Title

Neural connectivity moderates the association between sleep and impulsivity in adolescents

Authors

Tashjian, Sarah M
Goldenberg, Diane
Galván, Adriana

Publication Date

2017-10-01

DOI

10.1016/j.dcn.2017.07.006

Peer reviewed

Developmental Cognitive Neuroscience 27 (2017) 35–44

Contents lists available at ScienceDirect

Developmental Cognitive Neuroscience

journal homepage: www.elsevier.com/locate/dcn

Neural connectivity moderates the association between sleep and impulsivity in adolescents

Sarah M. Tashjian[a,*], Diane Goldenberg[a], Adriana Galván[a,b,*]

[a] Department of Psychology, University of California, Los Angeles, CA 90095, USA
[b] Brain Research Institute, University of California, Los Angeles, CA 90095, USA

ARTICLE INFO

Keywords:
Actigraphy
Adolescents
Default Mode
fMRI
Impulsivity
Sleep

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

* Corresponding authors at: Department of Psychology, University of California, 1285 Franz Hall, Los Angeles, CA, 90095-1563, USA.
E-mail addresses: smtashjian@ucla.edu (S.M. Tashjian), agalvan@ucla.edu (A. Galván).

http://dx.doi.org/10.1016/j.dcn.2017.07.006
Received 15 May 2017; Received in revised form 19 July 2017; Accepted 24 July 2017
Available online 26 July 2017

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

multiplied by the number of awakenings and then these durations were averaged across the study to calculate a single average of duration of awakenings for each participant.

2.2.1. Sleep environment

Adolescent participants completed a 19-item instrument to assess factors commonly reported as reasons for disrupted sleep. Participants rated 13 items on a 7-point Likert scale ranging from 1 (*not at all*) to 7 (*very much*) addressing six main categories of disturbances: noise level, temperature level, light, sleeping surfaces, sleeping partners, and technology. Composite scores for each category were calculated by summing the ratings for the individual items in that category and dividing by the number of items. Four items were binary yes/no questions and two items were open response. We focus on the 13 rated items.

2.3. Trait impulsivity

Adolescent participants completed the UPPS-P Impulsivity Scale (Cyders et al., 2007), a 59-item inventory designed to measure five distinct features of impulsive behavior: Negative Urgency, Lack of Perseverance, Lack of Premeditation, Sensation Seeking, and Positive Urgency. Negative Urgency refers to the tendency to experience strong impulses under conditions of negative affect. Lack of Perseverance refers to difficulties remaining focused on a task that may be long, boring, or difficult. Lack of Premeditation refers to the tendency to fail to think and reflect on the consequences of an act before engaging in that act. Sensation seeking encompasses two aspects (a) the tendency to enjoy and pursue exciting activities, and (b) an openness to trying new experiences that may or may not be dangerous. Positive urgency refers to the tendency toward rash action in response to a very positive mood. Negative Urgency, Positive Urgency, and Sensation Seeking are thought to comprise affect-driven impulsivity whereas Lack of Perseverance and Lack of Premeditation are thought to comprise cognitive impulsivity. This scale has been used to assess impulsivity in the context of drug and alcohol use (Zapolski et al., 2009), gambling (Del Prete et al., 2017), non-suicidal self-injury (Claes and Muehlenkamp, 2013), and risk taking generally (Billieux et al., 2010).

Each item is rated on a 4-point Likert scale ranging from 1 (*strongly agree*) to 4 (*strongly disagree*). A score of 1 indicates that the participant endorsed a low level of self-reported impulsivity and a score of 4 indicates a high level of self-reported impulsivity. Average total scores were calculated by first calculating the mean for each subscale and then taking the average score across the five subscales. The Cronbach's alphas of the five scales in the present study are as follows: Negative Urgency ($\alpha = 0.80$), Lack of Perseverance ($\alpha = 0.72$), Lack of Premeditation ($\alpha = 0.80$), Sensation Seeking ($\alpha = 0.73$), and Positive Urgency ($\alpha = 0.92$).

2.4. fMRI paradigm

Approximately two weeks after the initial study visit and at the conclusion of actigraphy data collection, participants completed an fMRI scan.

2.4.1. State impulsivity task

Participants completed a Go/No-Go (GNG) task to examine neural correlates of response inhibition (Fig. 1). Participants were presented with a series of rapid trials (.35 s each), each displaying a single white letter on a black background. Participants were instructed to respond with a button press as quickly as possible to all letters (go) except for "X" (no-go). Correct "go" trials were classified as hits, incorrect "go" trials were classified as misses, correct "no-go" trials were classified as inhibitions, and incorrect "no-go" trials were classified as false alarms. Impulse control was assessed using percentage of correct inhibitions on the GNG task whereas impulsivity was assessed using percentage of false alarms and reaction time for false alarms.

The X occurred on 50% of trials, which allowed for maximum uncertainty regarding the stimulus probability, created comparability for the number of go and no-go trials, and reduced the possibility that some the observed activation differences between the go and the no-go trials was due to different frequencies of the stimuli. Each run contained 48 trials presented in random order. The intertrial interval (ITI) was jittered according to a random gamma distribution ($M = 4.27$ s). Participants completed two functional runs and each run (48 trials and ITIs) lasted 4.02 min.

2.4.2. fMRI data acquisition

The scan was conducted on a Siemens 3T TIM Trio MRI scanner with a 32-channel head coil. Parameters for image acquisition were voxel size = 3.0 × 3.0 × 4.0 mm, slices = 34, slice thickness = 4.0 mm, repetition time = 2000 ms, echo time = 30 ms, flip angle = 90°, interleaved slice geometry, field of view = 192 mm, 118 vol. Preprocessing was conducted using FEAT (FMRI Expert Analysis Tool) version 6.00, part of FSL (FMRIB Software Library, www.fmrib.ox.ac.uk/fsl, RRID:SCR_002823). Preprocessing consisted of non-brain removal using BET, high-pass filtering (100-s cutoff), and spatial smoothing using a Gaussian kernel of FWHM 5 mm. Rigid body motion correction with six degrees of freedom was performed using MCFLIRT. A T2*weighted, matched bandwidth (MBW), high-resolution, anatomical scan and magnetization-prepared rapid-acquisition gradient echo (MPRAGE) scan were acquired for registration purposes (TR: 1900 ms; TE 2.26 ms; FOV: 250 mm; slice thickness: 1 mm; 176 slices). Each participant's functional data were registered to their MBW, then to the MPRAGE, and finally to MNI (Montreal Neurological Institute) stereotaxic space with 12 ° of freedom using FSL's registration method via FLIRT. Alignment was visually confirmed for all participants.

