Effects of sleep duration on neurocognitive development in early adolescents in the USA: a propensity score matched, longitudinal, observational study

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Summary

Background Although the American Academy of Sleep Medicine suggests at least 9 h of sleep per day for 6–12-year-olds, children in recent generations often report sleeping less than this amount. Because early adolescence is a crucial period for neurocognitive development, we aimed to investigate how insufficient sleep affects children's mental health, cognition, brain function, and brain structure over 2 years.

Methods In this propensity score matched, longitudinal, observational cohort study, we obtained data from a population-based sample of 9–10-year-olds from 21 US study sites in the ongoing Adolescent Brain Cognitive Development (ABCD) study. Participants were categorised as having sufficient sleep or insufficient sleep on the basis of a cutoff of 9 h sleep per day. Using propensity score matching, we matched these two groups of participants on 11 key covariates, including sex, socioeconomic status, and puberty status. Participants were excluded from our analysis if they did not pass a baseline resting-state functional MRI quality check or had missing data for the covariates involved in propensity score matching. Outcome measures retrieved from the ABCD study were behavioural problems, mental health, cognition, and structural and resting-state functional brain measures, assessed at baseline and at 2-year follow-up. We examined group differences on these outcomes over those 2 years among all eligible participants. We then did mediation analyses of the neural correlates of behavioural changes induced by insufficient sleep.

Findings Between Sept 1, 2016, and Oct 15, 2018, 11 878 individuals had baseline data collected for the ABCD study, of whom 8323 were eligible and included in this study (4142 participants in the sufficient sleep group and 4181 in the insufficient sleep group). Follow-up data were collected from July 30, 2018, to Jan 15, 2020. We identified 3021 matched sufficient sleep–insufficient sleep pairs at baseline and 749 matched pairs at 2-year follow-up, and observed similar differences between the groups in behaviour and neural measures at both timepoints; the effect sizes of between-group differences in behavioural measures at these two timepoints were significantly correlated with each other (r=0·85, 95% CI 0·73–0·92; p<0·0001). A similar pattern was observed in resting-state functional connectivity (r=0·54, 0·45–0·61; p<0·0001) and in structural measures (eg, in grey matter volume r=0·61, 0·51–0·69; p<0·0001). We found that cortico–basal ganglia functional connections mediate the effects of insufficient sleep on depression, thought problems, and crystallised intelligence, and that structural properties of the anterior temporal lobe mediate the effect of insufficient sleep on crystallised intelligence.

Interpretation These results provide population-level evidence for the long-lasting effect of insufficient sleep on neurocognitive development in early adolescence. These findings highlight the value of early sleep intervention to improve early adolescents' long-term developmental outcomes.

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Introduction

Young adolescents frequently report insufficient and poor sleep.[1] Because the brain maturation process is vulnerable to sleep loss,[2] young adolescents with insufficient sleep often show compromised neurocognitive functions, manifested as poorer academic performance and reduced social-emotional skills compared with their peers.[3] However, the neural mechanisms underlying the adverse effects of insufficient sleep on adolescent development are poorly understood.[4] This could be due to many reasons. First, several covariates, such as socioeconomic status,[5] sex,[6] and pubertal status,[7] can substantially influence adolescents' sleep patterns and their neurocognitive functions. For example, a child's sleep duration and household income are often highly correlated (appendix p 2), which would make it difficult to isolate the direct effect of insufficient sleep on neurocognitive development. Second, because previous studies have primarily used cross-sectional designs,[7,8] it is unknown whether insufficient sleep has a temporary or long-lasting effect on early adolescent development. For instance, although the American Academy of Sleep Medicine has

See Online for appendix

Research in context

Evidence before this study

Few studies have estimated the long-lasting effect of insufficient sleep on the developing brain. Of those few publications, most reported data have not simultaneously controlled for several key covariates, such as sex, socioeconomic status, and pubertal status. We searched PubMed and Google Scholar on March 28, 2022, using the terms "sleep", "sleep duration", "brain", "matched/matching", "control", "child/children", "adolescent", "youth", "young", and "longitudinal" from inception and with no language restrictions. We did not identify any matched cohort studies with a longitudinal design that estimated the long-lasting effect of insufficient sleep on early adolescents' neurocognitive development. We also did not find any studies that have considered both structural and functional brain measures in a longitudinal design.

