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Original article

Getting a Good Night's Sleep: Associations Between Sleep Duration and Parent-Reported Sleep Quality on Default Mode Network Connectivity in Youth

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A B S T R A C T

Purpose: Sleep plays an important role in healthy neurocognitive development, and poor sleep is linked to cognitive and emotional dysfunction. Studies in adults suggest that shorter sleep duration and poor sleep quality may disrupt core neurocognitive networks, particularly the default mode network (DMN)—a network implicated in internal cognitive processing and rumination. Here, we examine the relationships between sleep and within- and between-network resting-state functional connectivity (rs-FC) of the DMN in youth.
Methods: This study included 3,798 youth (11.9 ± 0.6 years, 47.5% female) from the Adolescent Brain Cognitive Development cohort. Sleep duration and wake after sleep onset (WASO) were quantified using Fitbit watch recordings, and parent-reported sleep disturbances were measured using the Sleep Disturbance Scale for Children. We focused on rs-FC between the DMN and anticorrelated networks (i.e., dorsal attention network [DAN], frontoparietal network, salience network).
Results: Both shorter sleep duration and greater sleep disturbances were associated with weaker within-network DMN rs-FC. Shorter sleep duration was also associated with weaker anticorrelation (i.e., higher rs-FC) between the DMN and two anticorrelated networks: the DAN and frontoparietal network. Greater WASO was also associated with DMN-DAN rs-FC, and the effects of WASO on rs-FC were most pronounced among children who slept fewer hours/night.
Discussion: Together, these data suggest that different aspects of sleep are associated with distinct and interactive alterations in resting-state brain networks. Alterations in core neurocognitive networks may confer increased risk for emotional psychopathology and attention-related vulnerabilities. Our findings contribute to the growing number of studies demonstrating the importance of healthy sleep practices in youth.

IMPLICATIONS AND CONTRIBUTION

The present study demonstrates relationships between core neurocognitive networks and sleep duration and quality in a large sample of children. Both objective and subjective measures of sleep were utilized. The findings contribute to public health concerns related to poor sleep in children and may inform future interventions.

Sleep is a critical component of healthy emotional and behavioral health, particularly during development [1]. Prior studies show that both quantity and quality of sleep play an

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important role in maintaining emotional health. For example, shorter sleep duration and poor quality of sleep (e.g., more fragmented sleep, greater sleep disturbances) have both been linked to higher self-reported stress, greater risk of internalizing psychopathology (e.g., depression, anxiety), and poorer self-reported emotional functioning [2]. According to the American Academy of Sleep Medicine, 6–12-year-old and 13–18-year-old children should be sleeping between 9–12 or 8–10 hours each night, respectively [3]. Unfortunately, some observational studies have reported that more than 90% of school-aged children do not meet these recommended guidelines, and that both sleep duration and quality decline during the second decade of life [4]. Given that the incidence of internalizing psychopathology increases with age during the transition from childhood to adolescence [5], it is important to understand the impact of both sleep duration and quality on core neurocognitive networks implicated in healthy and atypical cognitive and emotion-related functioning in youth.

Although several studies link poor sleep to increased risk of internalizing psychopathology, few studies have explored the underlying neural mechanisms in developing populations. Studies in youth are important given that childhood and adolescence are periods of psychiatric vulnerability and of dramatic changes within and between core neurocognitive networks that are known to be susceptible to sleep disturbances and implicated in the pathology of internalizing disorders [6,7]. Indeed, studies in adults demonstrate that total sleep deprivation and poor naturalistic sleep are associated with altered functional interactions within the default mode network (DMN) and between the DMN and anticorrelated networks. The DMN is anchored in several core regions, including the posterior cingulate cortex, precuneus, medial prefrontal cortex, and inferior parietal cortex [8]. The DMN—also called the "task-negative network"—is deactivated during goal-oriented tasks and is associated with self-referential thinking, rumination, and envisioning the future. Prior studies in adolescents and adults show that poor sleep duration [9] and quality [10] are associated with reduced within-network DMN resting-state functional connectivity (rs-FC). Given that weaker within-network DMN rs-FC is observed in deep sleep, some authors have interpreted this finding as a segment of the brain engaging in "local sleep" wherein regions of the brain engage in a sleep-like state during waking hours [10–13]. Similarly, weaker within-network DMN connectivity during wakefulness is associated with poor performance in behavioral tasks [14].

Total sleep deprivation has also been shown to interrupt interactions between the DMN and core neurocognitive networks, namely the dorsal attention network (DAN), frontoparietal network (FPN), and salience network (SN). In contrast to the DMN, these networks are engaged in responding to externally oriented tasks and typically show an oppositional pattern of rs-FC with the DMN at rest [15]. Prior studies report that, compared to well-rested individuals, adults deprived of sleep for 36 hours have higher DMN-DAN and DMN-SN rs-FC, and lower DMN-FPN rs-FC [16,17]. Partial sleep deprivation in adults is associated with similar patterns of weaker within-network DMN and weaker DMN-DAN anticorrelation (i.e., higher DMN-DAN rs-FC) [18]. Given that weaker within-network DMN rs-FC and altered between-network DMN rs-FC are also linked to the risk of emotional psychopathology [19], DMN rs-FC may explain the well-documented link between poor sleep and poor emotional health. Understanding the impact of sleep duration and quality on neural network-level interactions may therefore help to identify early markers that precede later behavioral and health consequences.