2.4.3. fMRI data analysis

We conducted Psychophysiological interactions (PPI) analyses (Friston et al., 1997) to examine whether sleep quality affected functional coupling between the DMN and other brain regions. The DMN is known to be more active during baseline than during task and successful suppression of the DMN is important for goal-oriented task performance (Anticevic et al., 2012; Satterthwaite et al., 2013). To capture activation in the DMN, we used a DMN mask defined independently from the current fMRI data and created in MNI space (Fig. 2; Shirer et al., 2012). This mask has been widely-used as an ROI and for template matching in adolescents (e.g., Lee et al., 2017; van Belle et al., 2015). We selected an independently defined mask following the recommendations of Kriegeskorte et al. (2009). The mask consisted of 9 functionally defined regions of interest (ROIs) including the posterior cingulate cortex/precuneus (Brodmann Areas (BA) 23, 30), medial prefrontal cortex (mPFC)/anterior cingulate cortex/orbitofrontal cortex (BA 9, 10, 24, 32, 11), bilateral angular gyrus (BA 39), bilateral hippocampus (BA 20, 36, 30), bilateral thalamus (BA N/A), bilateral angular gyrus (BA 39), midcingulate cortex (BA 23), right superior frontal gyrus (BA 9). Functionally defined ROIs have been found to outperform commonly used structural ROIs in classifying cognitive states across large-scale resting-state brain networks including the DMN (Shirer et al., 2012). This standard-space mask was then transformed to individual functional space using FLIRT, and the average time course of all voxels within the individual's mask were extracted using *fslmeants*. Using an adolescent specific mask from Sherman et al. (2014), we replicated these analyses (Supplemental Fig. S1).

At the individual level, one general linear model (GLM) was defined for each run of the GNG task. The GLM included multiple regressors for each event type: successful go trials (hits), successful no-go trials (inhibitions), unsuccessful no-go trials (false alarms), unsuccessful go trials (misses). Events were modeled with a with a canonical (double-gamma) hemodynamic response function for a 1 s duration to allow for motor

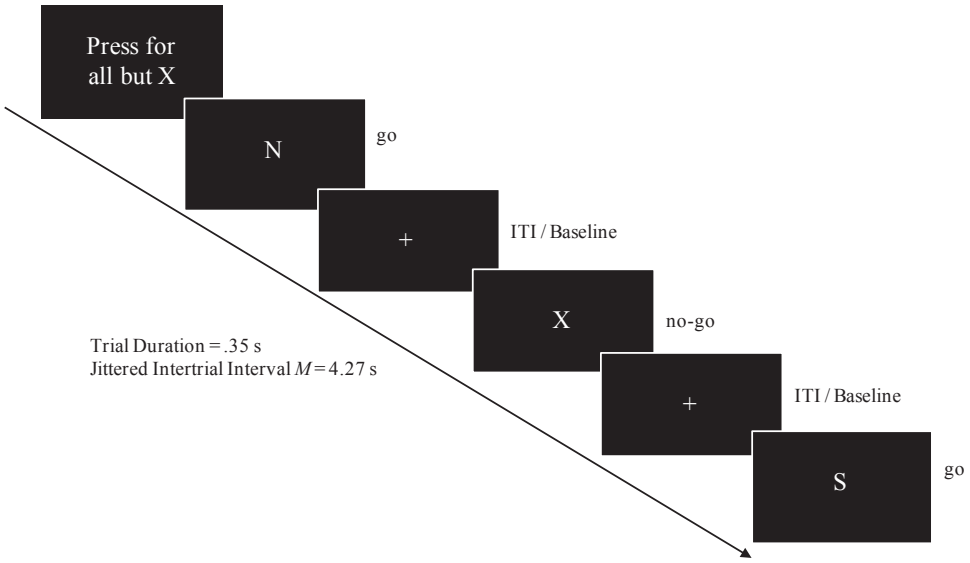

Fig. 1. The go/no-go task. Participants were instructed to press the button as quickly as possible for all letters ("go" trials) except the letter "X" ("no-go" trials). No-go trials occurred randomly, 50% of the time.

response after offset of the rapid-presentation visual stimuli. The jittered ITIs were not explicitly modeled and therefore served as the baseline of interest. Temporal derivatives and motion parameters were included as covariates of no interest for all regressors. Due to a task programming error, participant responses for some events were not recorded. The occurrence of these error trials was not systematic and these trials were modeled as discarded trials of no interest. The time-series extracted from the DMN mask and the product between the DMN timeseries (physical regressor) and the contrast of baseline − inhibitions (psychological regressor) was added to each participant's first-level GLM design matrix. The psychological regressor was zero-centered and the physical regressor was demeaned. This interaction term identified regions that covaried in a task-dependent manner with the DMN. The two runs for each participant were combined using a fixed effects voxel-wise analysis at the second level.

Two group-level analyses were performed using the FMRIB Local Analysis of Mixed Effects module in FSL (Beckmann et al., 2003). Demeaned regression scores for the sleep quality component were entered as a regressor in one whole brain regression analysis and demeaned sleep duration scores in the second analysis. Thresholded Z statistic images were prepared to show clusters determined by a corrected, cluster-forming threshold of $Z > 2.3$ and an extent threshold of $p < 0.05$ familywise error corrected using the Theory of Gaussian Random Fields (Poline et al., 1997). Outliers were de-weighted in the multi-subject statistics using mixture modeling (Woolrich, 2008). Activation maps were visualized using MRIcron software (http://www.sph.sc.edu/comd/rorden/mricron/). Statistical maps of all analyses were projected onto a standard MNI brain.

2.5. Moderation

Simple moderation (Model 1) was performed using Hayes' PROCESS macro for SPSS (Hayes, 2013). Analyses utilized a bootstrapping approach with 5000 samples, and significance was determined at 95% bias-corrected confidence intervals. All variables were continuous and centered prior to analysis, and the estimated effects are reported as unstandardized regression coefficients. When depicting significant interactions, the low value of the moderator is calculated as 1 *SD* below the mean, and the high value is 1 *SD* above the mean, consistent with procedures outlined by Aiken and West (1991).

3. Results

3.1. Sleep findings

We collected actigraph data to assess four metrics of adolescent sleep over two weeks: sleep duration, sleep efficiency (time asleep/sleep duration), number of nighttime awakenings, and duration of

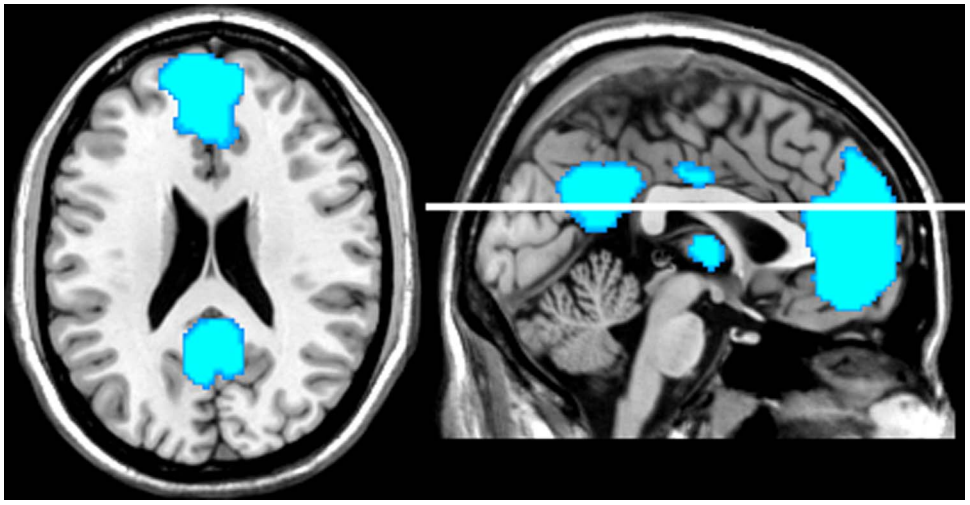

Fig. 2. DMN mask created in MNI space based on Shirer et al. (2012).

