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Original article

Sleep Disturbance Predicts Depression Symptoms in Early Adolescence: Initial Findings From the Adolescent Brain Cognitive Development Study

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A B S T R A C T

Purpose: The aim of the study was to investigate associations between sleep disturbances and mental health in adolescents.
Methods: Data are from a national sample of 11,670 U.S. participants (5,594 females, aged 9–10 years, 63.5% white) in the Adolescent Brain Cognitive Development study. Initial longitudinal analyses were conducted for a subset of the sample (n = 4,951). Measures of youth sleep disturbance (disorders of initiating and maintaining sleep, sleep–wake transition disorders, and disorders of excessive somnolence) and "typical" total sleep time (number of hours slept on most nights in the past 6 months) were obtained from the parent-report Sleep Disturbance Scale (Data Release 2.0). Parent-report measures of youth mental health (depression, internalizing, and externalizing behaviors) from the Child Behavior Checklist and typical screen time were included.
Results: At baseline, greater sleep disturbance and shorter total sleep time were associated with greater internalizing, externalizing, and depression scores. After controlling for baseline mental health symptoms, baseline sleep disturbance significantly predicted depression and internalizing and externalizing scores at 1-year follow-up. A significant interaction with sex indicated that the association between disorders of excessive somnolence and depression 1 year later was steeper for girls, compared with boys ($p < .001$; 95% confidence interval 1.04–3.45).
Conclusions: Sleep disturbances predicted future mental health, particularly depression in this young sample, highlighting the potential to harness sleep as a tool to mitigate the persistence of depression across early adolescence and potentially prevent an adolescent onset of major depressive disorder.
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IMPLICATIONS AND CONTRIBUTION

The incidence of depression increases markedly during adolescence, particularly for girls, and is associated with lifetime consequences. Sleep disturbance predicts depression symptoms in early adolescence, thus implicating sleep as a potential tool to limit the progression of depression and reduce the likelihood of an adolescent onset of major depressive disorder.

Conflicts of interest: None of the authors have potential conflicts of interest to be disclosed.
Disclaimer: This article reflects the views of the authors and may not reflect the opinions or views of the National Institutes of Health or Adolescent Brain Cognitive Development (ABCD) consortium investigators. The ABCD data repository grows and changes over time. The ABCD data used in this report came from https://dx.doi.org/10.15154/1503209.

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The incidence of depression increases markedly during adolescence [1], the phase of life stretching between childhood and adulthood (10–24 years) [2]. Depression is the most common psychiatric disorder in youth [3], is ranked worldwide as the most burdensome condition in those aged <25 years [4], and is a major risk factor for adolescent suicide [5]. An onset of major depressive disorder (MDD) during this period strongly predicts a lifetime trajectory of depressive episodes [6]. Thus, identifying positive modifiers of depression symptomology is key to developing preventative and treatment strategies that can be applied in early life, before an onset of MDD has occurred. Sleep is one potentially modifiable, behavior that undergoes considerable change across adolescence and is vital for brain plasticity. Improving sleep during adolescence may capitalize on ongoing neurodevelopmental processes to facilitate brain plasticity and lessen the impact of neural vulnerabilities to depression and reduce the risk of subsequent depressive episodes.

Sleep complaints are a key symptom of MDD in children and adolescents [7], a and reduced quantity of sleep increases the risk for MDD three-fold in adolescents [8]. Sleep disturbance is associated with an increased risk of suicide [9]. Specifically, adolescents who sleep <6 hours are three times as likely to consider suicide, compared with those who sleep for 8 hours [10]. Furthermore, sleep is a behavior that undergoes considerable change during mid-adolescence: adolescents frequently obtain insufficient sleep, driven by a normative delay in circadian phase and altered homeostatic dynamics [11] and extracurricular activities and academic and social pressures, which results in a preference for later bedtimes [12]. When these are combined with early school start times, insufficient sleep ensues. Recently, links between greater electronic device use and poor sleep have been identified [13], and sleep loss may mediate relationships between media use and depressive symptoms [14]. However, although negative relationships between adolescent media use and mental health are commonly posited, recent studies using large samples have identified marginal relationships [15], and it remains to be seen whether such associations exist in early adolescence.