Although fewer studies have been conducted in children and adolescents, recent work by our group and others has shown that sleep duration and quality can impact functional neural connectivity in youth [20–22]. To our knowledge, only four studies have examined the effects of sleep on within- and/or between-network DMN rs-FC in youth [10,21–23]. Similar to studies in adults, studies in youth report weaker within-network DMN rs-FC with shorter sleep duration and/or poorer sleep quality [10,21,22]. These findings suggest that poor sleep can negatively impact network functioning of the DMN earlier in life than previously reported, and may increase the risk of later emotional psychopathology [5]. A recent study in the Adolescent Brain Cognitive Development (ABCD) cohort found that the link between total sleep disturbances and mental problems at the one-year follow-up was mediated by within- and between-network DMN and DAN rs-FC [23]. However, no studies to-date have comprehensively examined the impact of both sleep duration and quality on within- and between-network rs-FC of the DMN, FPN, DAN, and SN, and no studies have incorporated both *objective* and subjective sleep measures. The former is important given that parent reports tend to overestimate child sleep as compared to more objective measures (e.g., watches, actigraphy) [24,25].

The present study aimed to measure the impact of sleep duration and quality on within- and between-network rs-FC of the DMN in youth in the ABCD study. We focused on three key sleep variables: (1) sleep duration and (2) wake after sleep onset (WASO), measured using objective data from Fitbit watches, and (3) sleep disturbances, measured using parent proxy reports. We aimed to both replicate and extend prior studies in youth using a larger and more diverse sample and incorporating both subjective and objective sleep measures for the first time. Based on prior literature in adults [16,17], we hypothesized that both shorter sleep duration and poorer sleep quality (i.e., greater WASO, greater parent-reported sleep disturbances) would be associated with weaker within-network DMN rs-FC and weaker (i.e., less negative) rs-FC between the DMN and anticorrelated networks (i.e., FPN, DAN, SN). We also explored interactions between sleep variations, age-by-sleep, and sex-by-sleep interactions on rs-FC, and associations among sleep variables, rs-FC, and internalizing symptoms.

Methods

Participants

Our sample consisted of 3,798 youth from the ABCD study (ages 10.6–13.4 years, *mean [M]* = 11.9, *standard deviation [SD]* = 0.6; see Table 1). The ABCD study is the largest prospective cohort study of brain development and child health in the United States. Informed consent was obtained from parents and guardians and assent was obtained from children. See Supplementary Material for more information.

Measures

Sleep duration and quality

Objective measures. Objective sleep data were collected from participants using Fitbit Charge HR2 watches. Average sleep

Table 1
Participant demographics

Variable	*n* (%)	*M (SD)*	Range
Age (years)		11.9 (0.6)	10.6–13.4
Biological sex (female)	1,805 (47.5)		
Race/Ethnicity			
Non-Hispanic White	2,433 (64.1)		
Non-Hispanic Black	390 (10.3)		
Hispanic	592 (15.6)		
Black Hispanic	14 (0.4)		
Asian, AIAN, NHPI	154 (4.1)		
Multiple	175 (4.6)		
Don't know/not reported	40 (1.1)		
Parent education			
Less than high school	172 (4.5)		
High school/GED	283 (7.5)		
Some college	1,141 (30.0)		
Bachelor's degree	1,134 (29.9)		
Postgraduate degree	994 (26.2)		
Refused to answer	5 (0.1)		
Annual household income			
<$5,000	68 (1.8)		
$5,000-$11,999	94 (2.5)		
$12,000-$15,999	62 (1.6)		
$16,000-$24,999	111 (2.9)		
$25,000-$34,999	195 (5.1)		
$35,000-$49,999	262 (6.9)		
$50,000-$74,999	513 (13.5)		
$75,000-$99,999	495 (13.0)		
$100,000-$199,999	1,227 (32.3)		
>$200,000	511 (13.5)		
Refuse to answer	127 (3.3)		
Don't know	132 (3.5)		
Pubertal categories			
Prepuberty	433 (11.4)		
Early puberty	860 (22.6)		
Mid-puberty	1689 (44.5)		
Late puberty	101 (2.7)		
Postpuberty	583 (15.4)		
Don't know/not reported	124 (3.3)		
Parent-reported child internalizing symptoms (CBCL *t*-scores)		47.7 (10.4)	33–90
Average head motion (mm)		0.2 (0.2)	0.2–1.9

AIAN = American Indian/Alaskan Native; NHPI = Native Hawaiian/Pacific Islander; GED = General Education Diploma; CBCL = Child Behavior Checklist; SD = standard deviation.

duration, bedtime, wake time, and WASO were measured for each participant. Fitbit data were collected from ~13.1 nights/participant (SD = 6.5 nights; min. = 1; max. = 98), yielding information on 49,875 person-nights. Average sleep efficiency was calculated by dividing the total sleep time by the total time in bed, with higher scores indicating greater efficiency. See Supplementary Material for further details.