Added value of this study

This was a large cohort study of early adolescents with well matched covariates based on propensity score matching. This approach controls for several key covariates that might influence the relationship between sleep duration and neurocognitive development, thus better clarifies the direct effect of insufficient sleep on adolescent development. Furthermore, by examining these matched group differences at baseline and at 2-year follow-up, our data provide much-needed insights into the long-lasting effect of insufficient sleep on early adolescents' neurocognitive development.

Implications of all the available evidence

Insufficient sleep can modulate neural development profiles over 2 years, leading to compromised cognitive functions and more behavioural problems in early adolescents. Our study solidifies these findings by ruling out various key covariates such as sex, socioeconomic status, and pubertal status in existing evidence. By clarifying these direct effects of insufficient sleep on neurocognitive development, our findings highlight the crucial need for early intervention to facilitate long-term developmental outcomes in adolescents.

suggested 9–12 h of sleep per day for 6–12-year-old children,[9] it is unclear how less than 9 h of sleep would affect children's brain and behaviour over time. Clarification of these issues is crucial for understanding neurocognitive vulnerability and resilience to insufficient sleep in the developing brain.[10] It will also inform early sleep interventions to improve long-term developmental outcomes in adolescents.

Therefore, in this study we applied propensity score matching to investigate how insufficient sleep affects the brain and behaviour in early adolescents across 2 years after controlling for various key covariates, such as age, sex, race, socioeconomic status, and pubertal status.[11] We hypothesised that insufficient sleep would have long-lasting adverse effects on participants' neurocognitive developmental outcomes captured by behavioural, functional brain, and structural brain measures. In addition, motivated by previous research,[12] we predicted that brain function and structure should mediate the long-term effects of sleep duration on early adolescents' cognitive and affective functions.

Methods

Study design and data source

In this propensity score matched, longitudinal, observational cohort study, we used data from a population-based sample of 9–10-year-olds from 21 US study sites in the ongoing Adolescent Brain Cognitive Development (ABCD) study.[13] Data used in the current study were from the ABCD data release 3.0 (baseline data collection Sept 1, 2016, to Oct 15, 2018; follow-up data collection July 30, 2018, to Jan 15, 2020), which includes behavioural and neural data collected at baseline, 1-year follow-up, and 2-year follow-up. Detailed protocols and designs of the ABCD study have been described previously.[13] Participants were recruited to the ABCD study using stratified sampling to reflect the diversity of the US population. Participants were excluded from our data analysis if they did not pass a baseline resting-state functional MRI quality check or had missing data for the covariates involved in propensity score matching (appendix pp 3–4). Written informed consent from the primary caregiver and assent from the children were obtained before the ABCD study. This study was approved by the Institutional Review Boards of local study sites (21 in total). The ABCD study received ethics approval in accordance with the ethical standards of the 1964 Declaration of Helsinki.

For information on the ABCD study see https://abcdstudy.org

Procedures

We estimated a child's sleep duration as an independent variable based on the parent-reported Sleep Disturbance Scale for Children administered at baseline, 1-year follow-up, and 2-year follow-up. We used the data from the question, "How many hours of sleep does your child get on most nights in the past six months?", which provide estimates of a child's sleep duration in five categories (≥9 h, 8–9 h, 7–8 h, 5–7 h, <5 h; appendix p 2). Based on the recommendation by the American Academy of Sleep Medicine, we identified children with less than 9 h of sleep per day at baseline as the insufficient sleep group and children with 9 h or more of sleep per day as the sufficient sleep group.[9]