To date, longitudinal studies investigating associations between sleep and depression from early–late adolescence are sparse [16], with most studies focusing on mid-older rather than young (<11 years old) adolescent periods. Results from several prospective studies on mid-late adolescence have suggested sleep disturbance is a precursor to depression [17]; however, large-scale studies across early adolescence are required to further establish how poor sleep health may facilitate the onset of adolescent MDD. Here, we used data from the Adolescent Brain Cognitive Development (ABCD) study to assess how sleep disturbance and typical total sleep time (TST) are associated with depression symptoms at baseline and 1-year follow-up (1YFU) in early adolescents without a diagnosis of MDD. We hypothesized that sleep disturbances would predict depression 1 year later, particularly in girls, who are uniquely vulnerable during this developmental period [18].

Method

Participants

A total of 11,875 participants, aged 9–10 years, were enrolled across the 21 ABCD data collection sites, which cover 17 states (https://abcdstudy.org/study-sites/). The ABCD study used a rigorous epidemiologically informed school-based recruitment strategy, designed with consideration of the demographic composition of the 21 ABCD sites and the U.S. as a whole [19]. Exclusionary criteria were minimal and are listed in Supplementary Material with the site list. Baseline analyses included 11,670 participants with complete data on sleep disturbances, mental health scales, parent-reported screen time, and relevant covariates: age, sex, race, ethnicity, and parental socioeconomic status (SES). Longitudinal analyses are on a subset of the sample (4,951 participants) who had completed their 1YFU assessment by the time of the data release (ABCD 2.0 release: https://nda.nih.gov/study.html?id=634).

Procedure

All adult (parent/caregiver) participants provided written informed consent, and all youth provided written assent along with permission from a parent/legal guardian. Parents completed self-report measures in private via Redcap on an iPad. Youth worked separately with a research assistant to answer questionnaires and complete cognitive tasks and a magnetic resonance imaging session.

Measures

Demography. Parents answered a demographic questionnaire [20]. As multiple races could be selected per child, steps were taken to reduce the number of race categories to be used for statistical models, which resulted in six final race categories for statistical analyses: white, black, Asian, multiple, other, and unsure. Supplementary Material provide the procedures used for race category reduction. For all models, white was the reference category. Hispanic/Latino ethnicity was also assessed in all models, using non-Hispanic as the reference category. SES was estimated by identifying the maximum level of education across parents/guardians and categorized as follows: less than high school, high school/GED, some college, associate degree, bachelor/professional degree, and postgraduate degree. Bachelor/professional degree was the reference category. Although household income is available, it is correlated with site, which is already controlled for in the model as random effects variable, and thus, we chose to include parental education only as an estimator of SES.

Family history of depression was also estimated from the parent-report item *"Has ANY blood relative of your child ever suffered from depression, that is, have they felt so low for a period of at least two weeks that they hardly ate or slept or couldn't work or do whatever they usually do?"* For this analysis, if the parent endorsed a family history of depression for any relative, family history was recorded as "yes." In all models, no family history was the reference category.

Sleep. Sleep characteristics were assessed from the parent-report measure; Sleep Disturbance Scale for Children (SDSC) [21] designed to categorize sleep disorders in children. The SDSC is a 27-item inventory rated on a 5-point Likert-type scale that provides scores on five subdomains, of which we focused on three: disorders of initiating and maintaining sleep (DIMS), disorders of excessive somnolence (DOES), and sleep–wake transition disorders (SWTD) that have been previously associated with mental health.

The individual item "How many hours of sleep does your child get on most nights?" was used as the measure for typical TST (TST). Possible responses were (1) 9–11 hours, (2) 8–9 hours, (3) 7–8 hours, (4) 5–7 hours, and (5) <5 hours. Given the small number of participants in the fifth category (n = 34, .3% of the sample), we combined categories 4 and 5. In all models that included TST, 9–11 hours was the reference category because it is the recommended TST for this age group [22].

Mental health. Mental health variables were derived from the Child Behavior Checklist (CBCL) [23], a widely used, standardized measure for evaluating maladaptive behavioral and emotional problems in youth aged 4–18 years. Parents rate how true each item is now or was within the past 6 months using a 3-point Likert scale. The CBCL yields scores on internalizing (i.e., anxious, depressive, and overcontrolled), externalizing (i.e., aggressive, hyperactive, noncompliant, and undercontrolled), and total problems and scores on DSM-oriented scales. All scores were standardized *t*-scores, based on national norms, scaled so that 50 is average for the youth's age and gender, with a standard deviation of 10 points. Higher scores indicate greater problems. Of the 13 items within the DSM depression scale, four related to sleep. Removing these items resulted in small changes to the scale's raw scores (9,112 participants had zero change to their raw score, and the mean raw score change was 1.2 [SD: .9]) and thus, to use the *t* score measures, which were computed based on all items of the scales, we did not remove these items. For comparison, we also assessed the CBCL withdrawn/depressed subscale, which did not comprise any items relating to sleep.

