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FULL-LENGTH REPORT

Screen time, sleep, brain structural neurobiology, and sequential associations with child and adolescent psychopathology: Insights from the ABCD study

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ABSTRACT

Background and Aims: The precise roles of screen media activity (SMA) and sleep problems in relation to child/adolescent psychopathology remain ambiguous. We investigated temporal relationships among sleep problems, SMA, and psychopathology and potential involvement of thalamus-prefrontal-cortex (PFC)-brainstem structural covariation. *Methods:* This study utilized data from the Adolescent Brain Cognitive Development study (n = 4,641 ages 9–12) at baseline, Year1, and Year2 follow-up. Cross-Lagged Panel Models (CLPMs) investigated reciprocal predictive relationships between sleep duration/problems, SMA, and psychopathology symptoms. A potential mediating role of baseline Thalamus-PFC-brainstem covariation on SMA-externalizing relationships was examined. *Results:* Participants were divided into discovery (n = 2,359, 1,054 girls) and replication (n = 2,282, 997 girls) sets. CLPMs showed 1) bidirectional associations between sleep duration and SMA in late childhood, with higher frequency SMA predicting shorter sleep duration (β = −0.10 [95%CI: −0.16, −0.03], p = 0.004) and vice versa (β = −0.11 [95%CI: −0.18, −0.05], p < 0.001); 2) externalizing symptoms at age 10–11 predicting sleep problems (β = 0.11 [95%CI: 0.04, 0.19], p = 0.002), SMA (β = 0.07 [95%CI: 0.01, 0.13], p = 0.014), and internalizing symptoms (β = 0.09 [95%CI: 0.05, 0.13], p < 0.001) at age 11–12; and 3) externalizing behavior at age 10-11 partially mediating the relationship between baseline thalamus-PFC-brainstem covariation and SMA at age 11–12 (indirect effect = 0.032 [95%CI: 0.003, 0.067], p-value = 0.030). Findings were replicable. *Conclusion:* We found bi-directional SMA-sleep-duration associations in late childhood. Externalizing symptoms preceded future SMA and sleep disturbances and partially mediated relationships between structural brain covariation and SMA. The findings emphasize the need for understanding individual differences and developing and implementing integrated strategies addressing both sleep concerns and screen time to mitigate potential impacts on psychopathology.

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KEYWORDS

screen media activity, addictive behaviors, Internet addiction, adolescent, insomnia, brain structural covariation

INTRODUCTION

Screen media activity (SMA) has become a dominant recreational pursuit among pre-teens, with electronic media devices prevalent, including in their bedrooms (Hale & Guan, 2015). The pervasive presence of SMA during formative years necessitates an exploration of its relationships to sleep difficulties and mental health (Office of the Surgeon General (OSG), 2023; Paulus et al., 2019, 2023; Zhao, Paulus, & Potenza, 2023). Understanding these relationships is crucial for developing targeted interventions that promote optimal cognitive and emotional development in pre-teen populations.

Greater SMA has been associated with sleep difficulties, with most studies strongly associating screen time with insufficient sleep duration and delayed bedtimes (Cain & Gradisar, 2010; Carter, Rees, Hale, Bhattacharjee, & Paradkar, 2016; Hale & Guan, 2015; Hisler, Twenge, & Krizan, 2020; Kelly, Zilanawala, Booker, & Sacker, 2018; LeBourgeois et al., 2017; Levenson, Shensa, Sidani, Colditz, & Primack, 2016; Parent, Sanders, & Forehand, 2016; Woods & Scott, 2016). Additionally, SMA has been associated with elevated internalizing/externalizing symptoms in youth (Shannon, Bush, Villeneuve, Hellemans, & Guimond, 2022; Twenge & Campbell, 2018), which may be partially attributed to compromised sleep quality (Hale, Li, Hartstein, & LeBourgeois, 2019). However, existing studies have often been limited by cross-sectional designs and modest effect sizes. Findings across studies remain inconclusive, with some showing no association (Odgers & Jensen, 2020) or intricate nonlinear relationships (Orben, Dienlin, & Przybylski, 2019). Moreover, much existing literature has focused on how SMA influences mental health and sleep outcomes, without adequate examination of potential reciprocal relationships. Such unidirectional inquiries fail to capture the complex, interwoven dynamics between these key factors. The lack of longitudinal data leaves unanswered questions regarding the directionality and temporal sequence of relationships, which are crucial for understanding causal pathways and designing effective interventions.