We then included dependent variables of interest from the following four categories: behavioural problems, cognition, mental health, and brain measures. For behaviour, we used the parent-reported Child Behaviour Checklist at baseline, 1-year follow-up, and 2-year follow-up to measure a child's behavioural problems in

emotional, social, and behavioural domains. For cognition, we used scores from the US National Institutes of Health (NIH) Cognition Battery Toolbox at baseline and 2-year follow-up to assess a child's general cognitive function. We estimated a child's overall mental health at baseline and 2-year follow-up on the basis of scores from the brief child version of the Prodromal Psychosis Scale;[14] the youth version of the Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency (UPPS-P) Impulsive Behaviour Scale;[15] and the Behavioural Inhibition Scale (a summary of these variable names used in the ABCD dataset is shown in appendix pp 14–15). For brain measures, all children had a standardised resting-state functional MRI scan, a structural MRI scan, and a diffusion tensor imaging scan at baseline and 2-year follow-up. Acquired images were processed and quality controlled at the Data Analysis, Informatics, and Resource Centre of the ABCD study.[16] Resting-state functional connectivity (rs-FC) of cortical networks was calculated as the average Fisher-transformed correlation between the time courses of each pair of regions within or between 12 cortical networks defined by the Gordon atlas. In addition, rs-FC between the 12 cortical networks and 19 subcortical regions were also calculated (appendix pp 10–11, 19–28). In total, there were 306 unique functional connectivity measures. Structural measures, including cortical area, grey matter volume, and cortical thickness from 148 regions were extracted on the basis of the Destrieux parcellation (appendix pp 28–39). Subcortical volumes of 36 regions and fractional anisotropy of 35 tracts from diffusion tensor imaging were also calculated on the basis of protocols in the ABCD study (appendix pp 40–41).

We included the following covariates in propensity score matching (appendix p 5): (1) basic demographic characteristics of a participant (age in months, sex, the interaction between age and sex, race, and study sites); (2) theoretically relevant factors in adolescent sleep patterns, including parent educational level and household income,[5] pubertal status[7] (assessed by ABCD Youth Pubertal Development Scale and Menstrual Cycle Survey History), and BMI;[17] and (3) functional MRI data quality indices, including average motion during resting scans (mean framewise-displacement) and the number of functional MRI timepoints that remained after preprocessing, as both head motion and image quality are crucial for interpreting paediatric structural and functional neuroimaging.[18]

Statistical analysis

At the outset, we did propensity score matching to control for covariates in the observational data so that the causal effects of insufficient sleep on the outcome measures could be estimated. We used the MatchIt R package (version 4.3.0)[11] for the propensity score matching. Participants were matched on the basis of the probability of being in a comparison group conditioned on observed covariates using logistic regression. As previously mentioned, these covariates included demographic variables, theoretically relevant factors, and data quality indices. We then matched participants with sufficient sleep versus insufficient sleep based on one-to-one matching without replacement within a predefined propensity score radius (ie, caliper=0·1). To check for matching quality, we compared the standardised mean difference of covariates between the sufficient sleep and insufficient sleep groups and found that all the covariates were well balanced between groups after matching (appendix p 6). Hence, additional group differences could not be attributed to these covariates. An independent-sample t test was used to assess these group differences, because this test tends to yield a more conservative effect size estimate compared with a paired-sample t test.[19] Because the effect of insufficient sleep on neurocognitive functions might be cumulative and even irreversible as suggested by animal studies,[20] we expected that participants with initial insufficient sleep would show adverse effects on behavioural and brain measures at follow-up assessments compared with the sufficient sleep group. Hence, we retained the group assignment for these well matched pairs in subsequent analyses to investigate the long-term effects of insufficient sleep on adolescent neurocognitive development. We next tested how insufficient sleep influenced the 42 behavioural measurements that represent adolescent behavioural problems (eg, aggression and rule-breaking; 20 items), cognitive functions (eg, fluid and crystallised intelligence; ten items), and mental health (eg, psychosis and impulsivity; 12 items) at baseline and at 2-year follow-up. For neuroimaging data, we focused on available participant pairs that had passed quality control for each corresponding brain measure.[16] Multiple comparisons were corrected for baseline measures based on false discovery rate. To test the extent to which sleep duration has similar effects on behavioural and neural outcome measures over time, we directly correlated the effect sizes of sleep duration with outcome measures at baseline and 2-year follow-up.[21]