Table 1
Descriptives for sleep variables of interest, $N = 55$.

	Average sleep duration (min)	Average sleep efficiency (%)	Average number of awakenings	Average duration of awakenings (min)
M	418.98 (6.98 h)	92.06	5.56	21.03
SD	43.89	5.72	3.85	20.03
Range	335.05–518.00	72.52–98.81	0.62–22.93	0.69–96.70

nighttime awakenings (Table 1).

Adolescents attained an average of 418.98 min (6.98 h) of sleep per night, including both weekdays and weekends for the duration of the study. On weekdays, they went to bed at 11:49 p.m. on average (range 9:30 p.m.–3:42 a.m.) and woke up at 7:14 a.m. on average (5:15 a.m.–10:00 a.m.). On weekends, adolescents went to bed at 12:34 a.m. on average (range 11:10 p.m.–3:38 a.m.) and woke up at 8:44 a.m. on average (range 6:25 a.m.–11:38 a.m.). On average, participants slept 37.39 min longer (*SD* 67.41) on weekends than on weekdays, had 1.15 (*SD* 3.76) more awakenings, and duration of awakenings were 2.20 min (*SD* 19.70) longer on weekends compared to weekdays. We did not obtain any weekend information from two participants. Independent samples *t*-tests revealed significant sex differences for all sleep variables (sleep duration, $t(53) = -3.01$, $p = 0.004$; sleep efficiency, $t(53) = -2.55$, $p = 0.01$; number of awakenings, $t(53) = 2.09$, $p = 0.04$) except duration of awakenings ($t(53) = 1.93$, $p = 0.06$), such that females attained better sleep (e.g., longer duration, less awakenings) than males.

3.1.1. Principal component analysis

To avoid multiple comparisons, sleep efficiency, number of awakenings, and duration of awakenings were reduced using principal component analysis (PCA). Sleep duration was not correlated with the other variables of interest (sleep efficiency $r(55) = 0.22$, $p = 0.10$; number of awakenings $r(55) = -0.02$, $p = 0.91$; duration of awakenings $r(55) = -0.02$, $p = 0.90$) and thus was not included in the PCA analysis. For sleep efficiency, number of awakenings, and duration of awakenings, several well-recognized criteria for using PCA were assessed. First, the items were highly correlated (sleep efficiency and number of awakenings $r(55) = -0.51$, $p < 0.001$; sleep efficiency and duration of awakenings $r(55) = -0.89$, $p < 0.001$; number of awakenings and duration of awakenings $r(55) = 0.68$, $p < 0.001$). The Kaiser-Meyer-Olkin measure of sampling adequacy was 0.57, slightly above the commonly recommended value of 0.50, and Bartlett's test of sphericity was significant ($\chi^2(3) = 118.19$, $p < 0.001$). The diagonals of the anti-image correlation matrix were all over 0.5. Given these indicators, PCA was deemed to be suitable. PCA with variance maximizing (varimax) rotation was used to fulfill the primary aim of identifying and computing composite scores for the components underlying these three sleep metrics. Components were extracted for factors with eigenvalues greater than 1. One component, henceforth identified as poor sleep quality, emerged explaining 79.97% of the variance. All items met a minimum criterion of having a primary factor loading of 0.5 or above and no item had a cross loading of 0.3 or above. Overall, these analyses indicated that one distinct component was underlying adolescent sleep efficiency, number of awakenings, and duration of awakenings. Given the factor loadings, sleep efficiency (−0.91), number of awakenings (0.80), and duration of awakenings (0.97), we interpreted this component to represent poor sleep quality. Higher scores on the sleep quality component indicated worse sleep quality (more disruption, less efficiency). Regression scores for this component were calculated and used as demeaned explanatory variables in fMRI analyses.

3.1.2. Sleep environment and sleep quality

Although we suspected that sleep environment would influence sleep quality, the literature relating to causes of poor sleep quality is sparse. The bulk of prior work focuses on sleep duration (Keyes et al., 2015), self-reported sleep quality (LeBourgeois et al., 2005), or focused assessments of environmental factors like caffeine (Orbeta et al., 2006). Thus, we conducted exploratory preliminary analyses to probe variables that contributed to sleep quality using a questionnaire that assesses the participant's sleep environment. The questionnaire assessed six environmental factors: noise level, temperature level, light, sleeping surfaces, sleeping partners, and technology. We created composite scores of questions included in each of these categories to determine what factors related to sleep quality (Table 2). Ratings of pillow comfort ($r(52) = -0.36$, $p = 0.01$) and noisy room/bed-mates ($r(16) = 0.51$, $p = 0.04$) were significantly correlated with the sleep quality component such that less comfortable pillows and noisier room/bed-mates related to poorer sleep quality. Sleep deficiencies have been found to increase in late adolescence (Owens et al., 2014), socioeconomic status has been linked to worse sleep (Bagley et al., 2015), and females in our sample achieved better sleep than males. Thus, we tested multiple linear regression models to examine whether pillow comfort uniquely related to poorer sleep quality over and above age, sex, and household income. In the first model, we analyzed whether participant age, sex, and household income were associated with sleep quality. The model was not significant, $F(3, 43) = 2.10$, $p = 0.11$. Pillow comfort was added to the first model, and the second model was significant, $F(4, 42) = 3.25$, $p = 0.02$. Pillow comfort was significantly related to sleep quality over and above age, sex, and household income, $\beta = -0.34$, $t(42) = -2.45$, $p = 0.02$, and the full model accounted for 24% of the

Table 2
Descriptive information for rated sleep environment variables and composite scores.

	Noise Level			Temperature Level		Light	Sleeping Surfaces		Sleeping Partners		Technology Use		
	Room noisy	Home noisy	Neighborhood noisy	Summer comfort	Winter comfort	Bright lights in room	Bed comfort	Pillow comfort	Enough space	Noisy room/ bed-mate	TV before bed	Phone before bed	Computer before bed
M	1.77	2.58	2.23	4.40	5.35	1.85	6.08	5.88	5.55	2.44	3.35	6.06	3.81
SD	1.19	1.55	1.51	1.91	1.93	1.66	1.34	1.55	2.39	1.93	2.39	1.53	2.30
Range	1–5	1–6	1–7	1–7	1–7	1–7	1–7	1–7	1–7	1–7	1–7	1–7	1–7
N	53	53	53	47	48	46	52	52	20	16	31	31	31
Composite *M*		2.20		4.87		N/A	5.98		3.84		4.41		
Composite *SD*		1.09		1.49		N/A	1.35		1.26		1.48		
Composite Range		1–5		1.5–7		N/A	1–7		1–6.5		1.67–7		
Composite *N*		53		47		N/A	52		16		31		

Note: Items were rated on a scale of 1 = *not at all*, to 7 = *very much*. Composite scores were calculated by summing the ratings for the individual items in each category and dividing by the number of items.

variance in sleep quality. Only 16 participants had room/bed-mates and thus we did not have sufficient power to test linear regression models with this factor.