Associations exist between screen time and widespread structural and functional brain features implicated in control and reward processes (Marciano, Camerini, & Morese, 2021; Paulus et al., 2019; Zhao et al., 2022). Frequent screen time during brain development may elevate the risk of accelerated neurodegeneration in adulthood, potentially leading to conditions like amnesia and early-onset dementia (Manwell, Tadros, Ciccarelli, & Eikelboom, 2022). Research has also linked extensive screen time during childhood to brain maturation and lower verbal intelligence (Dubicka, Martin, & Firth, 2019), prompting calls for limiting screen time to support healthy brain development in children (Horowitz-Kraus & Hutton, 2018). While these findings highlight potential influences of SMA on brain structure and function, there remains a gap in the integration of advanced neuroimaging techniques to comprehensively characterize the underlying neural circuits and brain regions implicated in the complex relationships between SMA, sleep, and mental health (Paulus et al., 2023). For example, it is unclear whether there are specific neural pathways or networks that underlie developmental pathways linking SMA to sleep and mental health, or vice versa. This level of mechanistic insight is critical for the development of targeted, biologically based interventions that can more effectively address the nuanced interplay between SMA, sleep difficulties, and psychopathology. This study sought to fill these gaps by utilizing longitudinal Adolescent Brain Cognitive Development (ABCD) study data (Luciana et al., 2018) and employing cross-lagged panel models (CLPMs) (Finkel, 1995) to test bidirectional effects among three factors: SMA, sleep difficulties, and psychopathology measures over time. We hypothesized that sleep difficulties may mediate relationships between SMA and subsequent internalizing and/or externalizing problems. We also explored the involvement in these relationships of a structural covariation pattern implicating a thalamus-prefrontal cortex (PFC)-brainstem circuit (Zhao, Constable, Hien, Chung, & Potenza, 2021) previously linked to SMA (Zhao et al., 2022).

METHODS

Data collection

We analyzed longitudinal data from baseline to Year 2 follow-up (data were collected from September 10, 2018, to December 31, 2019, involving participants aged 9–12 years) of the Adolescent Brain Cognitive Development (ABCD) Study (5.0 release) (Luciana et al., 2018). Detailed description of the ABCD sample recruitment and data collection procedures can be found elsewhere (Luciana et al., 2018). Briefly, data were obtained from 21 research sites across the US, comprising a cohort of over 11,000 children aged 9–10 years old. The ABCD study employed a longitudinal design, collecting extensive behavioral and neuroimaging data through structured interviews and neuroimaging scans. Assessments of sleep problems, SMA, and psychopathology, were conducted annually during each participant visit. Additionally, structural MRI data were and are acquired biennially, commencing at baseline, to provide insights into the evolving brain structure over time. Data are released in waves, and the process of data collection have been detailed in peer-reviewed publications (e.g., Luciano et al., 2018) and are described on the study website: https://abcdstudy.org/.

In this investigation, participants without valid baseline structural MRI data, missing data on sleep disturbance, SMA, psychopathology measures in any of three time points, or missing data on basic demographic variables including age, sex, race/ethnicity, family income, and pubertal development

stage were excluded. The final sample was then divided into discovery (n = 2,359, 1,054 girls) and replication (n = 2,282, 997 girls) sets using the ABCD reproducible matched samples (ARMS) algorithm (Cordova et al., 2020), enabling testing of replicability.

SMA

The SMA measure consisted of youth self-reports of the total number of hours spent on non-school-related SMA on a typical day, calculated as average of a typical weekday and weekend total screen times. SMA mainly included time spent watching TV shows/movies, watching videos, playing video games, texting on electronic devices, visiting social networking sites, and video-chatting.

