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Neural connectivity moderates the association between sleep and impulsivity in adolescents

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ABSTRACT

Adolescence is characterized by chronic insufficient sleep and extensive brain development, but the relation between adolescent sleep and brain function remains unclear. We report the first functional magnetic resonance imaging study to investigate functional connectivity as a moderator between sleep and impulsivity, a problematic behavior during this developmental period. Naturalistic differences in sleep have not yet been explored as treatable contributors to adolescent impulsivity. Although public and scientific attention focuses on sleep duration, we report individual differences in sleep quality, not duration, in fifty-five adolescents (ages 14–18) yielded significant differences in functional connectivity between the prefrontal cortex and default mode network. Poor sleep quality was related to greater affect-related impulsivity among adolescents with low, but not high, connectivity, suggesting neural functioning relates to individual differences linking sleep quality and impulsivity. Response inhibition and cognitive impulsivity were not related to sleep quality, suggesting that sleep has a greater impact on affect-related impulsivity. Exploring environmental contributors of poor sleep quality, we demonstrated pillow comfort was uniquely related to sleep quality over age, sex, and income, a promising advance ripe for intervention.

1. Introduction

Extensive research has sought to uncover neurobiological factors contributing to impulsivity, a characteristic trait of adolescence, in part because impulsivity can have dire consequences for health and well-being (Hamza et al., 2015; Heron, 2016). These efforts have largely focused on specialized mesolimbic reward circuitry and the frontal cortex, giving less attention to neural network functioning (Zhu et al., 2015). Naturalistic developmental declines in sleep (Hagenauer et al., 2009), which are linked to problematic behaviors such as poor impulse control and emotion regulation (Beebe, 2011), are also overlooked as mechanisms to understand adolescent impulsivity. The public enthusiasm for healthy sleep (Green, 2017) has outpaced our scientific understanding of the consequences of poor sleep, particularly with respect to the developing brain. This relative disregard of sleep precludes the neuroscience community from contributing to important policy debates concerning the role of changing sleep in adolescence.

Poor sleep has been linked to a myriad of negative outcomes in both humans and animals (Beebe, 2011; McCoy and Strecker, 2011). Human adolescents are particularly vulnerable to negative sleep-related outcomes, including impulsivity, due to both maturational lags in cognitive control pathways in the brain (Somerville et al., 2010) and puberty-influenced sleep deficiencies (Hagenauer et al., 2009). Sleep deficiencies exacerbate limited prefrontal cortex (PFC) cognitive capacity and further burden resources putatively needed to control impulsive behavior (Chee and Choo, 2004; Drummond et al., 2000). Individual differences in impulsivity have been linked to much of the health-compromising risk taking (i.e., substance use, reckless driving) observed during adolescence (e.g., Romer et al., 2009), motivating extensive research on the environmental and neurological factors that are associated with increased impulsivity during this period. However, little work has examined sleep-related differences in neural systems as a way of explaining impulsive behavioral problems, relying instead on neurocognitive differences during behavioral performance (Zhu et al., 2015).

Although impulsivity has been linked to important behavioral outcomes in adolescence, impulsivity is a multifaceted construct and the impact of sleep and neural functioning may depend on the type of impulsivity examined. Impulsivity consists of separable constructs of affect- or motivation-driven impulsivity, such as urgency and sensation seeking, and cognitive impulsivity, such as lack of premeditation and lack of perseverance (Whiteside and Lynam, 2001). Sleep loss is

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associated with mood disorders (Peterson and Benca, 2006), increased aggression (Kamphuis et al., 2012), and increased emotional reactivity (Kahn-Green et al., 2006), suggesting that affect-related impulsivity may be most affected by individual differences in sleep. For example, adults exhibit increased impulsivity to negative emotional stimuli following experimental sleep deprivation (Anderson and Platten, 2011). In neuroimaging research, cognitive impulsivity is frequently assessed as response inhibition or the ability to control a pre-potent motor response. Experimental sleep deprivation research has found that adults have impaired response inhibition during total sleep deprivation (Drummond et al., 2006). Together, this work suggests an association between sleep and impulsivity, but there has been little research investigating the relation between sleep and different dimensions of impulsivity. Thus, in the present study, we explored both state and trait impulsivity as they related to sleep and neural connectivity, the latter of which included measures of both affect-driven and cognitive impulsivity. Adolescence offers a unique period to investigate these constructs as impulse control and neural networks are developing, and sleep is affected by hormonal changes.

Motivations for investigating how sleep affects the large-scale default mode network (DMN) include: (1) reduced DMN connectivity has been linked to increased impulsivity in children (Inuggi et al., 2014); (2) the DMN undergoes considerable development during adolescence (Fair et al., 2008; Sherman et al., 2014); (3) adult research suggests sleep disrupts connectivity in the DMN (De Havas et al., 2012); and (4) DMN functioning has effects on a myriad of psychological and behavioral processes (Broyd et al., 2009; Sambataro et al., 2010; Schilbach et al., 2008). In adults, the DMN is identified as anterior-posterior midline regions of the medial prefrontal cortex (mPFC), medial parietal cortex, and lateral temporo-parietal cortex (Fox and Raichle, 2007). Tasks that demand sustained attention reliably suppress DMN activity, but if it is not suppressed, cognition and performance can be negatively affected (Anticevic et al., 2012; Whelan et al., 2012). Weaker connectivity in the DMN is related to less DMN suppression during goal-directed behavior (Zou et al., 2013). In addition to intrinsic connectivity within the DMN, the way it interacts with other brain regions influences cognitive performance after sleep deprivation (Lei et al., 2015). Although prior research suggests the adult DMN is vulnerable to sleep restriction (De Havas et al., 2012), links between sleep and adolescent DMN functioning have yet to be established. Given independent links between sleep and DMN functioning in adults, sleep and impulsivity, and DMN functioning and impulsivity, we explored whether individual differences in DMN functioning explained links between sleep and impulsivity in adolescents.