Motivated by the conceptual framework that brain measures would mediate the relationship between sleep patterns and development outcomes,[12] we also tested whether baseline brain measures can mediate the effect of baseline sleep duration on behavioural measures at baseline and 2-year follow-up after controlling for the 11 covariates that were used in propensity score matching (appendix p 7). For the analyses of 2-year follow-up data, the corresponding baseline behavioural measure was added as an additional covariate. This time-lag analysis further shows whether the identified brain measures can serve as biomarkers of behavioural changes over time.[22] Of primary interest, we focused on brain rs-FC and behaviour assessments that showed a Cohen's d greater than 0·15 between the sufficient sleep and insufficient sleep groups, which is 50% higher than a typical effect size identified

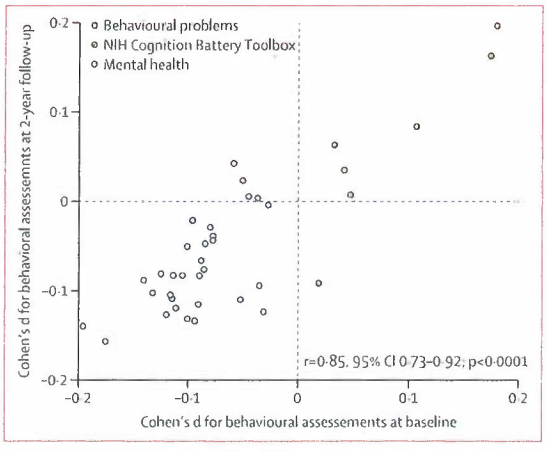

Figure 1: **The effects of insufficient sleep on behavioural measures**
Cohen's d for behavioural problems, cognition, and mental health in the comparisons between the sufficient sleep and insufficient sleep groups at baseline and at 2-year follow-up (appendix p 9). NIH=National Institutes of Health.

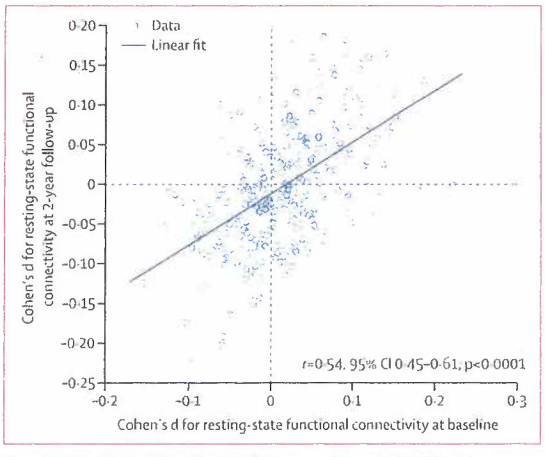

Figure 2: **The effects of insufficient sleep on resting-state functional connectivity**
Cohen's d for resting-state functional connectivity measures in the comparisons between the sufficient sleep and insufficient sleep groups at baseline and at 2-year follow-up (appendix pp 10–11).

based on the ABCD dataset (Cohen's d 0·03–0·09).[23] This application of effect size as a threshold measure might improve replicability in neuroimaging findings.[24] We implemented these analyses via an established neuroimaging mediation toolbox.[25] The significance of these mediation analyses was bootstrapped with 100 000 random-generated samples.

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

Of 11 878 individuals with data collected for the ABCD study, 1064 were excluded due to missing data, and 2491 were excluded because they did not pass the baseline resting-state functional MRI quality check. After exclusion, 8323 eligible participants were included in this study (appendix p 4), 4153 (49·9%) of whom were female and 4170 (50·1%) were male. There were 4142 participants in the sufficient sleep group and 4181 participants in the insufficient sleep group.