3.1.3. Sleep environment and sleep duration

Sleep duration is the most commonly studied variable but is often imprecisely measured because as typically assessed, it does not account for times when individuals awaken during the night. Thus, the metric does not account for differences in sleep quality, which may be more significantly associated with neural development during adolescence (Telzer et al., 2013). In our sample, age and sex were significantly related to sleep duration such that younger adolescents and females obtained longer nightly durations (age, $r(55) = -0.28$, $p = 0.04$; sex, $r(55) = 0.38$, $p = 0.004$). Pillow comfort ($r(52) = 0.08$, $p = 0.55$) and noisy room/bed-mates ($r(16) = -0.31$, $p = 0.24$) did not significantly relate to sleep duration. Ratings of home noise significantly correlated with sleep duration such that noisier homes related to shorter sleep durations ($r(53) = -0.28$, $p = 0.04$). Sleep duration did not relate to sleep quality ($r(55) = -0.10$, $p = 0.48$).

3.2. Impulsivity results

Participants completed a GNG task to examine neural correlates of response inhibition (Fig. 1, Supplemental Fig. S2). They successfully inhibited 72% ($SD = 0.16$) of the no-go trials (i.e., withheld the button press to the "X" stimuli), ranging from 29% to 100%. Participants successfully hit 96% ($SD = 0.08$) of the go trials (i.e., pressed the button to all non-"X" stimuli), ranging from 50% to 100%. Percent of correct inhibitions was significantly correlated with false alarm reaction time ($r(51) = 0.54$, $p < 0.001$) such that greater percent of correct inhibitions related to slower reaction time on false alarms. Contrary to our expectations, no significant associations were found between sleep quality or sleep duration and GNG task performance (RT or success rate).

Participants also completed the UPPS-P Impulsive Behavior Scale (Cyders et al., 2007), a 59-item inventory designed to measure five distinct features of trait-level impulsive behavior: Negative Urgency, Lack of Perseverance, Lack of Premeditation, Sensation Seeking, and Positive Urgency. The Negative Urgency ($r(44) = -0.43$, $p = 0.004$) and Positive Urgency ($r(48) = -0.38$, $p = 0.008$) subscales were negatively correlated with successful inhibitions when controlling for multiple comparisons with the five subscales ($p = 0.05/5$ subscales $= 0.01$) (Fig. 3). Reaction time on the GNG task was not correlated with any UPPS-P subscales.

3.3. Connectivity findings

PPI is a measure of task-specific increases in functional connectivity between different brain regions. We conducted PPI analyses (Friston et al., 1997) to examine whether sleep quality and/or sleep duration affected functional coupling between the DMN and other brain regions. To capture activation in the DMN, we used a DMN mask defined independently from the current fMRI data and created in MNI space (Shirer et al., 2012; Fig. 2). Sleep quality was associated with significant differences in connectivity between the DMN and lateral prefrontal regions during baseline compared to inhibitions (Table 3, Fig. 4). Poorer sleep quality positively correlated with strength of connectivity between the DMN and the PFC, namely the ventrolateral PFC (vlPFC), and dorsolateral PFC (DLPFC). The same results were observed using the Sherman et al. (2014) mask (Supplemental Fig. S3). When age and sex were included as covariates, the results remained the same.

For descriptive purposes, we plotted the association between the sleep quality component and connectivity between the DMN and PFC regions identified as significant clusters (Fig. 5). Sleep duration did not significantly relate to any differential DMN connectivity.

Table 3
Non-DMN regions that had greater connectivity with the DMN during baseline compared to inhibition in adolescents with poorer sleep quality.

Region label	R/L	Peak MNI coordinates			Max	Voxels
		x	*y*	*z*	*Z*-value	(mm^3)
Orbitofrontal cortex	L	−26	68	10	4.53	661
Dorsolateral prefrontal cortex	L	−40	54	22	4.05	
Ventrolateral prefrontal cortex	L	−46	46	22	3.57	

Note: *x*, *y*, and *z* refer to MNI coordinates; Voxels refer to each significant cluster; L and R refer to left and right hemispheres. All regions are significant at $Z = 2.3$, $p < 0.05$, cluster corrected.

3.3.1. Neural connectivity and impulsivity

Given the PPI results, we posited that greater DMN-PFC coupling was acting as a compensatory mechanism for those with poorer sleep quality. We analyzed links between DMN-PFC connectivity, sleep quality and the trait-like measure of impulsivity as assessed by the UPPS-P. Moderation results revealed a significant interaction effect of sleep quality and DMN-PFC connectivity values on UPPS-P average scores (Supplemental Fig. S4). Simple slopes analyses revealed that poorer sleep quality was associated with higher scores on the UPPS-P measure for those low in DMN-PFC connectivity but unrelated to those at average or high DMN-PFC connectivity. We next investigated whether this effect was driven by a particular UPPS-P subscale given the multidimensional nature of impulsivity captured by this measure. The Negative Urgency subscale was most robustly related to behavioral performance on the GNG task and was thus our first exploration. Forty-four participants provided complete responses to the UPPS-P Negative Urgency items. Negative Urgency measures an individual's tendency to

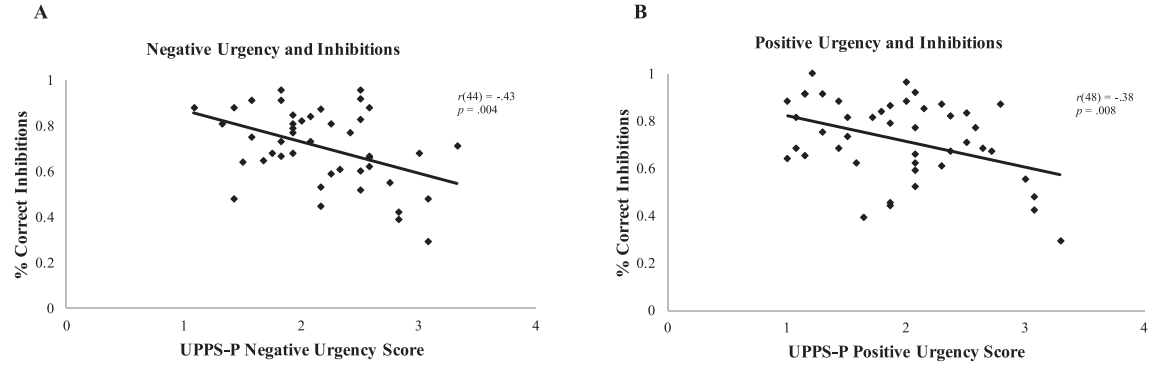

Fig. 3. Significant correlations between UPPS-P subscales and inhibition performance. Inhibition scores are percentage of correct response to no-go trials during the GNG task. Negative Urgency (a) and Positive Urgency (b) significantly correlated with no-go performance.