Sleep difficulties

Sleep duration and difficulties initiating and maintaining sleep (DIMS, reflecting common disorders of initiating and maintaining sleep of childhood) were the two primary sleep difficulty measures investigated. They were derived from the 26-item Sleep Disturbance Scale for Children (SDSC) (Bruni et al., 1996), a parent-report questionnaire assessing children's sleep behaviors over the prior 6 months. The SDSC produces six subscale scores for sleep problems including DIMS. In this study, sleep duration (i.e., sleepdisturb1_p, reverse coded) was defined on a five-point Likert scale with 1 = less than 5 h; 2 = 5–7 h; 3 = 7–8 h; 4 = 8–9 h; and 5 = 9–11 h. The DIMS score was calculated as the sum of all other DIMS items (Table S1 in Supplementary Materials) to isolate other DIMS problems from insufficient sleep duration.

Psychopathology symptoms

The Child Behavior Checklist (CBCL) was used to assess internalizing and externalizing problems (Achenbach, 2009). Internalizing problems refer to inwardly directed concerns such as anxiety, depression, and somatic disturbances, while externalizing problems encompass outwardly directed behaviors like rule-breaking and aggressive conduct. Caregivers completed 118 items evaluating their children's behaviors over the past six months. Each item was scored on a 3-point scale indicating how true the behavior was for their child (0 = not true, 1 = somewhat true, 2 = very true). Ratings were compiled into eight empirically derived syndromes. Following established procedures, internalizing problems scores were calculated by averaging withdrawn/depressed, somatic complaints, anxious/depressed, and social problems subscales, and externalizing problems scores by averaging the rule-breaking and aggressive-behavior subscales.

Structural covariation pattern

Structural MRI data were collected at 9–10 years (i.e., baseline) using 3T scanners; raw data were preprocessed using FreeSurfer-based pipelines (Hagler et al., 2019). Structural brain measurements of surface area, thickness, and gray-matter volume (GMV) were obtained for 68 cortical regions defined by the Desikan-Killiany atlas (Desikan et al., 2006). Subcortical GMV of 16 regions defined by the FreeSurfer aseg parcellation along with the brainstem were also included in analyses. The structural covariation pattern was derived using the Joint and Individual Variance Explain (JIVE) approach (Lock, Hoadley, Marron, & Nobel, 2013; Zhao, Klein, Castellanos, & Milham, 2019).

Statistical analysis

To explore how internalizing and externalizing symptoms relate to sleep difficulties and SMA over time, we employed separate Cross-Lagged Panel Models (CLPMs). These models enabled us to examine the reciprocal predictive relationships between psychopathology domains, sleep difficulties (specifically, DIMS and sleep duration), and SMA. The CLPMs considered autoregressive effects to measure stability over time, cross-lagged effects to evaluate predictive relationships, and concurrent correlations. Structural equation modeling was conducted using the R lavaan package v0.6 to estimate the models. Covariates included race/ethnicity, sex, family income, and both age and physical pubertal development stage as time-varying factors. We used a diagonally weighted least squares estimator and evaluated model fit with the comparative fit index (CFI, >0.95 excellent), root mean square error of approximation (RMSEA, <0.05 excellent), and standardized root mean square residual (SRMR, <0.05 excellent) (Hu & Bentler, 1999). Significance was evaluated using two-sided hypothesis-testing at an alpha of 0.05. All model results had excellent fit statistics and were validated in the replication sample. Sensitivity analyses assessed the robustness of the main findings without adjusting for pubertal development stage, family income, and/or race/ethnicity. Sex-specific analyses were also performed. Finally, mediation analyses explored whether the baseline covariation pattern was related to pathways associated with externalizing problems, based on prior findings (Zhao et al., 2022).