Youth and parents also completed the Kiddie Schedule for Affective Disorders and Schizophrenia clinical interview, which was assessed to ensure that none of the participants included in the longitudinal analyses here met criteria for current MDD.

Screen time. Parents reported how much time their child spent using different types of electronic devices (computer, cell phone, tablet, iPod, and other devices) on a typical weekday (Monday to Friday) and weekend day (Saturday to Sunday). Parents were asked to not include time spent on school-related work but to include time spent watching TV or videos online, texting and chatting, playing games, and visiting social networking sites. A measure of total daily screen time for weekdays and weekends was then computed, which was time summed across the two reported times and used in the analysis.

Statistical procedure

Generalized additive mixed models (GAMMs) were used to investigate how sleep may be associated with mental health, using Stata software. The GAMM model is appropriate for both cross-sectional and longitudinal regression analyses and allows for an explicit modeling of aspects of the study design such as nesting of participants within sites and family. For each model, demographic factors (race, ethnicity, sex, and parental education), site, and twin/sibling status were included as covariates. For depression models, a family history of depression (yes/no) was also included as a covariate. As sleep disturbance and screen time measures were non-normally distributed, they were log transformed before inclusion in the models.

For baseline analyses, separate GAMMs assessed each mental health measure (internalizing, externalizing, and DSM depression). As sleep measures were moderately highly associated with each other, individual GAMMs assessed the relationship between each mental health and sleep variable separately (TST, DOES, DIMS, and SWTDs) and then model fit statistics were used to determine which sleep variable was best associated with each mental health variable.

For longitudinal analyses, similar GAMMs were run to assess mental health at follow-up. For each model, baseline sleep measures and covariates were included, as well as the baseline mental health measure (i.e., the GAMM assessing how baseline sleep disturbance predicts depression at follow-up included baseline depression as an additional covariate). The follow-up model for depression included sex interaction effects (sleep disturbance × sex and baseline depression × sex).

Results

Sample characteristics at baseline

At baseline, participants were aged 9–10.92 years, with a mean age of 9.9 years, and were typically prepubescent (Table 1). The sample consisted of an almost equal distribution of the sexes (47.9% female) and a majority of non-Hispanic participants (79.5%). Moreover, 63.5% of the participants were white, and approximately 60% of the sample had parental education status, which was a bachelor's/professional degree or higher. On average, participant's scores on the internalizing, externalizing, and DSM depression scales were in the normal range, with 3% scoring within the "clinical range" for the DSM depression scale, 9% for internalizing, and 6% for externalizing scales.

Sleep disturbance scales from the SDSC indicated low levels of problems, and average TST was 9–11 hours.

Demographic differences in sleep and mental health at baseline

Multinomial logistic regression analyses identified age, race, and parental education ($p < .001$) were significantly associated with TST; black and multiple race and older age were all associated with a greater likelihood of shorter TST. Bachelor's and postgraduate parental education was associated with a greater likelihood of longer TST. Specifically, 55% of both white and Asian participants were reported to have the recommended 9–11 hours TST, compared with 25% of black participants and 35% of Hispanic participants. Controlling for all other demographic variables, being Hispanic reduced the probability of being in category 9–11 (odds ratio [OR] .706, 95% confidence interval [CI] .626–.727) and increased the probability of being in category 7–8 (OR 1.34, 95% CI 1.13–1.59). Conversely, being multiple race reduced the probability of being in category 9–11 (OR .626, 95% CI .474–.827) and increased the odds of being in category <5 to 7 (OR 3.29, 95% CI 1.96–5.56). Being black reduced the probability of being in category 9–11 (OR .389, 95% CI .342–.443) and increased the probability of being in category <5 to 7 (OR 4.55, 95% CI 3.44–6.01; Figure 1).

Demographic variables alone accounted for a small proportion of variance in sleep disturbance variables (~1%), externalizing (3%), internalizing (2%), and depression (4%) measures and thus are not discussed further.