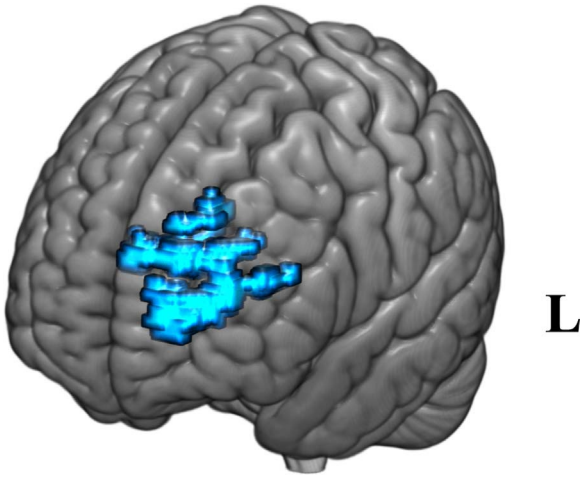

Fig. 4. Regions that had significantly greater connectivity with the DMN during baseline compared to inhibition in adolescents with poorer sleep quality. All analyses cluster-corrected at $Z = 2.3$, $p < 0.05$.

act impulsively when experiencing negative affect (e.g., "I have trouble resisting my cravings (for food, cigarettes, etc.)"; "In the heat of an argument, I will often say things that I later regret"). Scores on the Negative Urgency subscale ranged from 1.08 to 3.33, $M = 2.18$, $SD = 0.52$. Data was normally distributed with skewness of 0.12 ($SE = 0.36$) and kurtosis of −0.50 ($SE = 0.70$). Moderation results revealed a significant interaction effect of sleep quality and DMN-PFC connectivity values on Negative Urgency scores (Fig. 6). Worse sleep quality was associated with greater Negative Urgency scores for individuals low (-1SD) in DMN-PFC connectivity, $b = 0.30$, $t(40) = 2.29$, $p = 0.03$, but sleep quality was unrelated to Negative Urgency at average, $b = 0.16$, $t(40) = 1.63$, $p = 0.11$ or high (+1SD) levels of DMN-PFC connectivity, $b = 0.03$, $t(40) = 0.28$, $p = 0.78$. There were also non-significant trends for Positive Urgency (interaction p = 0.14) and Lack of Premeditation (interaction p = 0.12), but no trends for Lack of Perseverance or Sensation Seeking. Similarly, we observed a non-significant trend for false-alarm reaction time on the GNG task (interaction p = 0.07), but not percent of correct inhibitions.

4. Discussion

The goal of this study was to determine the role of normative differences in adolescent sleep and ongoing neural network development in adolescent impulsivity. In addition to examining sleep duration, we probed the quality of adolescents' sleep as a key factor in this complex brain-behavior interaction. We discovered that only sleep quality, not duration, influenced how the DMN functioned in the developing adolescent brain. Connectivity analyses revealed that the DMN was positively coupled with the left prefrontal cortex at baseline for individuals who experienced poorer sleep quality. Furthermore, the interaction between DMN-PFC coupling and sleep quality was significantly related to trait levels of impulsive behavior (average UPPS-P scores and Negative Urgency subscale). Although we did not have *a priori* hypotheses related to the direction of the sleep-DMN relation, further probing of the data suggested that individual differences in sleep-related DMN-PFC connectivity buffered against impulsivity; adolescents who demonstrated stronger connectivity did not show a significant relation between sleep quality and affect-related impulsivity, but adolescents with weaker connectivity showed a positive relation between poorer sleep quality and greater impulsivity. In other words, sleep quality related to trait level affect-related impulsive behavior for individuals with low connectivity, but not for those with moderate or high connectivity. These data suggest differences in how neural networks flexibly interact with the environmental stressor of disrupted sleep have important behavioral manifestations for adolescents.

Although prior research has linked sleep and impulsivity in adolescence, little of this work has sought to explain adolescent behavior in terms of naturalistic differences in sleep quality. Instead, the bulk of prior sleep research focuses on experimental sleep restriction or sleep disorders (Beebe, 2011), failing to extrapolate to the realistic patterns of adolescent sleep and the relation to brain development. A major innovation of this study is the use of naturalistic sleep differences in healthy adolescents, which tells us far more about the individual sleep-related differences in adolescent behavior than experimental manipulations of sleep. Additionally, even less is known about sleep-related differences in neural network functioning. The few neuroimaging studies instead focus on exacerbated mesolimbic-prefrontal developmental imbalances (Telzer et al., 2013), leaving network functioning unexplored. The present study provides a novel perspective regarding the question of adolescent impulsivity and identified intrinsic processes of sleep quality and DMN connectivity as biomarkers of individual differences in behavior.

Importantly, DMN connectivity was not affected by sleep duration, which dominates scientific and public health attention (NSF, 2014; Owens et al., 2014). Our data suggest that duration may not be the most informative metric by which to investigate neural development or

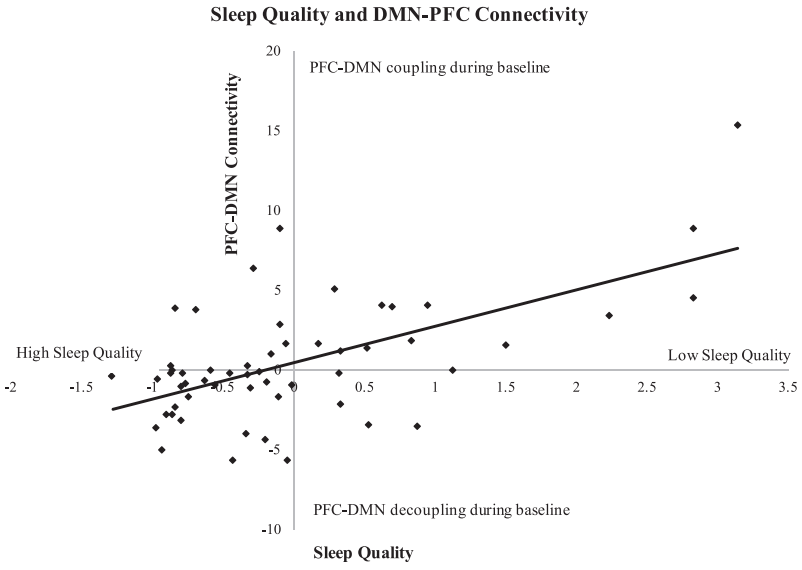

Fig. 5. Scatter plot showing a visual depiction of the relation between sleep quality and DMN-PFC connectivity for the contrast of baseline > inhibitions using peak voxel activation (x = −26, y = 68, z = 10). $N = 55$.