We first examined how participants' sleep patterns changed over time. Based on baseline measures, we matched participants in the sufficient sleep group with those in the insufficient sleep group on 11 key covariates using propensity score matching (appendix p 6). We identified 3021 matching pairs at baseline with both behavioural and neuroimaging data, of which 2762 pairs had behavioural data at 2-year follow-up and 749 pairs had neuroimaging data at 2-year follow-up. In follow-up assessments, we found that participants in the sufficient sleep group tended to gradually sleep less over 2 years, whereas sleep patterns of participants in the insufficient sleep group remained relatively stable (<20% change; appendix p 8).

We next tested how insufficient sleep influenced the 42 behavioural measurements that represent adolescent behavioural problems. Consistent with previous findings,[8] we found that insufficient sleep had widespread effects on baseline behavioural measures. Specifically, 32 of these 42 assessments showed a significant difference at baseline between the sufficient sleep and insufficient sleep groups after propensity score matching on covariates (all false discovery rate corrected p<0·05; appendix pp 9, 18–19). Among these effects, four behavioural measures had an absolute Cohen's d value greater than 0·15, including depression, thought problems, picture–vocabulary test performance, and crystallised intelligence. Additionally, the effect size of crystallised intelligence was about twice that of fluid intelligence (Cohen's d 0·17 *vs* 0·08; appendix p 9). We observed similar patterns at the 2-year follow-up, with similar effect sizes as those at baseline (appendix p 9). To quantify the extent to which insufficient sleep had similar effects on these behavioural measures at both timepoints,[21] we correlated the Cohen's ds of the difference between the sufficient sleep and insufficient sleep groups at 2-year follow-up and at baseline for all 42 behavioural measures. We found that these effects were significantly correlated with one another (r=0·85, 95% CI 0·73–0·92; p<0·0001; figure 1), suggesting that insufficient sleep has stable effects on adolescents' behavioural problems, neurocognition, and mental health over 2 years. Although the 1-year follow-up included only measures of behavioural problems, we observed similar findings between baseline and 1-year follow-up assessments (appendix p 10).

We next examined how sleep duration affects the intrinsic functional organisation of brain networks in the developing