Table 1
Parent-reported demographic, mental health, sleep, and screen time use characteristics of the baseline Adolescent Brain Cognitive Development sample [24]

Measure	Frequency	Percent
Sex		
Female	5,594	47.9
Male	6,076	52.1
PDS		
Prepuberty	5,833	49.1
Early puberty	2,712	22.8
Midpuberty	2,872	22.5
Late puberty	170	1.4
Post puberty	10	.1
Ethnicity		
Hispanic	2,391	20.5
Not-Hispanic	9,279	79.5
Race		
White	7,411	63.5
Black	2,266	19.4
Asian	604	5.2
Multiple	292	2.5
Other	937	8.0
Unsure	160	1.4
Parental education		
Less than high school	574	4.9
High school/GED	1,104	9.5
Some college	1,485	12.7
Associate degree	1,550	13.3
Graduate/professional degree	3,547	30.4
Postgraduate degree	3,410	29.2
Total sleep time		
9–11 hours	5,543	47.5
8–9 hours	4,349	37.3
7–8 hours	1,384	11.9
<5 to 7 hours	394	3.4

Measure	Mean or median	Range	SD
Age (months)	119	108–131	7.5
BMI	17.7[a]	10.1–45	4.2
Internalizing (CBCL t-score)	48.4[a]	33–93	16.6
Externalizing (CBCL t-score)	44.0[a]	33–84	10.3
DSM5 Depression (CBCL t-score)	50.0[a]	50–89	5.7
DIMS (SDSC)	11[a]	7–35	3.8
DOES (SDSC)	6[a]	5–25	2.6
SWTD (SDSC)	7[a]	6–30	2.4
Weekday screen time (hours)	2	0–24	2.7
Weekend screen time (hours)	3.5	0–24	2.6

BMI = body mass index; CBCL = Child Behavior Check List; DIMS = disorders initiating and maintaining sleep; DOES = disorders of excessive somnolence; PDS = puberty status from the Pubertal Development Scale; SD = standard deviation; SDSC = Sleep Disturbance Scale for Children; SWTD = sleep–wake transition disorders.

[a] Median is reported instead of the mean because of the skewed distribution of the data.

Baseline associations between TST, sleep disturbance, and mental health

Sleep disturbance scores (DOES, DIMS, or SWTDs) accounted for 17%–24% of the variance in internalizing (R^2 = .19), externalizing (R^2 = .17), and depression scores (DSM scale: R^2 = .25, withdrawn/depressed scale: R^2 = .17), after adjusting for sociodemographic variables, site, and sibling/twin status. Although all sleep disturbance measures were significantly associated with mental health, model fit statistics (Bayesian Information Criteria) indicated marginally better fits when using different sleep disturbance variables; internalizing symptoms were most associated with DOES, whereas externalizing symptoms and depression scores were most associated with DIMS sleep

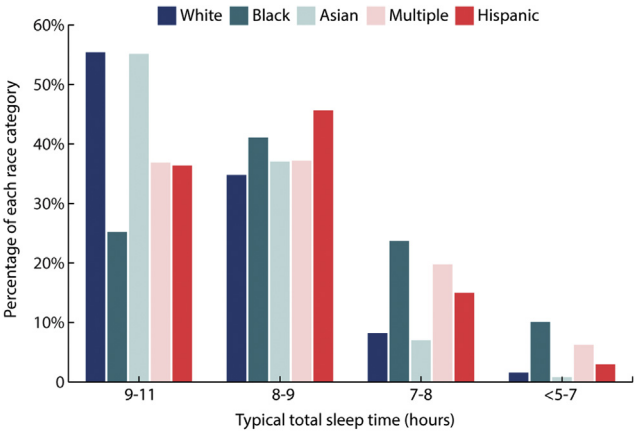


Figure 1. Proportion of each race category divided according to parent-reported total sleep time (TST) duration. After controlling for other demographic variables, black and multiple race participants were significantly more likely to belong to the shortest TST categories compared with white participants.

(Figure 2). Table 2 provides model statistics. In all cases, greater sleep disturbance was associated with poorer mental health (i.e., greater *t* score).

Models that included TST were significantly poorer fit compared with those that included sleep disturbance, accounting for 4%–9% of the variance in mental health scores (Table 2). Within TST models, all TST categories were significant ($p < .001$), indicating that those sleeping less than 9–11 hours had significantly poorer mental health scores, compared with those sleeping 9–11 hours. Greatest effects were always identified for the shortest TST category (<5–7 hours), compared with the longest (9–11 hours).

In all models, although total screen time was significantly associated with mental health score, including it did not result in a significant increase in model fit and increased the variance explained by less than 1%. Additional models that replaced total screen time with weekday or weekend screen time resulted in almost identical outcomes. As such, screen time was not included in the final models.

