspects of sleep architecture (including different sleep stages, such as rapid eye movement, or REM, and slow-wave sleep). As such, broad generalizations about sleep are challenging and results may differ based on the methods used.

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ADOLESCENT SLEEP PATTERNS

Precise sleep habits in adolescents are challenging to define. Cultural differences and individual circumstances yield varying attitudes, practices, and customs related to sleep. However, fairly consistent estimates using either self-report (National Sleep Foundation, 2014) or objectively recorded sleep (Crowley, Wolfson, Tarokh, & Carskadon, 2018) suggest that adolescent sleep varies from ~ 6 hr in high school seniors (~17–18 years old) to 8.4 h in early adolescents (~11–12 years old). Some estimates report that sleep decreases by ~ 90 min from sixth to twelfth grade among American adolescents (Tarokh, Saletin, & Carskadon, 2016). The Youth Behavior Risk Surveillance Data from 2007, 2009, 2011, and 2014 ($n = 50,370$ U.S. students) reported that two thirds of students in grades 9 to 12 obtained 7 hr or less sleep on school nights (Wheaton, Olsen, Miller, & Croft, 2016), with similar trends in other countries. This finding is corroborated with actigraphy-based research with 12- to 18-year-olds (Galland et al., 2018), despite evidence that roughly 9 to 9.25 hr per night is necessary for optimal cognitive performance and emotion regulation (Owens et al., 2014).

WHY DOES ADOLESCENT SLEEP DECLINE?

The need for sleep does not change as children become adolescents (Fuligni et al., 2017; Short et al., 2018). However, psychosocial and biological factors contribute to a decline in sleep as children grow older. Socializing (Arora, Broglia, Thomas, & Taheri, 2014), screen time (Gradisar et al., 2013), and studying (Adam, Snell, & Pendry, 2007) increase the likelihood of less optimal sleep in adolescents. Although sleep modifications are consistent with typical development, sleep loss and daytime sleepiness can adversely affect adolescents' cognition, mood, health, and safety (Owens et al., 2014).

Biological alterations to circadian and homeostatic systems at puberty are also related to a delay in sleep phase because they alter the body's internal clock, which makes it increasingly challenging for adolescents to fall asleep (Hagenauer et al., 2009). The circadian system, in the brain's suprachiasmatic nucleus of the hypothalamus, governs the body's internal clock. Research using both self-report and objective measures (Carskadon, Acebo, & Jenni, 2004) indicates that as puberty progresses, the central circadian clock shifts to a later time (Crowley et al., 2018), an effect that is also observed in nonhuman animals (Hagenauer et al., 2009). Two hypotheses explain this puberty-related shift in circadian rhythm: The first, based on research on animals (McGinnis, Lumia, Tetel, Molenda-Figueira, & Possidente, 2007) and humans (Carskadon et al., 2004), posits that the internal length of day of the circadian system increases over the course of puberty (Crowley et al., 2018). However, a recent study failed to find age- or puberty-related differences in circadian periods (Crowley & Eastman, 2018). The second hypothesis proposes that the circadian response to light changes across

puberty (Hagenauer et al., 2009), but recent research does not support this explanation (Crowley et al., 2018).

The homeostatic system also undergoes substantial developmental change. It induces increasing pressure to sleep the longer one is awake and favors wakefulness the longer one is asleep. Relative to younger (pre- or early pubertal) adolescents, adolescents exhibit a longer delay in buildup sleep pressure, which means that adolescents who are more mature pubertally do not feel the need to sleep until later at night (Jenni, Achermann, & Carskadon, 2005).