Ethics

IRB approval and informed written consent was obtained from participating ABCD sites during data collection. The current analyses involved deidentified data and were exempted by the Yale IRB, the Yale Human Investigation Committee. Thus, the study is in accordance with the principles of the Declaration of Helsinki.

RESULTS

The discovery and replication sets included 2,359 (1,054 girls [44.7%]) and 2,282 (997 girls [43.7%]) adolescents, respectively. Groups did not differ on age, sex, race ethnicity, baseline family income, pubertal development stage or primary outcome measures (Table 1). A portion (n = 2,759, 26.4%) of participants who had missing psychopathology measures at 11–12 years were more likely from families with lower incomes and parental educational backgrounds, to be

Table 1. Sample characteristics between participants in discovery and validation sets

	Discovery (N = 2,359)	Validation (N = 2,282)	p value
Baseline age (yrs) [mean (SD)]	10.0 (0.616)	10.0 (0.614)	0.186
Sex (Female)	1,054 (44.7%)	997 (43.7%)	0.516
Race ethnicity			
White	1,433 (60.7%)	1,406 (61.6%)	0.089
Black	239 (10.1%)	220 (9.6%)	
Hispanic	419 (17.8%)	361 (15.8%)	
Asian	52 (2.2%)	42 (1.8%)	
Other	216 (9.2%)	253 (11.1%)	
Baseline family income			
[<$50,000]	555 (23.5%)	541 (23.7%)	0.651
[$50,000–$100,000]	702 (29.8%)	642 (28.1%)	
[$100,000–$200,000]	808 (34.3%)	801 (35.1%)	
[≥$200,000]	294 (12.5%)	298 (13.1%)	
Prepuberty Stage at baseline (Yes)	715 (30.3%)	703 (30.8%)	0.737
Internalizing T Score [mean (SD)]			
Baseline (9–10 yrs)	48.2 (10.1)	48.4 (10.5)	0.478
Year1 (10–11 yrs)	48.3 (10.4)	48.4 (10.4)	0.584
Year2 (11–12 yrs)	47.7 (10.3)	47.8 (10.4)	0.754
Externalizing T Score [mean (SD)]			
Baseline (9–10 yrs)	45.0 (9.91)	45.2 (10.1)	0.637
Year1 (10–11 yrs)	44.4 (9.80)	44.7 (9.83)	0.234
Year2 (11–12 yrs)	44.2 (9.62)	44.1 (9.52)	0.943
Total Screen Time in a typical day [mean (SD)]			
Baseline (9–10 yrs)	3.42 (2.73)	3.55 (2.76)	0.115
Year1 (10–11 yrs)	4.26 (3.26)	4.28 (3.38)	0.534
Year2 (11–12 yrs)	3.73 (2.95)	3.76 (3.04)	0.353
Sleep problems - DIMS score [mean (SD)]			
Baseline (9–10 yrs)	9.89 (3.26)	10.0 (3.38)	0.183
Year1 (10–11 yrs)	9.94 (3.32)	10.1 (3.46)	0.063
Year2 (11–12 yrs)	10.0 (3.42)	10.0 (3.30)	0.931

DIMS: Difficulties Initiating and Maintaining Sleep.

slightly younger, and of Black race and Hispanic ethnicity. These respondents spent more time on SMA across all three time points and had higher externalizing scores at 10–11 years (Table S2 Supplementary Materials).

Temporal consistency in sleep difficulties, SMA, and psychopathology symptoms

Across all CLPMs examined, we observed a considerable degree of temporal consistency in sleep patterns, SMA, and psychopathology symptoms over time. Standardized path coefficients (β) ranging from 0.56 to 0.74 for sleep duration, 0.67 to 0.68 for DIMS, 0.44 to 0.74 for SMA, 0.68 to 0.76 for internalizing symptoms, and 0.74 to 0.79 for externalizing symptoms (all $p < 0.001$; Figs 1–3). These findings suggest relatively persistent within-individual levels of these variables in the study cohort.