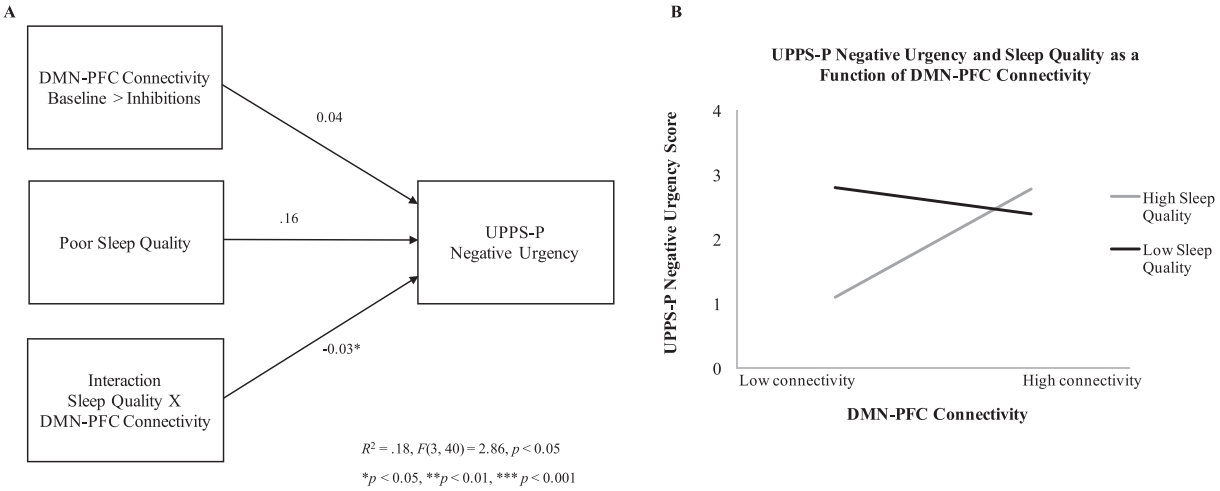

Fig. 6. DMN-PFC connectivity significantly moderated the link between sleep quality and Negative Urgency scores. $N = 44$. (A) Simple moderation (Model 1) was performed using Hayes' PROCESS macro for SPSS (Hayes, 2013), utilizing a bootstrapping approach with 5000 samples, and significance was determined at 95% bias-corrected confidence intervals. All variables were continuous and centered prior to analysis, and the estimated effects are reported as unstandardized regression coefficients. Sleep quality was the predictor variable and UPPS-P Negative Urgency score was the outcome variable. DMN-PFC connectivity was tested as a moderator. The model demonstrates a significant interaction effect of sleep quality and DMN-PFC connectivity on Negative Urgency scores. (B) Simple slopes analyses showing that poorer sleep quality was associated with greater Negative Urgency for those low in DMN-PFC connectivity, but sleep quality was unrelated to Negative Urgency at average and high levels of DMN-PFC connectivity. The low value of the moderator (connectivity) is calculated as 1 SD below the mean, and the high value is 1 SD above the mean, consistent with procedures outlined by Aiken and West (1991).

impulsivity during adolescence. The current study calls for additional research investigating the influence of sleep quality on adolescent neural development as well as the need for more nuanced measures of sleep that better approximate actual sleep experiences of developing youth. The sleep quality component in the present study included micro-awakenings that went undetected by the participants. These disruptions are not captured in self-report measures or measures of duration, but, as these data suggest, they have important implications for neural and behavioral functioning. As research expands to consider sleep quality, micro-awakenings should be explored further.

Although we assessed impulsivity in different domains, including trait and state measures, the sleep and connectivity interaction was driven by the Negative Urgency subscale of the UPPS-P impulsivity measure. This subscale is considered to be a representation of affect-driven impulsivity because it is defined as the tendency to act impulsively when distressed or under negative affect. Negative Urgency has been linked to risky sexual behavior (Deckman and DeWall, 2011), smoking in preadolescents, and drug use, drinking, and conduct disorder in young adults (Settles et al., 2012). These are all behaviors that increase during adolescence (Kann et al., 2016). Further, sleep-deprivation research suggests that poor sleep has a strong association with emotional responsivity and as such may be more acutely detectable when evaluating affect-related impulsivity. Although we cannot delineate directionality of our results and it is possible that negative urgency may be leading to poorer sleep quality rather than poor sleep quality leading to altered brain connectivity and behavior, this is a normative and healthy sample of adolescents with no diagnoses of psychopathology or clinical sleep issues. Thus, we interpret our findings to demonstrate that PFC-DMN connectivity may down-regulate affect-related impulsive behavioral proclivities in sleep disrupted teens.

Prior work has identified alterations in PFC activation after sleep deprivation in adults (Drummond et al., 2000), such that the PFC exhibited greater activation to cognitive demands after a single night of sleep deprivation compared to normal sleep. These results suggest the brain can dynamically respond to sleep loss with increased cerebral activation to better meet cognitive demands. This type of cortical compensation has been investigated in direct relation to neural activation during cognitive activity. Our results suggest that these mechanisms also act on broader network circuitry and, as such, may provide a more flexible buffer to ongoing environmental stressors like disrupted sleep. Flexible hubs are brain regions such as the DLPFC that quickly shift their functional connectivity patterns to implement cognitive control (Cole et al., 2013). Flexible hubs implement cognitive control by redirecting information flow across large-scale functional networks (Miller and Cohen, 2001). We extended this framework to demonstrate that lateralized prefrontal regions like the DLPFC flexibly modulate DMN activity. Our results indicate that this modulation relates to real-world behavior.

DMN-PFC coupling was observed during baseline, which was interspersed between inhibition trials. The long baseline trials allowed us to reliably capture DMN activation but also likely resulted in the participant planning for the next active trial. The coupling we observed may not only buffer against real-world impulsivity, but might also indicate that adolescents with poorer sleep quality needed to engage additional cognitive resources to achieve successful performance on the laboratory GNG task. Connectivity was not related to state level impulsivity as assessed by GNG task performance. However, task performance was related to Negative Urgency and Positive Urgency scores suggesting future work should explore the consistency between trait and state levels of impulsive behavior using a variety of tasks. Prior work has similarly found stronger links between brain activation during GNG tasks and trait measures of impulsivity compared with task performance (Ding et al., 2014). Our lack of significant task behavior may be due to the fact that we used a simple design with equiprobable go/no-go signals and stable response association across the task resulting in adolescents performing well on the task ($M = 72\%$ successful inhibitions). Thus, the task may not have been complex enough to evoke sleep-related inhibitory failures. Given that performance typically deteriorates as task difficulty increases (Eigsti et al., 2006), it is possible that the brain is able to compensate for the effects of sleep quality during less-taxing tasks. By using a task that does not involve strenuous cognitive performance, we were able to observe this compensatory neural mechanism despite no task behavioral differences. Future work should also explore differences between cognitive and emotional GNG tasks given our finding that sleep quality was specifically associated with affect-driven trait impulsivity.

The present findings add to our growing understanding of what has been recently called "an adolescent sleep epidemic". Not only are adolescents obtaining insufficient sleep in terms of recommended sleep duration, but the adolescents in our sample also experienced a high

number of nightly sleep awakenings that in some cases disrupted sleep for up to 97 min in a single night. Importantly, we propose that pillow comfort may be an easy target for remedying this problem. Given our findings that sleep quality was reflected in adolescent neural functioning, identifying possible interventions, particularly those as simple and cost-effective as comfortable pillows, is imperative for efforts to improve adolescent outcomes. Environmental interventions aimed at improving sleep could have far-reaching implications for improving academic, health, and psychological outcomes, particularly for teens of low-socioeconomic status who often experience poorer sleeping conditions (Bagley et al., 2015).

Interpretation of the current findings should be considered in the context of potential limitations of the study. To probe sleep-related changes in DMN functioning, we used a GNG task for which adolescents exhibit high competence. Future work should consider whether the PFC-DMN connectivity observed in this study is consistent across different cognitive tasks of varying difficulty. The relation between sleep, impulsivity and connectivity was driven by the Negative Urgency subscale; other subscales on the UPPS-P only showed a trend or non-significance. It is possible that given the small sample size due to the nature of fMRI we lacked sufficient power to detect significant results in the other subscales. Replication of these findings in larger samples will continue to expand our understanding of how sleep influences adolescent brain development and behavior.

By approaching the question of adolescent impulsivity from a new perspective, one that integrates the basic biological process of sleep, this study provides new evidence that the development of PFC-DMN coupling may serve a protective factor to diminish impulsivity. These results add to the timely discussion of the adolescent sleep epidemic by drawing attention to the complex interplay between sleep-altered neural systems and impulsive behavior. Our findings also raise novel insights for examining sleep and impulsivity and have implications for potentially simple and significant interventions.

Funding

Support for this study was provided by a Scholars Grant from the William T. Grant Foundation and the Jeffrey/Wenzel Chair in Behavioral Neuroscience to Dr. Adriana Galván. Preparation of this manuscript was supported in part by the National Science Foundation Graduate Fellowship to Sarah M. Tashjian.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.dcn.2017.07.006.