A NEURAL OVERLAP BETWEEN DEVELOPMENT AND VULNERABILITY TO SLEEP

Sleep Research in the Adult Brain

Neuroscientific studies of sleep in adults paint a clear picture about the brain regions that are most susceptible to sleep loss. Sleep deprivation leads to significant operative changes in affective and regulatory systems (Krause et al., 2017). For example, subcortical brain regions implicated in reward processing, including the ventral striatum (VS), medial prefrontal cortex (mPFC), and orbitofrontal cortex, become hypersensitized by sleep deprivation, and are activated in an exaggerated way when anticipating and receiving monetary rewards (Venkatraman, Chuah, Huettel, & Chee, 2007). Responses in the amygdala to emotional images are also amplified following deprived sleep (Yoo, Hu, Gujar, Jolesz, & Walker, 2007). There are cortical repercussions, too, including decreased activation in the dorso-lateral prefrontal cortex (DLPFC) and intraparietal sulcus following sleep deprivation; these neural effects are accompanied by poor attentional skills and impaired memory (Yoo et al., 2007). One night of sleep deprivation hinders learning performance, hippocampal engagement (Yoo et al., 2007), and functional connections between the hippocampus and the DLPFC (Chee & Chuah, 2007). Simply put, the brain processes identical emotional, rewarding, or cognitive information differently when it has been deprived of sleep.

Sleep Disruption in the Adolescent Brain

How does sleep disruption affect the adolescent brain? Despite extensive research on adults regarding sleep and the brain, we lack equivalent data on children and adolescents. Given the substantial neural changes that occur across development, sleep effects are probably unique at different developmental stages. A Pubmed search suggests that few studies have investigated brain functioning following experimentally restricted sleep. One reason may be that experimental sleep restriction may not be ecologically appropriate, is often less practical, and may be more difficult given shifts in sleep pressure (the need to sleep) in youth. However, these studies have great value in demonstrating how restricting or extending sleep affects brain activity and behavior. In studies using electroencephalography (EEG) activation, sleep restriction (50% of habitual sleep) in 5- to 12-year-

olds yielded high neuronal responsiveness in parieto-occipital regions (Kurth et al., 2016). In studies using EEG and polysomnography in 15- to 17-year-olds, sleep spindles, a hallmark of stage 2 sleep, were lower in amplitude and longer in duration than is typical after sleep was restricted for five hours (Reynolds, Gradisar, Coussens, & Short, 2018). This is relevant to adolescence because of the important role sleep spindles play in cognitive functioning and development (Fogel & Smith, 2011).

In another study that used functional magnetic resonance imaging (fMRI), 13- to 15-year-olds participated in two experimental conditions (sleep restriction of 4 hr and “normal” sleep of 8 hr) 1 week apart, each followed by fMRI (Robinson, Erath, Kana, & El-Sheikh, 2018). The teenagers whose sleep was restricted engaged in more risky behaviors and made more behavioral errors than their peers whose sleep was not restricted; researchers also saw connectivity disruption in the youth’s limbic and regulatory systems (Robinson et al., 2018). These findings corroborate research on sleep disruption in 18- to 25-year-olds (Mullin et al., 2013). In yet another study, 15-year-olds participated in a 3-week protocol: a baseline week, followed in random order by a week of sleep restriction (6½ hr a night in bed), and a week in which they spent 10 hr a night in bed. Brain scans at the end of each experimental week revealed greater neural activation in response to a working memory paradigm after the sleep restriction than after the longer sleep (Beebe et al., 2009). This study was the first to demonstrate feasibility of an in-home sleep restriction protocol, but it needs replication because the sample was limited to six participants and the scanning was conducted across two scanners.

A Brief Overview of Adolescent Brain Development

Neuroscientific tools have provided a window into ontogenetic neurobiological changes at puberty. First, adolescents evince greater activation in the VS to rewarding stimuli than do children or adults (Casey, 2014); this pattern extends to neural activation in the VS and hippocampus during learning (Davidow, Foerde, Galván, & Shohamy, 2014; Peters & Crone, 2017). Second, the prefrontal cortex (PFC) exhibits ongoing development through late adolescence and early adulthood (Tamnes et al., 2017). Given the pivotal role of the PFC in reasoning, learning, impulse control, and cognition, adolescent impulsivity and risk-taking have been attributed to the protracted maturation of the PFC (Casey, 2014). Third, as children become adolescents, their brains respond to contextual input by significantly strengthening and weakening connections among key regions and in the default mode network (Fair et al., 2008).