Longitudinal associations between sleep disturbance and mental health

For each model, after accounting for baseline mental health symptoms, baseline sleep disturbance significantly predicted mental health symptoms at 1YFU. In line with the results from the baseline analyses, models all performed similarly, with marginally better fits which indicated greater baseline DOES best predicted internalizing ($\beta = .07$; $p < .001$) and depression (DSM scale: $\beta = .13$, $P < .001$, withdrawn/depressed scale: $\beta = .08$, $P < .001$) scores at 1YFU and greater baseline DIMS best predicted externalizing scores at 1YFU ($\beta = .05$; $p < .001$; Figure 3). For the depression model, a significant DOES × sex interaction identified that the association between DOES and 1YFU depression was steeper for girls, compared with boys ($p < .001$; 95% CI 1.04–3.45), while holding all other variables constant. For each 1-unit increase in DOES, 1YFU depression increased by 3.7 units in boys and 8.2 units in girls (1YFU depression = 5.97 + 2.24 × sex (Coefficient for the sex interaction only is reported for brevity,

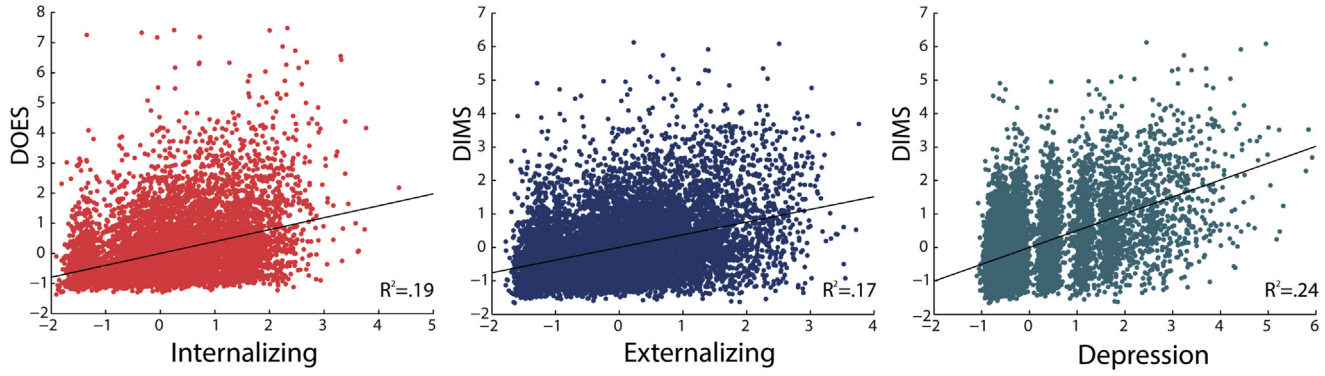


Figure 2. Associations between parent-reported sleep disturbance and mental health score in the ABCD sample at baseline. Child Behavior Checklist (CBCL) and Sleep Disturbance Scale for Children (SDSC) residuals are plotted after controlling for demographic variables (age, sex, race, ethnicity, parent education. Family history of depression was also included in the depression model), site, and twin/sibling status. DIMS: disorders initiating and maintaining sleep; DOES: disorders of excessive somnolence. The sleep disturbance that was best associated with each mental health score is presented. R^2 represents the full model (including all covariates above).

the full model included all covariates as listed in the Method), where boys are coded as −1 and girls as +1).

Short baseline TST (<5–7 hours) also significantly predicted externalizing symptoms at 1YFU ($\beta = .03$; $p = .003$) after controlling for baseline externalizing score. Greater baseline mental health scores were strongly associated with greater scores at 1YFU (internalizing $\beta = .57$, depression $\beta = .64$, and externalizing $\beta = .70$). Adding baseline screen time to the models did not significantly improve model fit.

For all analyses, models that replaced age with pubertal status, as assessed by the parental Pubertal Development Scale, performed almost identically, likely given the restricted range of pubertal development at this time point.

Discussion

Here we identified cross-sectional and longitudinal associations between sleep characteristics and mental health in a large sample of 9- and 10-year-olds. Sleep disturbance was a better predictor of depression symptoms than typical sleep duration, which should be interpreted cautiously, given the limitations of the single TST measure discussed below. We also found evidence that girls may be at greater risk of developing depression symptoms as a consequence of sleep disturbance, compared with boys.