References

Acebo, C., Sadeh, A., Seifer, R., Tzischinsky, O., Wolfson, A.R., Hafer, A., Carskadon, M.A., 1999. Estimating sleep patterns with activity monitoring in children and adolescents: how many nights are necessary for reliable measures? Sleep 22, 95–103.
Aiken, L.S., West, S.G., 1991. Multiple Regression: Testing and Interpreting Interactions. Sage, Newbury Park, CA.
Anderson, C., Platten, C.R., 2011. Sleep deprivation lowers inhibition and enhances impulsivity to negative stimuli. Behav. Brain Res. 217, 463–466.
Anticevic, A., Cole, M.W., Murray, J.D., Corlett, P.R., Wang, X.J., Krystal, J.H., 2012. The role of default network activation in cognition and disease. Trends Cogn. Sci. 16, 584–592.
Bagley, E.J., Kelly, R.J., Buckhalt, J.A., El-Sheikh, M., 2015. What keeps low-SES children from sleeping well: the role of presleep worries and sleep environment. Sleep Med. 16, 496–502.
Beckmann, C.F., Jenkinson, M., Smith, S.M., 2003. General multilevel linear modeling for group analysis in fMRI. Neuroimage 20, 1052–1063.
Beebe, D.W., 2011. Cognitive, behavioral, and functional consequences of inadequate sleep in children and adolescents. Pediatr. Clin. North Am. 58, 649–665.
Billieux, J., Gay, P., Rochat, L., Van der Liden, M., 2010. The role of urgency and its underlying psychological mechanisms in problematic behaviours. Behav. Res. Ther. 48, 1085–1096.
Broyd, S.J., Demanuele, C., Debener, S., Helps, S.K., James, C.J., Sonuga-Barke, E.J., 2009. Default-mode brain dysfunction in mental disorders: a systematic review. Neurosci. Biobehav. Rev. 33, 279–296.
Chee, M.W., Choo, W.C., 2004. Functional imaging of working memory after 24 h of total sleep deprivation. J. Neurosci. 24, 4560–4567.
Claes, L., Muehlenkamp, J., 2013. The relationship between the UPPS-P impulsivity dimensions and nonsuicidal self-injury characteristics in male and female high-school students. Psychiatry J. 2013, 1–5.
Cole, M.W., Laurent, P., Stocco, A., 2013. Rapid instructed task learning: a new window into the human brain's unique capacity for flexible cognitive control. Cogn. Affect. Behav. Neurosci. 13, 1–22.
Cyders, M.A., Smith, G.T., Spillane, N.S., Fischer, S., Annus, A.M., Peterson, C., 2007. Integration of impulsivity and positive mood to predict risky behavior: development and validation of a measure of positive urgency. Psychol. Assess. 19, 107–118.
De Havas, J.A., Parimal, S., Soon, C.S., Chee, M.W.L., 2012. Sleep deprivation reduces default mode network connectivity and anti-correlations during rest and task performance. Neuroimage 59, 1745–1751.
Deckman, T., DeWall, C.N., 2011. Negative urgency and risky sexual behaviors: a clarification of the relationship between impulsivity and risky sexual behavior. Personal. Individ. Differ. 51, 674–678.
Del Prete, F., Steward, T., Navas, J.F., Fernández-Aranda, F., Jiménez-Murcia, S., Oei, T.P., Perales, J.C., 2017. The role of affect-driven impulsivity in gambling cognitions: a convenience-sample study with a Spanish version of the Gambling-Related Cognitions Scale. J. Behav. Addict. 6, 51–63.
Ding, W., Sun, J., Sun, Y., Chen, X., Zhou, Y., Zhuang, Z., Li, L., Zhang, Y., Zu, J., Du, Y., 2014. Trait impulsivity and impaired prefrontal impulse inhibition function in adolescents with internet gaming addiction revealed by a Go/No-Go fMRI study. Behav. Brain Funct. 10, 1–9.
Drummond, S.P.A., Brown, G.G., Gillin, J.C., Stricker, J.L., Wong, E.C., Buxton, R.B., 2000. Altered brain response to verbal learning following sleep deprivation. Nature 403, 655–657.
Drummond, S.P., Paulus, M.P., Tapert, S.F., 2006. Effects of two nights sleep deprivation and two nights recovery sleep on response inhibition. J. Sleep Res. 15, 261–265.
Eigsti, I.M., Zayas, V., Mischel, W., Shoda, Y., Ayduk, O., Dadlani, M.B., Davidson, M.C., Lawrence, A.J., Casey, B.J., 2006. Predicting cognitive control from preschool to late adolescence and young adulthood. Psychol. Sci. 17, 478–484.
Fair, D.A., Cohen, A.L., Dosenbach, N.U., Church, J.A., Miezin, F.M., Barch, D.M., et al., 2008. The maturing architecture of the brain's default network. Proc. Natl. Acad. Sci. U. S. A. 105, 4028–4032.
Fox, M.D., Raichle, M.E., 2007. Spontaneous fluctuations in brain activity observed with functional magnetic resonance imaging. Nat. Rev. Neurosci. 8, 700–711.
Friston, K.J., Buechel, C., Fink, G.R., Morris, J., Rolls, E., Dolan, R.J., 1997. Psychophysiological and modulatory interactions in neuroimaging. Neuroimage 6, 218–229.
Green, P., 2017. Sleep is the New Status Symbol. New York Times. April 8, p. ST1. Retrieved from: https://www.nytimes.com/2017/04/08/fashion/sleep-tips-and-tools.html.
Hagenauer, M.H., Perryman, J.I., Lee, T.M., Carskadon, M.A., 2009. Adolescent changes in the homeostatic and circadian regulation of sleep. Dev. Neurosci. 31, 276–284.
Hamza, C.A., Willoughby, T., Heffer, T., 2015. Impulsivity and nonsuicidal self-injury: a review and meta-analysis. Clin. Psychol. Rev. 38, 13–24.
Hayes, A.F., 2013. Introduction to Mediation, Moderation, and Conditional Process Analysis. A Regression-Based Approach. Guilford Press, New York, NY.
Heron, M., 2016. Deaths: leading causes for 2014. National Vital Statistics Reports 65. National Center for Health Statistics, Hyattsville, MD.
Inuggi, A., Sanz-Arigita, E., González-Salinas, C., Valero-García, A.V., García-Santos, J.M., Fuentes, L.J., 2014. Brain functional connectivity changes in children that differ in impulsivity temperamental trait. Front. Behav. Neurosci. 8, 1–14.
Kamphuis, J., Meerlo, P., Koolhaas, J.M., Lancel, M., 2012. Poor sleep as a potential causal factor in aggression and violence. Sleep Med. 13, 327–334.
Kann, L., McManus, T., Harris, W.A., Shanklin, S.L., Flint, K.H., Hawkins, J., et al., 2016. Youth risk behavior surveillance – United States, 2015, surveillance summaries. Morb. Mortal. Wkly. Rep. 65, 1–174.
Kahn-Green, E.T., Lipizzi, E.L., Conrad, A.K., Kamimori, G.H., Killgore, W.D.S., 2006. Sleep deprivation adversely affects interpersonal responses to frustration. Personality and Individual Differences 41, 1433–1443.
Keyes, K.M., Maslowsky, J., Hamilton, A., Schulenberg, J., 2015. The great sleep recession: changes in sleep duration among US adolescents, 1991–2012. Pediatrics 135, 1–9.
Kriegeskorte, N., Simmons, W.K., Bellgowan, P.S., Baker, C.I., 2009. Circular analysis in systems neuroscience: the dangers of double dipping. Nat. Neurosci. 12, 535–540.
LeBourgeois, M.K., Giannotti, F., Cortesi, F., Wolfson, A.R., Harsh, J., 2005. The relationship between reported sleep quality and sleep hygiene in Italian and American adolescents. Pediatrics 115, 257–265.
Lee, T.H., Miernicki, M.E., Telzer, E.H., 2017. Families that fire together smile together: resting state connectome similarity and daily emotional synchrony in parent-child dyads. Neuroimage 152, 31–37.
Lei, Y., Shao, Y., Wang, L., Zhai, T., Zou, F., Ye, E., Jin, X., Li, W., Qi, J., Yang, Z., 2015. Large-scale brain network coupling predicts total sleep deprivation effects on cognitive capacity. PLoS One 10, e0133959.
Marco, C.A., Wolfson, A.R., Sparling, M., Azuaje, A., 2012. Family socioeconomic status and sleep patterns of young adolescents. Behav. Sleep Med. 10, 70–80.
Marino, M., Li, Y., Rueschman, M.N., Winkelman, J.W., Ellenbogen, J.M., Solet, J.M., et al., 2013. Measuring sleep: accuracy, sensitivity, and specificity of wrist actigraphy compared to polysomnography. Sleep 36, 1747–1755.
McCoy, J.G., Strecker, R.E., 2011. The cognitive cost of sleep lost. Neurobiol. Learn. Mem. 96, 564–582.
Miller, E.K., Cohen, J., 2001. An integrative theory of prefrontal cortex function. Ann.