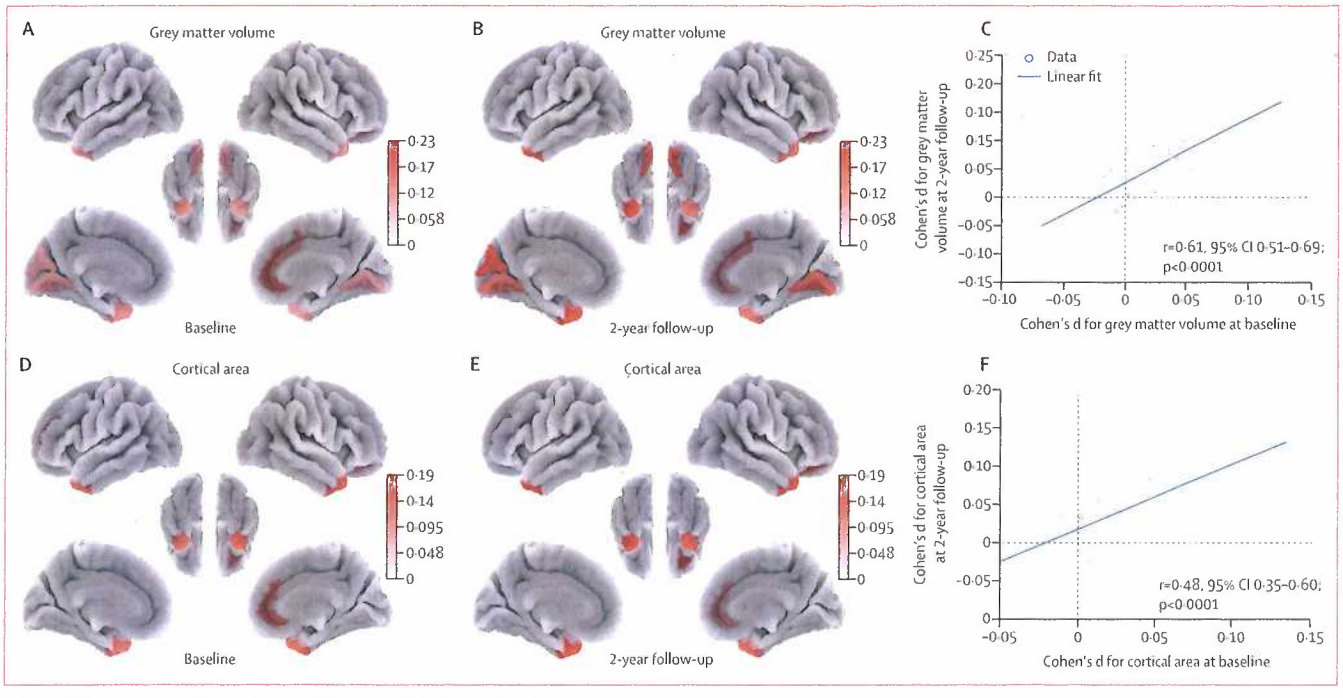

Figure 3: **The effects of insufficient sleep on brain structural measurements**
(A) Cohen's d for grey matter volume at baseline. (B) Cohen's d for grey matter volume at 2-year follow-up. (C) Correlation between the effect of insufficient sleep on grey matter volume measures at baseline and 2-year follow-up. (D) Cohen's d for cortical area at baseline. (E) Cohen's d for cortical area at 2-year follow-up. (F) Correlation between the effect of insufficient sleep on cortical area measures at baseline and 2-year follow-up. Only regions that passed false discovery rate correction at baseline are shown in panels A, B, D, and E.

brain at baseline and at 2-year follow-up. At baseline, we found that 93 of the 306 unique functional connections showed a significant difference between the sufficient sleep and insufficient sleep groups (all false discovery rate corrected p<0·05; appendix pp 10–11, 19–28). Noticeably, four out of the five connectivities that had a Cohen's d value greater than 0·15 involved regions of the basal ganglia, which plays a core role in regulating the sleep–wake cycle.[16] Additionally, the effect sizes of sleep duration on all 306 available functional connectivities were significantly correlated with that in the 2-year follow-up (r=0·54, 95% CI 0·45–0·61; p<0·0001; figure 2). These results suggest that certain functional connectivity measures are consistently susceptible to insufficient sleep over time.

We further investigated how sleep duration affects the development of brain structures at baseline and at 2-year follow-up. At baseline, we found that grey matter volume in 12 of 184 regions showed a significant difference between the sufficient sleep and insufficient sleep groups (all false discovery rate corrected p<0·05; figure 3A; appendix pp 28–33). A similar pattern was found at the 2-year follow-up (figure 3B). The pattern of Cohen's d for grey matter volume in the 184 regions at baseline was significantly correlated with that at the 2-year follow-up (r=0·61, 95% CI 0·51–0·69, p<0·0001; figure 3C), suggesting that some structural measures are consistently susceptible to insufficient sleep over time. Although we obtained similar results based on cortical areas (r=0·48, 95% CI 0·35–0·60, p<0·0001; figure 3D–F; all false discovery rate corrected p<0·05; appendix pp 33–36), we did not observe statistically significant differences between sufficient sleep and insufficient sleep groups in measures of cortical thickness or fractional anisotropy (all false discovery rate corrected p>0·05; appendix pp 36–41). Although not significant, three white matter tracts connect the striatum in the basal ganglia with the cerebral cortex had highest effect size (appendix p 11).