Significant associations between sleep disturbance, depression, and internalizing and externalizing behaviors were seen even within this sample of adolescents who are, on average, within the healthy range for these mental health scales. Identifying that sleep predicts depression symptomology 1 year later, in the absence of clinical disorder, is important and suggests a potential mechanism for future preventative tools. Preventing adolescent MDD is of particular importance because it is associated with a lifelong trajectory of future depression episodes [25] and multiple negative outcomes, including lower educational attainment [26] substance abuse [27] and suicide [9].

Our results also show that those children with poorer mental health scores at baseline exhibited poorer scores at follow-up, which corresponds with the literature that early emerging, subclinical symptoms of depression are stable and predictive of future depressive disorders, particularly in girls [28]. Although we found little difference between boys and girls in internalizing or depression symptom scores at this young age, we identified sex differences in the relationship between sleep disturbance and depression. Although sleep disturbance predicted follow-up depression symptoms in boys and girls, girls exhibited almost twice the sleep disturbance associated increase in depression symptoms compared with boys when controlling for baseline depression and all demographic variables. This sex-specific interaction between sleep disturbance and depression at this early age is an important finding, given that by the end of adolescence, girls are almost three times as likely to have experienced an onset of depression compared with boys [18,29]. Although this sex difference in postpubertal onset is often attributed to hormonal differences, there is no conclusive evidence as to why girls are at higher risk of depression during this time. Likely, the reasons are multifactorial and encompass social and biological domains, including direct or indirect effects on the brain of pubertal sex hormones [30].

We found both types of sleep disorders, DIMS and excessive daytime somnolence, predicted future mental health symptoms. There were marginal differences between models, suggesting specific aspects of sleep disturbance may better predict depression (or other mental health disorders) in adolescence, which warrants further investigation. A large body of research has identified associations between excessive daytime somnolence and depression, largely in adults, and although the reason for this association remains unclear, it is possible that it may reflect disrupted circadian rhythm of sleep propensity [31]. Further research to establish whether excessive somnolence is a better predictor of adolescent depression onset, compared with sleep disturbance in general, is required. An important factor to consider when investigating sleep and depression is that given sleep issues and depression symptoms are commonly co-morbid, scales assessing depression symptomology often include sleep-related items. Thus, correlating sleep measures and depression scales may be subject to some bias. Here we found that sleep disturbance predicted two measures of depression symptomology, one that included some items related to sleep and one that did not. While DOES was the strongest predictor of follow-up depression, DIMS (which overlap less with specific depressive symptoms, e.g., having a hard time getting out of bed in the morning) still predicted a similar proportion of the variance in depression symptoms, suggesting that sleep disruption plays

Table 2
Outcomes for feneralized additive mixed model models assessing cross-sectional associations between baseline sleep and mental health in the Adolescent Brain Cognitive Development sample

Baseline model statistics
Covariates: age, sex, race, ethnicity, parent education, site, and twin/sibling status. Family history of depression was also included in the depression models

	Internalizing	Externalizing	Depression
DIMS	BIC: 85,982	**BIC: 85,468**	**BIC: 67,757**
	R^2: .18	**R^2: .17**	**R^2: .25**
	β: .41	**β: .39**	**β: .46**
	CI: 31.51–34.33	**CI: 28.32–32.60**	**CI: 20.10–21.61**
DOES	**BIC: 85,938**	BIC: 85,514	BIC: 68,165
	R^2: .19	R^2: .17	R^2: .22
	β: .41	β: .37	β: .43
	CI: 31.0–33.74	CI: 27.256–29.95	CI: 18.06–19.57
SWTD	BIC: 86,243	BIC: 85,895	BIC: 69,208
	R^2: .15	R^2: .12	R^2: .12
	β: .36	β: .31	β: .30
	CI: 28.73–31.73	CI: 23.35–26.31	CI: 12.94–14.64
TST	BIC: 87,729	BIC: 86,963	BIC: 69,773
	R^2: .03	R^2: .04	R^2: .08
	β (7–8 hours): .08	β (7–8 hours): .08	β (7–8 hours): .15
	CI: 1.86–3.18	CI: 1.78–3.05	CI: 2.30–3.0
	β (<5–7 hours): .12	β (<5–7 hours): .12	β (<5–7 hours): .21
	CI: 5.54–7.75	CI: 5.71–7.85	CI: 6.15–7.35

Longitudinal model statistics
Covariates: baseline mental health score, age, sex, race, ethnicity, parent education, site and twin/sibling status. Family history of depression was also included in the depression models