Rev. Neurosci. 24, 167–202.
(NSF) National Sleep Foundation, 2014. Sleep in America Poll.
Orbeta, R.L., Overpeck, M.D., Ramcharran, D., Kogan, M.D., Ledsky, R., 2006. High caffeine intake in adolescents: associations with difficulty sleeping and feeling tired in the morning. J. Adolesc. Health 38, 451–453.
Owens, J., Adolescent Sleep Working Group, Committee on Adolescence, 2014. Insufficient sleep in adolescents and young adults: an update on causes and consequences. Pediatrics 134, e921–e932.
Petersen, A.C., Crockett, L., Richards, M., Boxer, A., 1988. A self-report measure of pubertal status: reliability, validity: and initial norms. J. Youth Adolesc. 17, 117–133.
Peterson, M.J., Benca, R.M., 2006. Sleep in mood disorders. Psychiatric Clin. 29, 1009–1032.
Poline, J.B., Worsley, K.J., Evans, A.C., Friston, K.J., 1997. Combining spatial extent and peak intensity to test for activations in functional imaging. Neuroimage 5, 83–96.
Romer, D., Betancourt, L., Giannetta, J.M., Brodsky, N.L., Farah, M., Hurt, H., 2009. Executive cognitive functions and impulsivity as correlates of risk taking and problem behavior in preadolescents. Neuropsychologia 47, 2916–2926.
Sadeh, A., Sharkey, K., Carskadon, M.A., 1994. Activity-based sleep-wake identification: an empirical test of methodological issues. Sleep 17, 201–207.
Sambataro, F., Murty, V.P., Callicott, J.H., Tan, H.Y., Das, S., Weinberger, D.R., Mattay, V.S., 2010. Age-related alterations in default mode network: impact on working memory performance. Neurobiol. Aging 31, 839–852.
Satterthwaite, T.D., Wolf, D.H., Erus, G., Ruparel, K., Elliott, M.A., Gennatas, E.D., et al., 2013. Functional maturation of the executive system during adolescence. J. Neurosci. 33, 16249–16261.
Schilbach, L., Eickhoff, S.B., Rotarska-Jagiela, A., Fink, G.R., Vogeley, K., 2008. Mind at rest? Social cognition as the default mode of cognizing and its putative relationship to the default system of the brain. Conscious. Cogn. 17, 457–467.
Settles, R.E., Fischer, S., Cyders, M.A., Combs, J.L., Gunn, R.L., Smith, G.T., 2012. Negative urgency: a personality predictor of externalizing behavior characterized by neuroticism, low conscientiousness, and disagreeableness. J. Abnorm. Psychol. 121, 160–172.
Sherman, L.E., Rudie, J.D., Pfeifer, J.H., Masten, C.L., McNealy, K., Dapretto, M., 2014. Development of the default mode and central executive networks across early adolescence: a longitudinal study. Dev. Cogn. Neurosci. 10, 148–159.
Shirer, W.R., Ryali, S., Rykhlevskaia, E., Menon, V., Greicius, M.D., 2012. Decoding subject-driven cognitive states with whole-brain connectivity patterns. Cereb. Cortex 22, 158–165.
Somerville, L.H., Jones, R.M., Casey, B.J., 2010. A time of change: behavioral and neural correlates of adolescent sensitivity to appetitive and aversive emotional cues. Brain Cogn. 72, 124–133.
Telzer, E.H., Fuligni, A.J., Lieberman, M.D., Galván, A., 2013. The effects of poor quality sleep on brain function and risk taking in adolescence. Neuroimage 71, 275–283.
van Belle, J., van Raalten, T., Bos, D.J., Zandbelt, B.B., Oranje, B., Durston, S., 2015. Capturing the dynamics of response variability in the brain in ADHD. Neuroimage Clin. 7, 132–141.
Whelan, R., Conrod, P.J., Poline, J.B., Lourdusamy, A., Banaschewski, T., Barker, G.J., Bellgrove, M.A., Büchel, C., Byrne, M., Cummins, T.D.R., et al., 2012. Adolescent impulsivity phenotypes characterized by distinct brain networks. Nat. Neurosci. 15, 920–925.
Whiteside, S.P., Lynam, D.R., 2001. The Five Factor Model and impulsivity: using a structural model of personality to understand impulsivity. Personal. Individ. Differ. 30, 669–689.
Winsler, A., Deutsch, A., Vorona, R.D., Payne, P.A., Szklo-Coxe, M., 2015. Sleepless in Fairfax: the difference one more hour of sleep can make for teen hopelessness, suicidal ideation and substance use. J. Youth Adolesc. 44 (362), 378.
Woolrich, M., 2008. Robust group analysis using outlier inference. Neuroimage 41, 286–301.
Zapolski, T.C.B., Cyders, M.A., Smith, G.T., 2009. Positive urgency predicts illegal drug use and risky sexual behavior. Psychol. Addict. Behav. 23, 348–354.
Zhu, X., Cortes, C.R., Mathur, K., Tomasi, D., Momenan, R., 2015. Model-free functional connectivity and impulsivity correlates of alcohol dependence: a resting-state study. Addict. Biol. 22, 206–217.
Zou, Q., Ross, T.J., Gu, H., Geng, X., Zuo, X.N., Hong, L.E., Gao, J.H., Stein, E.A., Zang, Y.F., Yang, Y., 2013. Intrinsic resting-state activity predicts working memory brain activation and behavioral performance. Hum. Brain Mapp. 34, 3204–3215.