Mesolimbic Circuitry

In fMRI, tasks that require individuals to make risky choices or receive rewards elicit activation of the mesolimbic system. The mesolimbic system is a dopaminergic pathway that transports the neurotransmitter dopamine from the midbrain to regions of

the PFC; it also monitors and responds to affect in the environment (Haber & Knutson, 2009). In one study (Telzer, Fuligni, Lieberman, & Galván, 2013), activation in mesolimbic regions, including the insula and striatum, varied at the individual level as a function of sleep quality: 14- to 16-year-olds who reported less optimal sleep had more insula activation and less functional coupling between the DLPFC and insula and the DLPFC and striatum. Teenagers who reported greater likelihood of risky behavior and took more risks on the fMRI task had less optimal quality of sleep (Telzer et al., 2013).

In another study (Uy & Galván, 2017), under conditions of self-reported stress and in contrast to a comparison group of 25- to 30-year-olds, 15- to 17-year-olds took more risks on a computerized risk-taking task and exhibited greater activation of mesolimbic circuitry. Participants completed a daily diary that indicated their levels of stress for 2 weeks. In the laboratory, they had brain scans while completing risky decision-making tasks when under self-reported high and low levels of stress. All participants, adolescents and adults, with greater sleep duration exhibited fewer nonadvantageous risk-taking during high stress than those who had less sleep each night. Moreover, more hours of sleep were related to greater functional connectivity between the insula and DLPFC under high stress but not under low stress. This suggests that the number of hours of sleep per night may exaggerate the impact of daily stress and modify risky decision-making behavior through neural interactions. Together, these behavioral and neural profiles align with speculation that sleep disruption triggers an indiscriminate engagement of mesolimbic circuitry so the brain codes differences in reward value less accurately (Krause et al., 2017).

Regulatory Circuitry

Activation in the PFC varies among adolescents as a function of quality of sleep. In one study, less optimal quality of sleep correlated with decreased DLPFC activation during a cognitive control task (Telzer et al., 2013). Individual variation in this region was associated with variability in impulsivity and risk-taking behavior on health-compromising risk behaviors (e.g., cigarette smoking; Galván et al., 2013). In addition, longitudinal changes in ventrolateral PFC activation related to declines in self-reported risk-taking (Qu, Galván, Fuligni, Lieberman, & Telzer, 2015).

White Matter Development

Frontocortical and frontostriatal tracts of white matter undergo increasing myelination through the adolescent and early adulthood years (Asato, Terwilliger, Woo, & Luna, 2010). This maturation process expedites faster neural transmission and cognitive processing. As such, white matter maturation that is relatively delayed or altered can affect cognition and behavior, including externalizing issues (Li, Mathews, Wang, Dunn, & Kronenberg, 2005), substance use (Acheson et al., 2014), and poor cognitive performance (Chaddock-Heyman et al., 2013). Indeed,

in one study (Telzer, Goldenberg, Fuligni, Lieberman, & Galván, 2015), greater variation in duration of sleep 1 year before an fMRI was linked to delayed maturation of white matter in 14- to 16-year-olds.

In this study, adolescents provided daily reports of sleep habits, including bedtime and wake time at two time points 1 year apart. After the second collection of daily reports, the adolescents underwent a diffusion tensor imaging scan. This type of scan is used to investigate the microstructural components of white matter. Fractional anisotropy, which measures the organization of fibers in white matter, is used to identify axonal maturation. Based on the self-reports of sleep duration, greater variation in sleep duration 1½ years before the diffusion tensor imaging scan was linked to lower fractional anisotropy values accounting for the effects of sleep duration and variability in bedtime. The effect was specific to fractional anisotropy in frontocortical, frontostriatal, and parietocortical tracts. Together, these white matter tracts are vital for transmitting information between regions in the PFC and regions in the subcortical brain, and are therefore necessary for optimal cognitive and emotional control (Asato et al., 2010). These data suggest that suboptimal sleep may manifest in brain development over time.