	Internalizing	Externalizing	Depression
DIMS	BIC: 33,708	**BIC: 32,552**	BIC: 28,882
	R^2: .47	**R^2: .54**	R^2: .41
	β: .07	**β: .05**	β: .11
	CI: 4.45–8.33	**CI: 2.07–5.47**	CI: 4.41–6.75
DOES	**BIC: 33,578**	BIC: 32,561	**BIC: 28,859**
	R^2: .47	R^2: .54	**R^2: .41**
	β: .07	β: .05	**β: .13**
	CI: 3.89–7.74	CI: 2.16–5.55	**CI: 4.79–7.15**
SWTD	BIC: 33,657	BIC: 32,507	BIC: 28,870
	R^2: .47	R^2: .54	R^2: .40
	β: .06	β: .03	β: .07
	CI: 3.20–7.14	CI: .96–4.41	CI: 2.36–4.72
TST	BIC: 33,766	BIC: 32,585	BIC: 28,974
	R^2: .46	R^2: .54	R^2: .40
	β (7–8 hours): $.01^{NS}$	β (7–8 hours): .03	β (7–8 hours): $.02^{NS}$
	CI: −.29 to 1.28	CI: .29 to 1.67	CI: .04 to 1.0
	β (<5 to 7 hours): $.001^{NS}$	β (<5 to 7 hours): .03	β (<5 to 7 hours): $.02^{NS}$
	CI: −1.42 to 1.57	CI: .47 to 3.12	CI: −.08 to 1.77

R^2 is reported for the full model.
As TST was a categorical variable, β and CI are reported for two categories (7–8 hours and <5–7 hours), the longest TST category (9–11 hours, the recommended TST for 9- to 10-year-olds [20]) was the reference category in the TST models. Models with the best fit for each mental health score are highlighted in bold.
β = standardized beta for the sleep variable in the model; BIC = Bayesian Information Criteria to assess model fit; BMI = body mass index; CI = confidence interval for the sleep variable in the model; NS = not significant.

some role in future depressive symptomology and that results are not driven by largely overlapping measures. These associations are likely complex and could reflect shared genes and/or environmental variables or could reflect parental tendencies to rather attribute emotional difficulties to overtiredness [32].

We did not find evidence to suggest that screen time contributed to predictions of future mental health, supporting recent studies that did not find substantial negative associations between media use and well-being [15] or depression [33] in older adolescents. Although common public opinion is that excessive screen time has negative consequences on mental health or cognition, and some links have been identified by some [14], the scientific field is relatively new, and currently, findings are inconclusive. More research is required, using more objective measures of screen time to ascertain exactly how specific aspects of screen time behavior (e.g., social media vs. gaming) may be related to a clinical manifestation of depression and whether it has greater consequences for those already at risk.

One major limitation of this work is the fact that youth behaviors and symptoms were ascertained from parent report, which may be subject to shared method variance. Furthermore, the sleep disturbance and mental health measures were similar in format (health-valenced items on a Likert-type scale), which may have elicited reporter-specific response patterns that may inflate associations between measures. Reliance on parent report is a common limitation of pediatric research and can result in either under- or over-reporting of the child's behaviors and symptoms. In addition, although analyses identified that TST was less of a predictor compared with sleep disturbance measures, several limitations exist with the current TST measure. Unlike the sleep disturbance measures, TST was estimated from a single question and was an ordinal variable that collapsed across multihour categories and did not distinguish between weekdays and weekends. Additional measures of sleep (both objective and more fine-grained continuous and subjective measures on weekdays and weekends), which have been added to the ABCD protocol, will be available for future analyses eliminating the need to rely solely on parent report measures. These future annual waves of ABCD data will facilitate the exploration of how multiple measures of sleep may be differentially (or similarly) related to mental health outcomes, as discussed by others [34], and will allow us to assess how short sleep and daytime somnolence could potentially share a common underlying construct such as inappropriate sleep schedules [35]. Finally, parent report of family history of depression was not verified against clinical diagnosis and thus may not be entirely accurate. Despite these measurement limitations, the present study provides preliminary evidence that a modifiable behavior (sleep) predicts depression (and externalizing) symptomology early in development and before any clinical diagnoses, suggesting a potential preventative mechanism, which warrants future clinical investigation.