Subjective measures. Parent-reported sleep disturbances were measured using the Sleep Disturbance Scale for Children (SDSC). The SDSC provides an overall score and categorizes sleep disorders into five subdomains: disorders of initiative and maintaining sleep, sleep breathing disorders, disorders of arousal, sleep-wake transition disorders, disorders of excessive somnolence, and sleep hyperhidrosis. The SDSC shows adequate validity and reliability (α = 0.71 to 0.79; [26]). Cronbach's alpha for this sample was α = 0.83. Given previously reported discrepancies between parent-reported sleep duration and actigraphy [24], our primary analyses focused on sleep duration measured using the Fitbit watches. However, we also explored correlations between parent-reported sleep and Fitbit sleep data.

Internalizing symptoms. Internalizing symptoms (*t*-scores) were measured using the well-validated parent-reported Child Behavior Checklist [27].

Default mode network resting-state analyses

Within- and between-network rs-FC of the DMN was calculated using the Gordon network parcellation scheme [28]. We focused a priori on the DMN and three other core neurocognitive networks (DAN, FPN, SN; see Figure 1). Here, we used the tabulated rs-FC data available on the NIMH Data Archive for the following four connections: DMN-DMN (i.e., within-network rs-FC), DMN-DAN, DMN-FPN, and DMN-SN. magnetic resonance imaging (MRI) processing steps are described in detail in Hagler et al. [26]. In brief, the Gordon network parcellation [28] was used as the template for rs-FC data. Average network correlation was calculated as the average Fischer-*r*-to-*Z* correlations for each pairwise combination of regions of interest that belong to each network (e.g., DMN, FPN).

Statistical analysis

First, to explore the distribution of our primary sleep variables (i.e., duration, WASO, parent-reported sleep disturbances) across the sample, we computed histograms. For parent-reported sleep disturbance scores, we used a cutoff of 39 to identify children with significant sleep disturbances, following Bruni et al. (1996) [29]. Next, we explored associations between age and sleep variables using bivariate correlation. Differences in sleep variables by sex (female, non-female) and race (White, non-White; largest groups) were explored using two-sample *t*-tests ($p < .05$). Spearman correlation at a significance level of $p < .05$ was used to explore associations among sleep variables, family income, and parent education. Our main analyses examined main effects of the three primary sleep-related variables (i.e., sleep duration, WASO, sleep disturbances) on rs-FC (i.e., DMN-DMN, DMN-DAN, DMN-FPN, DMN-SN) using regression. False discovery rate (FDR) was used to control for multiple comparisons. To ensure results were robust to potential confounds, regression analyses were repeated adjusting for the following covariates: age, sex, race, parent education, family income, number of Fitbit readings, puberty, and head motion. Puberty was included as a covariate because prior studies have demonstrated unique impacts of puberty from age on rs-FC and the development of brain networks [7,30–32]. Mean head motion (measured by framewise displacement) during the rs-FC scan was entered as a covariate following prior studies and recommendations [23,33]. We repeated analyses controlling for family identification (ID) nested within site ID as random effects, and separately with family ID nested within MRI manufacturer (Siemens, GE, or Philips) as random effects. We also tested whether results were robust to the inclusion of other primary sleep variables. Collinearity was assessed using variance inflation factor (VIF). Where separate significant main effects were present for a given connection, we also tested for interactive effects(e.g., sleep duration-by-sleep disturbances). The interaction term was computed by multiplying the two continuous variables, following mean centering. Exploratory analyses tested for age-by-sleep and sex-by-sleep interactions on rs-FC using regression analyses, and associations among sleep, rs-FC, and internalizing symptoms using Pearson