Finally, to assess how brain measures mediate the relationship between sleep patterns and behavioural outcomes, we tested whether the brain measures identified with larger effect sizes in respective categories at baseline (ie, five rs-FC measurements, three cortical areas, and three grey matter volume measurements, appendix p 12) mediated the effects of sleep duration on the four behavioural measures identified with a Cohen's d greater than 0·15 (ie, depression, thought problems, picture–vocabulary test performance, and crystallised intelligence). In this analysis, we regressed out covariances used in the propensity score matching. At baseline, we found that the identified functional and structural brain measures robustly mediated the effects of sleep duration on picture–vocabulary test performance and crystallised intelligence (all bootstrapped p<0·05; appendix pp 12, 41). Three of five rs-FC measures also mediated the effect of

insufficient sleep on depression and thought problems (all bootstrapped p<0·05; appendix pp 12, 41). In addition, we identified distributed mediation patterns across all behavioural measures (appendix p 13).

To investigate the robustness of these observations over time, we did a longitudinal mediation analysis.[22] We found that some of the identified functional and structural measures mediated the effects of sleep duration on thought problems (ie, retrosplenial-temporal network—right ventral diencephalon connectivity), picture–vocabulary test performance, and crystallised intelligence (ie, retrosplenial-temporal network—right ventral diencephalon connectivity and left and right temporal pole structural measures; appendix pp 12–13).

Discussion

By controlling for various key covariates of sleep duration, we estimated the long-term effect of insufficient sleep on neurocognitive development in early adolescence. In contrast to recent adolescent sleep research,[1,8] this study identified two important neural mechanisms of insufficient sleep on neurocognitive development that have not previously been examined systematically in the literature. First, we showed that changes in rs-FC between the basal ganglia and cortical regions might underlie the widespread adverse behavioural effects induced by insufficient sleep. Second, our findings suggest that structural properties of the temporal pole mediate the effects of sleep duration on crystallised intelligence. These effects lasted for at least 2 years, suggesting long-lasting consequences of insufficient sleep on adolescents' neurocognitive development. If confirmed, our findings might provide empirical and theoretical groundings for early sleep intervention programmes to improve long-term developmental outcomes in adolescence.

The effects of insufficient sleep on rs-FC converged on the connections between the basal ganglia and cortical regions (see appendix p 11 for the effects on structural connectivities in the corticostriate tracts). The basal ganglia, along with the prefrontal cortex, undergoes the greatest structural changes across the whole brain during adolescence.[26] The basal ganglia has a key role in regulating sleep–wake behaviour through cortical activation.[26] Insufficient sleep might reciprocally disrupt normal functioning of the basal ganglia, such as through the dopamine or adenosine signalling pathway, and subsequently impair the cortex–basal ganglia–thalamus–cortex circuit.[27] This disruption might result in weakened attention and poor information processing, leading to impairment in cognitive and affective functions.[27] However, the durability of these detrimental effects has been unknown until now.[4] Here, using well matched samples, we show that disrupted basal ganglia–cortex connections could last for at least 2 years. These connections also mediate the effect of insufficient sleep on depressed mood, thought problems, and crystallised intelligence.

Furthermore, given the increased statistical power and well balanced covariates using propensity score matching, our results resolve the previously mixed findings in the literature on the relationship between insufficient sleep and intelligence.[1] We found that insufficient sleep had a long-lasting effect on crystallised intelligence; the magnitude of this effect was about twice that of fluid intelligence (Cohen's d 0·17 *vs* 0·08). Our mediation analyses showed that this behavioural effect was associated with changes in core neural substrates underlying the representation (temporal pole in the anterior temporal lobe)[28,29] and retrieval (retrosplenial temporal network)[10] of structuralised knowledge. In particular, we found that structural properties of the anterior temporal lobe and the retrosplenial temporal network mediated the long-term detrimental effects of insufficient sleep on picture–vocabulary test performance and crystallised intelligence. These findings raise the possibility that insufficient sleep might disrupt memory consolidation by delaying temporal lobe maturation in the adolescent brain.[2,11,12] Future research could examine how insufficient sleep influences the formation of crystallised knowledge at even younger ages.[13]