In this diverse sample, we identified race differences in TST, consistent with previous studies in children and adults: we identified that black and multirace children were significantly more likely to experience shorter TST, compared with white participants. Only 25% of black participants and 35% of Hispanic participants in the sample were reported to typically achieve the recommended sleep duration for their age (9–11 hours). Although one study in adults identified a genetic component to race differences in slow-wave sleep and slow-wave electroencephalography power [36], which may reflect genetic differences in brain structure, they did not find associations with sleep duration or quality. Instead, most studies that have identified poorer sleep in ethnic minorities have suggested that these differences are driven largely by health and environmental disparities between races [37]. Although a small number of studies have reported similar disparities in children [38–40], longitudinal studies are required to establish whether

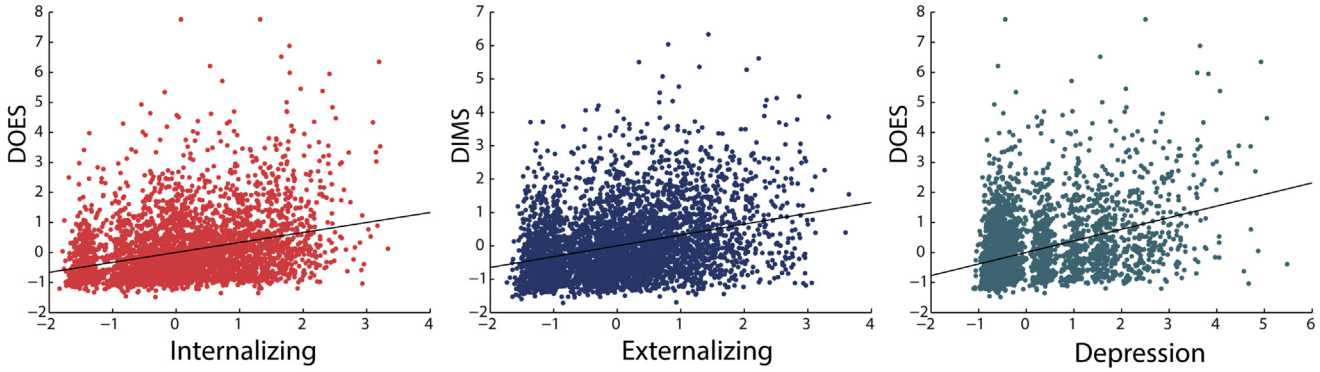


Figure 3. Associations between parent-reported sleep disturbance and mental health score in the ABCD sample at 1-year follow-up (1YFU). 1YFU Child Behavior Checklist (CBCL) and baseline Sleep Disturbance Scale residuals are plotted after controlling for demographic variables (age, sex, race, ethnicity, parent education. Family history of depression was also included in the depression model), site, and twin/sibling status. DIMS: disorders initiating and maintaining sleep; DOES: disorders of excessive somnolence. The sleep disturbance that was best associated with each mental health score at follow-up is presented.

differences identified in childhood are indicative of disparities than persist across the lifetime. Considering the vital role of sleep in maintaining brain plasticity, particularly during the adolescent period when vast brain developmental change occurs, and the links between poor quality sleep and negative outcomes (depression, suicide, substance use, and poor academic achievement), early identification of adolescents at risk for short, or poor quality, sleep is vital. As we consider sleep as a potential tool to limit the trajectory of depression, we must also address underlying health disparities and psychosocial factors that further complicate, yet necessitate, the need to identify at-risk youth.

Furthermore, it should also be noted that although the ABCD study is a large, nationwide study, its sample should not be considered entirely representative of U.S. youth. Although great efforts were taken to recruit typically under-represented race and socioeconomic groups and to minimize systematic sampling biases in recruitment at the school level, the sample is still skewed by several factors [24]. Nonetheless, the ABCD study consists of a population-based, demographically diverse sample, which provides opportunities for scientific discovery that will be generalizable to a large majority of U.S. youth. As the currently released longitudinal data does not include the full sample ($n = 4{,}951$ of 11,670 included for the baseline analyses), it is worth noting that the longitudinal sample characteristics were largely consistent with the full, baseline sample (Supplementary Table 1), although there was a lower proportion of black participants and families with lower parental education. It is unlikely that these small demographic differences affected the longitudinal outcomes.

In summary, we identified cross-sectional and longitudinal relationships between sleep and mental health in a large sample of 9- and 10-year-olds in the early stages of adolescence. Although further research is required, findings suggest that sleep, as a modifiable behavior, could be targeted early in childhood to disrupt otherwise detrimental developmental trajectories that would result in an onset of depression during adolescence. The associated brain developmental processes during adolescence may offer a unique and critical window to target such interventions, which warrants further investigation to address the burgeoning prevalence of adolescent depression in today's world.

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Supplementary Data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.jadohealth.2019.12.005.

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