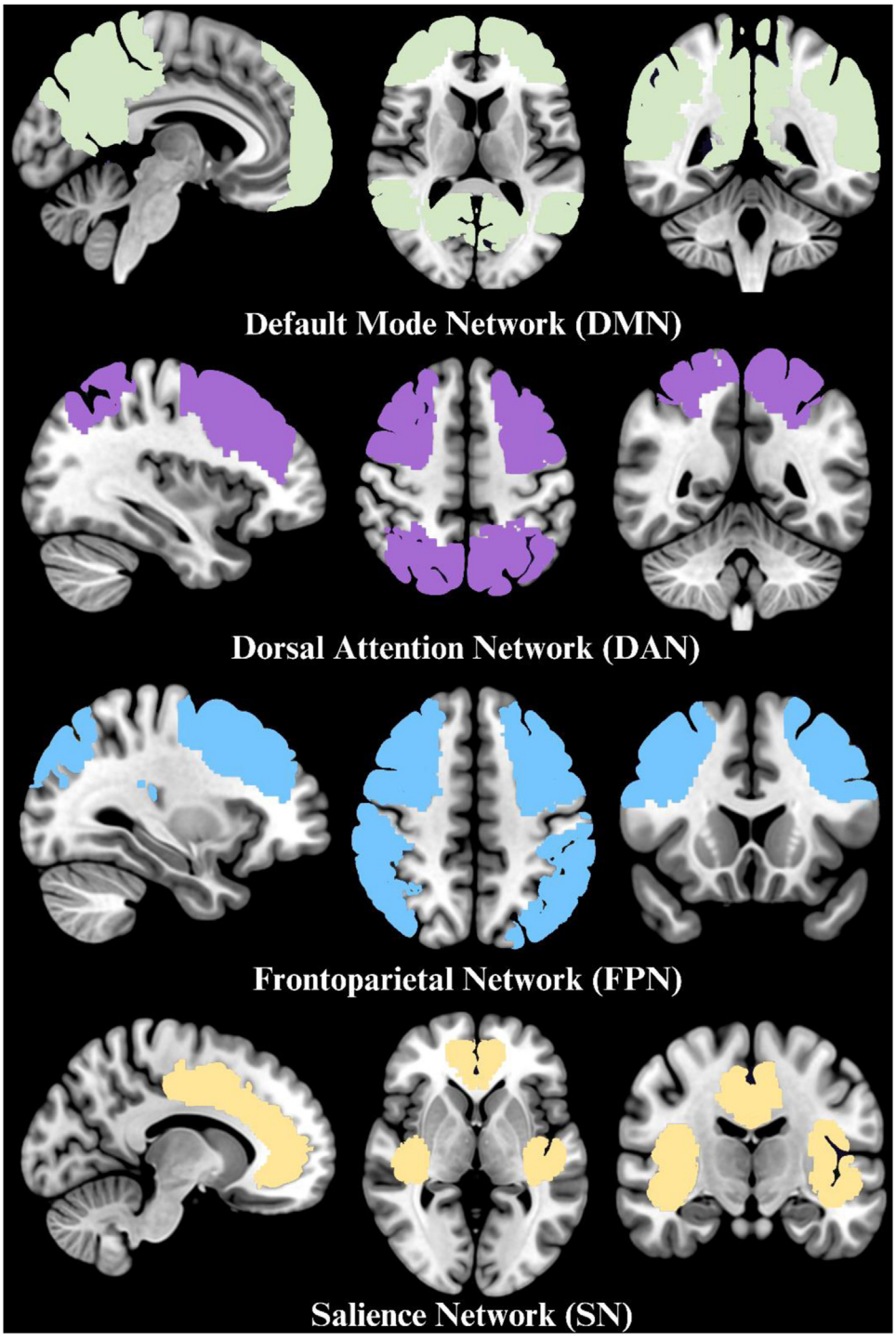

Figure 1. Four neurocognitive networks of interest; default mode network (DMN), dorsal attention network (DAN), frontoparietal network (FPN), and salience network (SN). This figure is intended for color reproduction on web, but not in print.

bivariate correlation. Significant interactions were followed up with group cutoffs, using a median split (e.g., by age) or by suggested scale cutoffs (e.g., sleep disturbance scale). Exploratory analyses were considered significant at $p < .05$. Statistical analyses were performed using SPSS v.27 (IBM Corp). For details about outlier analyses see Supplementary Material.

Results

Overall sleep duration and quality

Average sleep duration across the sample ranged from 3.04 to 14.1 hours ($M = 7.4$, $SD = 0.73$; see Table A1) and was weakly