Another important contribution of this study is the estimated effect of insufficient sleep on neurocognitive development based on propensity score matching that resembles randomised experiments.[14] Because key covariates that might influence sleep duration and neurocognitive measures were well balanced between the sufficient sleep and insufficient sleep groups, the group comparison cannot be accounted for by covariates, such as socioeconomic status,[5] sex,[6] pubertal status,[7] and urbanity approximated by study sites. Therefore, this approach resolves the uncertainty about whether sleep-related effects on neurocognitive development can be attributed to these individual differences. In addition, although 9 h or more was selected by paediatricians and sleep experts as a reasonable amount of sleep for early adolescents, evidence supporting this recommendation has been mixed.[15] Our data provide clear evidence that 9 h or more of sleep is beneficial for neurocognitive development in early adolescents, and hence substantiate this recommended sleep duration for this age group.

A few limitations of our study should be noted. First, one caveat of propensity score matching is that it can only control for observed covariates, leaving unobserved variances that might influence the selection of matched pairs. A subset of participants could not be matched based on the current method (appendix p 6), suggesting that additional factors might introduce further variability. For example, different school programmes have been used to improve students' sleep habits,[16] but the ABCD dataset has little information related to these variables. Second, although propensity score matching offers an approach to draw causal inferences on the basis of observational data, the causal relationship needs to be confirmed with experimental approaches.

For example, future research could examine whether school programmes designed to prolong adolescents' sleep duration could improve adolescents' long-term developmental outcomes.[4] Third, with data from only two timepoints for most measures, the current dataset is not adequate to distinguish within-person and between-person effects on the association between sleep duration and behavioural and neural measures. Because the ABCD study is ongoing, future research incorporating data from more follow-up visits could better address this issue. Fourth, other sleep measures, such as architectural and microstructural features of sleep, as well as circadian rhythms, might also contribute to the relationship between sleep, brain, and behaviour. Future studies need to investigate how these features of sleep (based on wearable, electronic, activity-tracking devices) are related to neurocognitive development in a comprehensive model.

Although our findings showed that 9 h or more of sleep is beneficial for neurocognitive development, these results by no means indicate the longer sleep duration the better. Both short and long sleep duration could be associated with compromised mental health in adults.[9] Because the current ABCD dataset only provides coarse estimates of sleep duration (appendix p 2), how a prolonged sleep duration (eg, >12 h)[9] would affect neurocognitive development in adolescents has not yet been tested. Given that insufficient sleep has become a pronounced global issue for early adolescents,[1] our current findings highlight the crucial need for early sleep intervention in young people.

This study estimates the long-term effects of insufficient sleep on neurocognitive development in early adolescence while controlling for key covariates, including sex, socioeconomic status, puberty status, and physical health indicators. Our findings suggest that cortico–basal ganglia connections have an important role in mediating the effect of insufficient sleep on cognitive and affective functions, and that structural properties of the anterior temporal lobe might contribute to the effect of insufficient sleep on crystallised intelligence. These effects can last at least 2 years, highlighting the importance of early sleep intervention at young ages to improve long-term neurocognitive development outcomes.

Contributors

FNY conceptualised the study, analysed the data, generated figures, and wrote the original draft of the manuscript. WX contributed to study conceptualisation and data visualisation, and edited the manuscript. ZW contributed to study conceptualisation and design, supervised the study, contributed to data interpretation, and edited the manuscript. FNY and WX accessed and verified the data in the study. All authors had access to all the data in the study. All authors were responsible for the final decision to submit for publication.

Declaration of interests

We declare no competing interests.

Data sharing

The ABCD data used in this study are available in the National Institutes of Mental Health Data Archive. Information about the ABCD consortium is available of their website.

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For the **National Institutes of Mental Health Data Archive** see https://nda.nih.gov/abcd

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