Healthy sleep is recognized as important for optimal functioning, but the factors that contribute to disrupted and insufficient sleep are not well known. Few studies investigate associations between environment and poor sleep in healthy adolescents, but those that do have identified media use and caffeine as contributors to shorter sleep durations (Owens et al., 2014). Socioeconomic status, neighborhood quality, and sleep hygiene have also been associated with insufficient sleep durations and increased sleep variability (Marco et al., 2012). While informative, Marco et al. (2012) did not assess individual environmental contributors to poor sleep (e.g. bedding comfort) and, thus, specific intervention targets could not be identified. Additionally, little is known about what contributes to poor sleep quality, compared with sleep duration.

This study combined measures of actigraphy with functional connectivity to explore sleep-related alterations in the brain and links to impulsivity in 55 adolescents (14–18 years; 28 female). Given insufficient nighttime sleep is more pervasive for middle and older teens, particularly during the transition to high school (NSF, 2014; Winsler et al., 2015), we focused our age range on high-school adolescents. As this is the first fMRI study to explore the associations between sleep, DMN connectivity and impulsivity in adolescents, we did not proffer *a priori* hypotheses but rather used a data driven approach to explore how these processes interact. Our findings indicate sleep quality and DMN functioning interact to affect adolescent affect-driven impulsivity. We also explored environmental contributors of individual differences in sleep.

2. Methods

2.1. Participants

Data were collected for 59 adolescents (29 female, M_{Age} = 16.31 years, SD = 1.12, range = 14–18 years). Two adolescents were excluded from the fMRI scan due to a metal implant and self-reported attention-deficit hyperactivity disorder (ADHD) diagnosis, respectively. One adolescent taking psychotropic medications and one adolescent whose motion parameters exceeded 2.0 mm were excluded from analyses. Data are presented for 55 adolescents (28 female, M_{Age} = 16.22 years, SD = 1.12, range = 14–18 years), with a median annual family income of $46,500 (range $11,000 to $1,000,000). Ninety-one percent of participants reported post-pubertal status (Petersen et al., 1988). Males and females did not differ as to age (females M_{Age} = 16.25, SD = 1.00, range = 14–18 years; males M_{Age} = 16.20 years, SD = 1.24, range = 14–18 years). Fifty percent of our sample identified as Hispanic/Latino, 23% Caucasian, 10% African American, 7% mixed ethnicity, and 5% reported “other”. All included participants were right-handed, free of metal, and reported no current medical or neurological disorders. Participants completed written consent and assent in accordance with the university’s Institutional Review Board and were compensated for their participation.

2.2. Sleep

Sleep indices were tracked using a Micro Motionlogger® Sleep Watch actigraph by Ambulatory Monitoring, Incorporated (AMI). Each participant was instructed to wear the actigraph device on their non-dominant wrist at night for 14 days. Adolescents’ body movement during nighttime sleep was monitored in 1-min epochs using zero crossing mode. Adolescents were asked to push the event marker button when they turned off the lights to go to sleep and again when they got out of bed in the morning. Adolescent reports of sleep and wake times were collected via daily text messages. The in-bed period began at the time of the first event marker indicating when participants turned off the lights to go to sleep and ended at the time when the participant got out of bed in the morning. If event markers were not available for a particular night, adolescent report was used. Significant discrepancies in adolescent report and the actigraph record were reconciled by discussion among two trained coders using additional indices of sleep onset and offset (e.g., light monitoring and time stamps). Each nightly record was scored using validated AMI algorithms (Action4 software package; Sadeh et al., 1994) for the portion indicated as nighttime sleep (sleep onset to sleep offset). Actigraphy has been validated for use in adolescent populations (Acebo et al., 1999; Sadeh et al., 1994) and as a reliable assessment of sleep quality when compared with polysomnography (Marino et al., 2013; Sadeh et al., 1994).

Data from the actigraph device was used to calculate four sleep indices of interest, *sleep duration*, *sleep efficiency*, *number of awakenings*, and *duration of awakenings*. Sleep duration was calculated by averaging across the 14 days the number minutes of sleep attained each night from the time adolescents fell asleep to the time they awoke the next morning (average number of actigraphy days collected per participant M = 13.69 days, SD = 2.43). Consistent with standard use of sleep duration, this measurement included times when participants experienced awakenings. Sleep efficiency was calculated as the percentage of time spent asleep each night (time asleep/sleep duration), with larger percentages reflecting greater sleep efficiency. Number of awakenings experienced each night were averaged across the 14-day duration of the study. For each night, average duration of the awakenings was