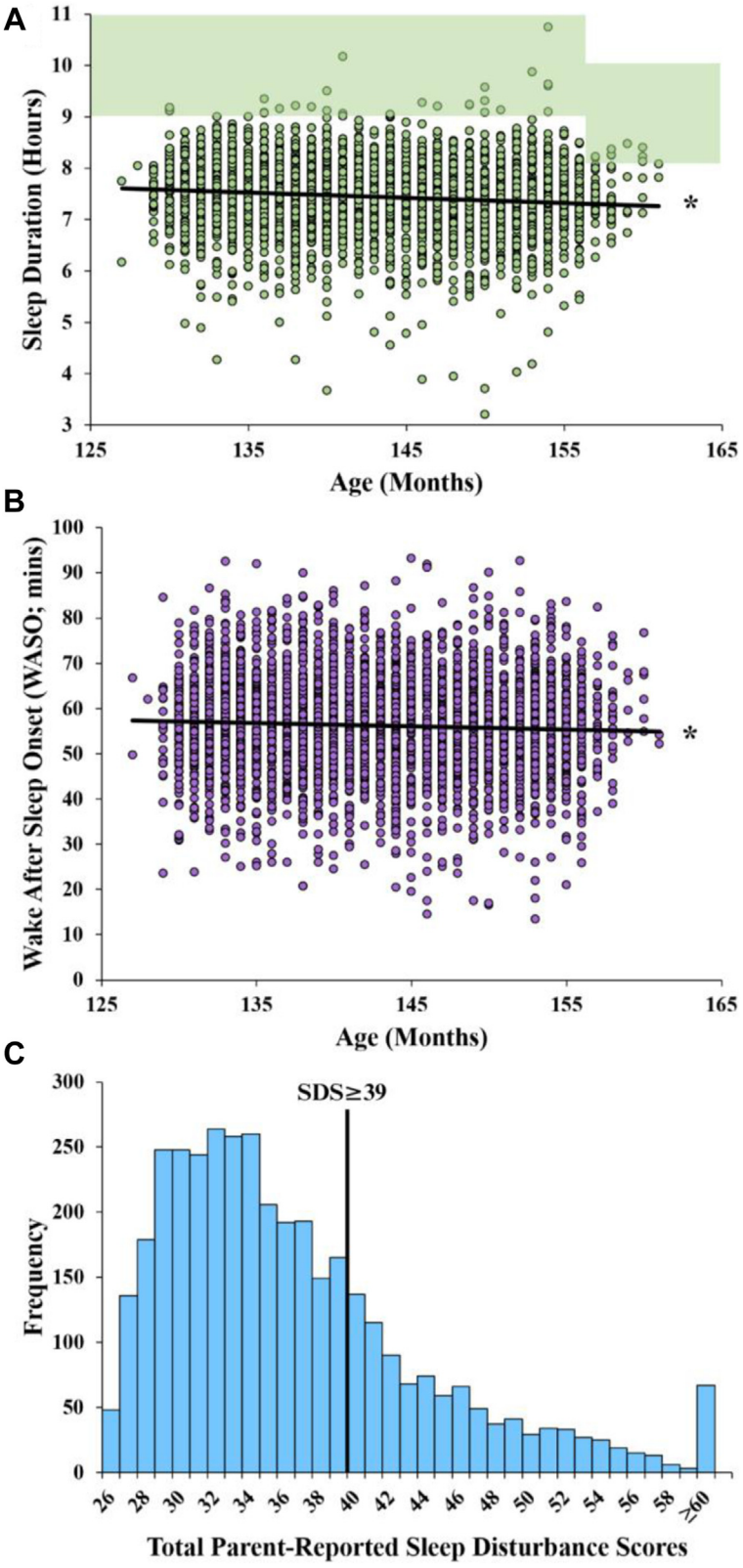

Figure 2. Distribution of sleep-related variables across the sample with outliers removed. (A) Negative association between age and sleep duration. Shaded green regions represent recommendations from the American Academy of Sleep Medicine (9–12 hours for children between 6 and 12 years of age; 8–10 hours for children between 13 and 18 years of age) [34]. (B) Negative association between age and wake after sleep onset (WASO). (C) Histogram showing distribution of total sleep disturbance scores across the sample with higher numbers indicating greater disturbances. Of note, 26.5% of participants had scores above 39, indicated by the vertical line, which may indicate significant sleep disturbances [28]. *$p < .05$. SDS = Sleep Disturbance Score. This figure is intended for color reproduction on web, but not in print.

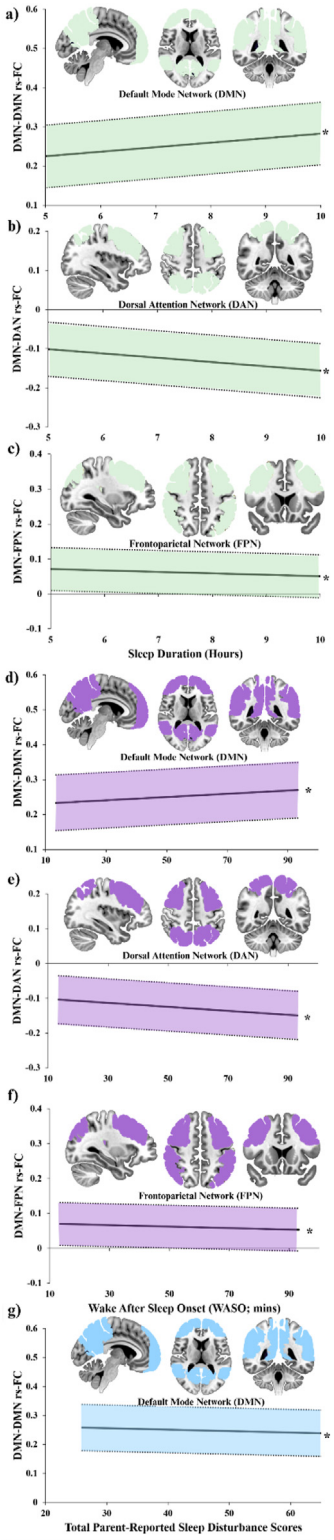

Figure 3. Effects of sleep duration (A–C), wake after sleep onset (WASO; D–F), and parent-reported sleep disturbance scores (G) on within and between DMN rs-FC. Shaded regions represent one standard deviation above or below the line of best fit, for display purposes. DMN = default mode network; DAN = dorsal attention network; FPN = frontoparietal network; rs-FC = resting-state functional connectivity. *$p < .05$, regression. This figure is intended for color reproduction on web, but not in print.

correlated with parent-reported sleep duration (r (3795) = 0.29, $p < .001$). Sleep duration is plotted by age in Figure 2A, and recommended sleep duration for children according to the American Academy of Sleep Medicine [3] is shown in the shaded area. As shown in Figure 2A, 98.3% of youth did not meet recommendations for sleep duration. Average wake time ranged from 1:25 A.M. to 7:45 P.M. (M = 7:31 A.M., SD = 1:12) and average sleep efficiency ranged from 17% to 95% (M = 86.6%, SD = 3.8%; Figure A1B; see Table A1) with 86.5% of youth showing a sleep efficiency above 85%, indicating typical sleep efficiency [34]. Average WASO ranged from 13.5 to 133.5 minutes (M = 56.7, SD = 11.4; Figure 2B). Bedtime and wake time are given in Figure A1A–C, respectively. Total parent-reported sleep disturbance scores ranged from 26 to 101 (M = 36.5, SD = 7.9), and 26.5% of youth had total scores above 39, suggesting significant sleep disturbances (see Figure 2C). See Supplementary Material for breakdown of sleep disturbances, by subscale, for correlations among sleep variables, and effects of demographic variables.