Development of Gray Matter

Data unequivocally demonstrate the continued development of gray matter in the brain throughout childhood and adolescence. For example, a longitudinal study of four data sets with 7- to 29-year-olds showed profound anatomical decreases in cortical thickness and volume with increasing age (Tamnes et al., 2017). In another study, hippocampal gray matter was related positively to sleep duration in 5- to 12-year-olds (Taki et al., 2012). Research with 8- to 19-year-olds also suggests a strong relation between cortical maturation and slow-wave activity (Buchmann et al., 2011), showing that slow-wave activity during sleep might represent a good electrophysiological marker of cortical maturation. Relatedly, in a recent cross-sectional analysis of polysomnographic, EEG, and MRI data from healthy 12- to 21-year-olds, cortical thickness and volume in frontal and parietal regions mediated the relation between sleep and slow-wave activity during adolescence (Goldstone et al., 2018). In other words, the commonly observed decrease in slow-wave activity in more mature adolescents may be linked to reductions in volume of gray matter and cortical thickness.

Default Mode Network

Similar to data from studies of adults, sleep disruption impairs optimal functioning of the default mode network in adolescents (Tashjian, Goldenberg, Monti, & Galván, 2018). This is a network anatomically identified by the anterior-posterior midline regions of the mPFC (ventromedial PFC, anterior cingulate cortex), the medial parietal cortex (posterior cingulate cortex, precuneus, retrosplenial cortex), and the lateral temporo-parietal cortex (supramarginal gyrus, angular gyrus, superior temporal

sulcus). At rest (i.e., when an individual is not laboring over a cognitively demanding task), these regions correlate robustly, hence the moniker *default mode* (a.k.a. rest) network. At optimal brain function, this metabolic activity attenuates when an individual is engaged in tasks that are cognitively intensive (Andrews-Hanna, Reidler, Huang, & Buckner, 2010). Research in adults shows a reduction in resting state default mode network connectivity after sleep deprivation compared to connectivity following a “normal” night of sleep, which suggests that intrinsic connectivity in this network may be susceptible to drastic sleep changes.

In another study of 14- to 18-year-olds (Tashjian et al., 2018), researchers combined actigraphy measures with resting state functional connectivity to identify the relation between connectivity in the regions of the default mode network and sleep patterns. Less optimal sleep quality, but not duration of sleep, was related to weaker intranetwork connectivity in the default mode network (Tashjian et al., 2018). Moreover, adolescents who had greater affect-related impulsivity with low but not high connectivity had less optimal sleep quality, suggesting that individual differences linking sleep quality and impulsivity relate to variation in brain function (Tashjian et al., 2018).

CONCLUSIONS

Problematic sleep in adolescents is a topic of great interest to teachers, parents and other caregivers, and policymakers. Most initiatives to address sleep problems emphasize the need to increase the duration of adolescents' sleep (National Sleep Foundation, 2014), overlooking the equally important value of the *quality* of that sleep. The upsurge of research on the complex relation between sleep and the developing brain is long overdue. Hopefully, we can make up for lost time by vigorously and swiftly pursuing the rigorous science necessary to uncover the long-lasting impact of sleep health to keep pace with the continuous decline in sleep.

The studies I have reviewed are a promising step into launching this interdisciplinary research, but we need to take further steps to help fill gaps in our knowledge. For example, despite studies identifying differences in sleep patterns among ethnically and socioeconomically diverse groups (Yip et al., 2019), we lack research on whether these differential sleep patterns have distinct effects on brain development among diverse populations. We also lack information about the link between sleep and other indicators of health (e.g., immune functioning, obesity) in adolescents. Both of these areas merit further study to deepen our understanding of the implications of variations in sleep patterns.

Moreover, the field faces methodological challenges in characterizing the relation between sleep and the developing brain. Although the studies I have described are methodologically strong in imaging techniques, they could be improved in terms of their ecological validity. Brain researchers are limited by the

extent to which they can simulate sleep practices in a scanner (e.g., to characterize sleep architecture) that retain some semblance of real-life sleep. Although polysomnography studies circumvent this to some extent, researchers cannot keep someone in a scanner overnight to examine different sleep stages. The many approaches used to define, monitor, and measure sleep (e.g., actigraphy, self-reports) can also be problematic because they limit generalizability and comparison among studies. This may be in part because many of the neuroimagers who have taken an interest in sleep are not trained as sleep researchers and vice versa. We also need greater communication among sleep researchers and developmental neuroscientists to translate the brain results into meaningful implications. However, despite these limits, the increasing interest in the link between sleep and brain development is promising.

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