Associations between sleep and rs-FC

Sleep duration. Shorter sleep duration was associated with lower within-network DMN rs-FC (R^2 = 0.009, F (1, 3796) = 36.3, $p < .001$; see Figure 3 and Table 2), higher DMN-DAN rs-FC (R^2 = 0.01, F (1, 3796) = 47.3, $p < .001$), and higher DMN-FPN rs-FC (R^2 = 0.002, F (1, 3796) = 8.3, p = .004). These associations survived FDR correction. These findings also remained significant after removing outliers in sleep variables, children with excessive head motion, and children with an excessively high number of Fitbit readings (see Supplementary Material), and after adjusting for MRI manufacturer, site ID, and family ID. Sleep duration remained a significant predictor of DMN-DMN, DMN-DAN, and DMN-FPN rs-FC after adding both WASO and sleep disturbances to the model (VIF < 2). However, the association between sleep duration and DMN-FPN rs-FC was not significant after adjusting for covariates (see Supplementary Material). Sleep duration was not associated with DMN-SN rs-FC (p = .9).

WASO. Greater WASO was associated with higher DMN-DMN rs-FC (R^2 = 0.003, F (1, 3796) = 13.2, $p < .001$; Figure 3A), lower DMN-DAN rs-FC (R^2 = 0.008, F (1, 3796) = 29.3, $p < .001$; Figure 3B), and lower DMN-FPN rs-FC (R^2 = 0.001, F (1, 3796) = 5.0, p = .03; Figure 3C). These results survived FDR correction. These associations also remained significant after removing sleep variable outliers, children with high head motion, and children with an excessively high number of Fitbit readings (see Supplementary Material). The DMN-DMN rs-FC and DMN-DAN rs-FC association remained significant after adjusting for covariates, and after adjusting for MRI manufacturer, site ID, and family ID; however, the association between WASO and DMN-FPN rs-FC did not (p = .2; see Supplemental). When adding sleep duration and sleep disturbances as covariates, the association between WASO and DMN-DAN rs-FC remained significant, such that higher WASO is associated with lower DMN-DAN rs-FC (R^2 = 0.014, F (2, 3795) = 26.1, p = .03). However, the effects of WASO on within-network DMN rs-FC (R^2 = 0.01, F (2, 3795) = 18.3, p = .5) and DMN-FPN rs-FC (R^2 = 0.002, F (2, 3795) = 4.5, p = .4) were no longer significant. This suggests that sleep duration and sleep disturbances (but not WASO) predict DMN-DMN and DMN-FPN rs-FC (VIFs < 2).

Table 2
Regression analysis between sleep variables and rs-FC

Sleep Variable	Connection	B	Se	β	*t*	*p*
Sleep duration	**DMN-DMN***	**0.097**	**0.000029**	**0.000177**	**6.021**	**<.001**
	DMN-DAN*	**−0.110970**	**0.000026**	**−0.000176**	**−6.880**	**<.001**
	DMN-FPN*	**−0.046669**	**0.000023**	**−0.000066**	**−2.878**	**.004**
	DMN-SN	0.001662	0.000033	0.000003	.102	.918
Wake after sleep onset (WASO)	**DMN-DMN***	**0.058805**	**0.000113**	**0.000412**	**3.629375**	**<.001**
	DMN-DAN*	**−0.087507**	**0.000099**	**−0.000533**	**−5.412232**	**<.001**
	DMN-FPN*	**−0.036172**	**0.000087**	**−0.000195**	**−2.230069**	**0.025801**
	DMN-SN	−0.000317	0.000126	−0.000002	−0.019518	0.984429
Parent-reported sleep disturbances	**DMN-DMN***	**−0.054087**	**0.000163**	**−0.000543**	**−3.336863**	**<.001**
	DMN-DAN*	0.035872	0.000142	0.000313	2.211262	.027077
	DMN-FPN	−0.000479	0.000125	−0.000004	−0.029482	.976482
	DMN-SN	0.025596	0.000180	0.000283	1.577296	.114811

DMN = default mode network; DAN = dorsal attention network; FPN = frontoparietal network; SN = salience network; rs-FC = resting-state functional connectivity.
* Significant at $p < .05$. Bolded: significant after multiple comparison correction (FDR). FDR = false discovery rate.

Sleep disturbances. Greater parent-reported sleep disturbances were associated with lower DMN-DMN rs-FC ($R^2 = 0.003$, $F(1, 3795) = 11.1$, $p < .001$; Figure 3D). This association survived FDR correction, was significant after adjusting for covariates, including site ID, family ID, and scan manufacturer, and after removing sleep variable outliers, children with high head motion, and children with an excessively high number of Fitbit readings (see Supplementary Material). Sleep disturbances also remained a significant predictor of DMN-DMN rs-FC after adjusting for sleep duration and WASO ($R^2 = 0.011$, $F(1, 3796) = 14.645$, $p < .001$). Exploratory *post hoc* analyses showed the following subscales were significant predictors of DMN-DMN rs-FC: disorders of initiating and maintaining sleep ($R^2 = 0.06$, $F(9, 3566) = 24.4$, $p = .01$), disorders of arousal ($R^2 = 0.06$, $F(9, 3566) = 24.2$, $p = .02$), and sleep wake transition disorders ($R^2 = 0.06$, $F(9, 3566) = 24.1$, $p = .03$) after adjusting for covariates. These associations remained significant after FDR correction. However, when all six subtypes were included, disorders of maintaining sleep remained significant ($R^2 = 0.005$, $F(6, 3790) = 3.4$, $p = .02$). Sleep disturbance scores were also positively associated with DMN-DAN rs-FC ($R^2 = 0.001$, $F(1, 3795) = 4.9$, $p = .03$). However, this association did not survive FDR correction and was not significant after adjusting for covariates (see Supplementary Material).

Exploratory analyses

Given that both sleep duration and parent-reported sleep disturbances showed associations with DMN-DMN rs-FC, and that sleep duration and WASO showed associated with DMN-DAN rs-FC, we performed follow-up exploratory analyses to test for potential interactive effects. We also explored sex-by-sleep and age-by-sleep interactions on all rs-FC connections, and associations with internalizing symptoms (see Supplementary Material for further details).

Sleep duration-by-parent-reported sleep disturbance interactions on DMN-DMN rs-FC. There were no significant interactive effects, $p = .561$.

Sleep duration-by-WASO interactions on DMN-DAN rs-FC. There was a significant sleep duration-by-WASO interaction on DMN-DAN rs-FC ($R^2 = 0.014$, $F(3, 3797) = 18.86$, $B = −0.03$, $p = .039$; Figure A2). Follow-up analyses in sleep duration groups (median split: lower [<7.5 hrs/night], higher [≥7.5 hrs/night]) showed that the negative association between WASO and DMN-DAN rs-FC was significant only among youth who slept fewer hours/night ($R^2 = 0.01$, $F(1, 1871) = 19.33$, $B = −0.001$, $p < .001$), as compared to youth who slept longer hours ($R^2 = 0$, $F(1, 1925) = 1.11$, $B = −0.024$, $p = .29$).

Discussion

To our knowledge, this is the first study to incorporate both objective and subjective measures of sleep quality and duration and examine their relation to rs-FC within and between core neurocognitive networks in a youth sample. Alarmingly, 98.3% of participants in this ABCD sample did not meet recommendations for sleep duration in youth [3] and one in four youth showed significant sleep disturbances. This is in line with and exceeds typical rates reported in prior studies of sleep quality and duration in youth [35]. Our rs-FC analyses indicated that both duration and quality of sleep are associated with alterations in DMN rs-FC in youth. Both shorter sleep duration and greater parent-reported sleep disturbances were associated with weaker within-network DMN rs-FC, which replicates prior studies in adults (see summary in Figure A3). Shorter sleep duration was also associated with weaker anticorrelation (i.e., higher rs-FC) between the DMN and two anticorrelated networks: the DAN and FPN. Greater WASO was also associated with DMN-DAN rs-FC, and the effects of WASO on DAN rs-FC were most pronounced among children who slept fewer hours/night on average. Taken together, these data suggest that different aspects of sleep (e.g., duration, WASO, disturbances) can have both distinct and interactive effects on resting-state networks in the brain. Importantly, weakened within-network rs-FC of the DMN and heightened connectivity between the DMN and anticorrelated networks (i.e., weakened anticorrelation) are implicated in cognitive dysfunction [36,37] and risk of internalizing psychopathology [38,39]. Therefore, alterations in core neurocognitive networks may underlie the link between poor sleep and poor emotional health in youth. It is important to note that our results do not shed light on the directionality of this relationship. Alternatively, children with disturbances in rs-FC related to cognitive dysfunction and psychopathology may have trouble achieving adequate sleep quality and duration.

Our results both replicate and extend prior studies in adults and in youth linking poor sleep to weaker within-network DMN rs-FC [9,10,21,22]. Indeed, prior studies in sleep deprivation and

disorders, and poor naturalistic sleep patterns, are associated with weaker DMN connectivity at rest. Here, we integrated objective and subjective measures of both sleep duration and quality (i.e., parent-reported sleep disturbances) to study the impact of naturalistic sleep on DMN rs-FC in youth. We found that both shorter sleep duration and increased parent-reported sleep disturbances are independently associated with weaker within-network DMN rs-FC. A recent study within the baseline ABCD cohort by Brooks and colleagues [22] linked shorter *parent-reported* sleep duration to a similar pattern of lower integration and efficiency within the DMN, including lower global efficiency and median connectivity. The present study replicates and extends these prior findings by utilizing *objective* Fitbit-derived measures of duration and using complementary measures of DMN connectivity (i.e., bivariate rs-FC correlations).

Interestingly rumination, which is associated with alterations in within-network rs-FC of the DMN [40–43], is also associated with delayed onset and lower subjective sleep quality [44–46]. A previous study by Antypa et al. [47] found that rumination was a significant mediator in the association between evening chronotype (e.g., later sleep onset) and depression in healthy and depressed adults. Moreover, in a sample of university students, the positive effects of a self-compassion intervention and improved sleep quality were mediated by a reduction in ruminative thoughts [48]. The links among poor sleep, rumination, and the DMN are especially relevant given that rumination can predict psychopathology (e.g. major depressive disorder) among adolescents [49] and evidence indicates that rumination mediates the association between stress, affect, and symptoms of both depression and anxiety. These data suggest that ruminative thoughts confer sensitivity to stress [50] in late adolescents and young adults. These linkages highlight dynamic and complicated relationships which merit future research. Elucidation of these associations may illuminate important regulatory mechanisms and opportunities for health promotion interventions.

In addition to alterations in within-network DMN connectivity, we found that poor sleep was associated with weaker rs-FC with anticorrelated networks (i.e., FPN and DAN) in youth. In particular, consistent with a prior sleep deprivation study in adults [16], shorter sleep duration was associated with heightened between-network rs-FC of the DMN with the DAN in youth. The DAN, consisting of the intraparietal sulcus and frontal eye field, is a task-positive network implicated in top-down attention to external stimuli and completing goal-directed tasks [51,52]. The DAN is typically anticorrelated with the DMN at rest [15], which may reflect opposing modes of attention to internal (e.g., self-directed thoughts, DMN) versus external stimuli (e.g., DAN). Weaker DMN-DAN anticorrelation has been associated with attentional problems [19], which may explain the frequently reported attentional deficits following sleep deprivation [53,54]. An alternative explanation for these findings is that children with disrupted rs-FC reflective of attentional problems may have trouble achieving appropriate sleep quality and duration. There may be some overlap in the brain regions involved in sleep, arousal and attention regulation, which is currently a research priority [55]. We also observed that WASO was associated with DMN-DAN rs-FC in this sample, and that WASO interacted with sleep duration to impact rs-FC such that the effects of WASO on rs-FC were apparently only in youth who slept for fewer hours/night than more well—rested youth. Together, these results extend prior studies in adults to a developing sample, and highlight the importance of considering multiple aspects of sleep on neural network interactions.

The FPN is another task-positive network and consists of the dorsolateral prefrontal cortex and the inferior parietal lobule. The FPN is implicated in goal-directed tasks and executive function [56] and prior studies show that increasingly cognitive demanding tasks are associated with stronger DMN-FPN anticorrelation (i.e., lower connectivity) [57]. Conversely, weaker DMN-FPN anticorrelation is linked to poorer cognitive control [58] and internalizing psychopathology in adults [40,59]. Here, we found that sleep duration was associated with weakened DMN-FPN anticorrelation. Given that poor cognitive control is consistently reported following sleep deprivation [54,60], future studies should explore weakened DMN-FPN anticorrelation as a prospective predictor of the association between poor sleep and cognitive dysfunction.

Strengths and limitations of this study should be considered. Strengths include the use of a large, diverse, nation-wide neuroimaging sample of youth, and the inclusion of both objective and subjective measures of sleep duration and quality. Limitations include the reliance on cross-sectional data, which precludes our ability to examine prospective associations among sleep, rs-FC, and behavioral outcomes. Future releases of the ABCD data set will be used to evaluate these longitudinal associations. The present study also uses prospectively collected sleep data over an average of 13 nights. Although this is a strength when compared to studies which utilize only a single night of sleep or retrospective recall of habitual sleep, it may fail to capture long-term relationships between sleep and DMN rs-FC. This limitation may be addressed using longitudinal data with future releases of the ABCD study. There may also be inconsistencies in the sleep disturbance measure due to parent report data. This limitation was offset, in part, by the inclusion of both subjective and objective measures of sleep quality. Another limitation is that the ABCD Fitbit protocol captures sleep patterns over a three-week period, which may have varied depending on time of year due to demands from school and adherence to the suggested recording protocol. However, we performed additional sensitivity analyses to ensure that this variability did not explain the results reported here. These findings both replicate and extend prior studies that use parent-report measures of sleep, which may diverge from objective measures [25]. Actigraphy and wearable devices are more accurate than self-reported data, and wearable devices (e.g., Fitbits) may be more easily accessible to participants. However, a recent meta-analysis shows that Fitbits may overestimate total sleep time [61]; therefore, future studies should aim to replicate these findings using polysomnography. Furthermore, participants were not administered a sleep diary to accompany the Fitbit data. Thus, we cannot rule out the possibility that disruptions to sleep were not reported in the ABCD post-assessment parent survey for the Fitbit protocol. In addition, the effect sizes in the relationships between sleep variables and rs-FC were modest in size, which is a known limitation of using large data sets, including ABCD data [62,63]. Therefore, results should be interpreted cautiously. Another important limitation is that rs-FC data were collected using three different MRI manufacturers across ABCD study sites, which may limit the comparability of imaging results. However, as outlined by Casey et al. [64], several steps were prospectively taken to mitigate potential between-manufacture and between-site differences, including using a standardized protocol and imaging parameters

and acquiring images in planes compatible with all three manufacturers. We have also taken steps to reduce the potential impact of scan platform and site in our analyses, including repeating analyses adjusting for these factors. This limitation should be weighed against the strengths of multisite studies, including increasing sample size and statistical power.

Conclusion

The present study demonstrates associations between sleep and DMN rs-FC in a cross-sectional sample of early adolescents from the ABCD study. This study utilized objective (e.g., Fitbit watches) and subjective (e.g., sleep disturbance scale) measures of sleep. We found that poor sleep was associated with diminished within-network DMN rs-FC and increased rs-FC between the DMN and anticorrelated networks (e.g., DAN and FPN). Given the critical role of the DMN and interactions with anticorrelated networks in mediating healthy brain functioning, including learning, memory, attention, and emotion regulation, alterations in core neurocognitive networks may contribute to the well-established link between poor sleep and negative sequela. Together, this study adds to the growing public health concerns related to insufficient sleep in youth and may have implications for interventions and public policy (e.g., school start times). Both sleep duration and quality should be considered when designing interventions for promoting behavioral health and improving academic outcomes among youth.

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Supplementary Data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.jadohealth.2023.01.010.

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