Reciprocal relationships between SMA and sleep duration

Considering externalizing symptoms, SMA, and sleep duration in the models, a reciprocal predictive relationship between sleep duration and SMA emerged between 9-10 years and 10–11 years (Fig. 1A). Specifically, a shorter sleep duration at 9–10 years significantly predicted higher SMA at 10–11 years (discovery: $\beta = -0.11$ [95%CI: −0.18, −0.04], $p = 0.001$; validation: $\beta = -0.09$ [95%CI: −0.16, −0.03], $p = 0.005$). Conversely, higher SMA at 9–10 years predicted shorter sleep duration at age 10–11 years (discovery: $\beta = -0.10$ [95%CI: −0.16, −0.03], $p = 0.004$; validation: $\beta = -0.09$ [95%CI: −0.16, −0.03], $p = 0.006$). However, this effect was smaller at ages 10–11 and 11–12. These findings remained consistent when the models included internalizing symptoms (Fig. 1B), suggesting that sleep duration and SMA influence each other bi-directionally and equally in late childhood, but that such effects decrease in emerging/early adolescence.

Externalizing symptoms at 10–11 years predicted higher future DIMS, SMA, and internalizing symptoms

Greater externalizing symptoms at 10–11 years predicted higher DIMS (discovery: $\beta = 0.11$ [95%CI: 0.04, 0.19], $p = 0.002$; validation: $\beta = 0.08$ [95%CI: 0.02, 0.14], $p = 0.013$) and more SMA (discovery: $\beta = 0.07$ [95%CI: 0.01, 0.13], $p = 0.014$; validation: $\beta = 0.06$ [95%CI: 0.01, 0.12], $p = 0.028$) at 11–12 years (Fig. 2A). When internalizing and externalizing symptoms were examined simultaneously in the CLPMs, both SMA (discovery: $\beta = 0.05$ [95%CI: 0.02, 0.09], $p = 0.003$; validation: $\beta = 0.04$ [95%CI: 0.01, 0.08], $p = 0.015$) and sleep duration (discovery: $\beta = -0.11$ [95% CI: −0.15, −0.07], $p < 0.001$; validation: $\beta = -0.09$ [95%CI: −0.13, −0.05], $p < 0.001$) at 9–10 years predicted externalizing problems at 10–11 years (Fig. 3). Externalizing symptoms at 10–11 years robustly predicted internalizing symptoms one year later (e.g., Fig. 3B: discovery: $\beta = 0.09$ [95%CI: 0.05, 0.13], $p < 0.001$; validation: $\beta = 0.09$ [95%CI: 0.05, 0.13], $p < 0.001$).

Internalizing symptoms predicted future DIMS

Internalizing symptoms predicted future DIMS, but the reciprocal effect was less robust (Fig. 2B). Higher internalizing symptoms at 9–10 years predicted greater DIMS one year later (discovery: $\beta = 0.09$ [95%CI: 0.01, 0.17], $p = 0.021$; validation: $\beta = 0.10$ [95%CI: 0.02, 0.18], $p = 0.011$). A similar significant effect was observed at 10–11 years (discovery: $\beta = 0.11$ [95%CI: 0.03, 0.18], $p = 0.005$; validation: $\beta = 0.09$ [95%CI: 0.02, 0.18], $p = 0.011$). However, the impact of DIMS on subsequent internalizing behavior was less robust. DIMS at 9–10 years predicted internalizing behavior at 10–11 years ($\beta = 0.09$ [95%CI: 0.02, 0.16], $p = 0.018$) in the discovery but not validation dataset ($\beta = 0.06$ [95%CI: −0.01, 0.13], $p = 0.11$). In addition, DIMS at 10–11 years only marginally predicted internalizing behavior at 11–12 years in both datasets (discovery: $\beta = 0.07$ [95%CI: −0.003, 0.13], $p = 0.062$; validation: $\beta = 0.06$ [95% CI: −0.01, 0.13], $p = 0.075$). However, when internalizing and externalizing symptoms were examined simultaneously in the CLPM (Fig. 3A), DIMS robustly predicted internalizing behaviors at subsequent years.

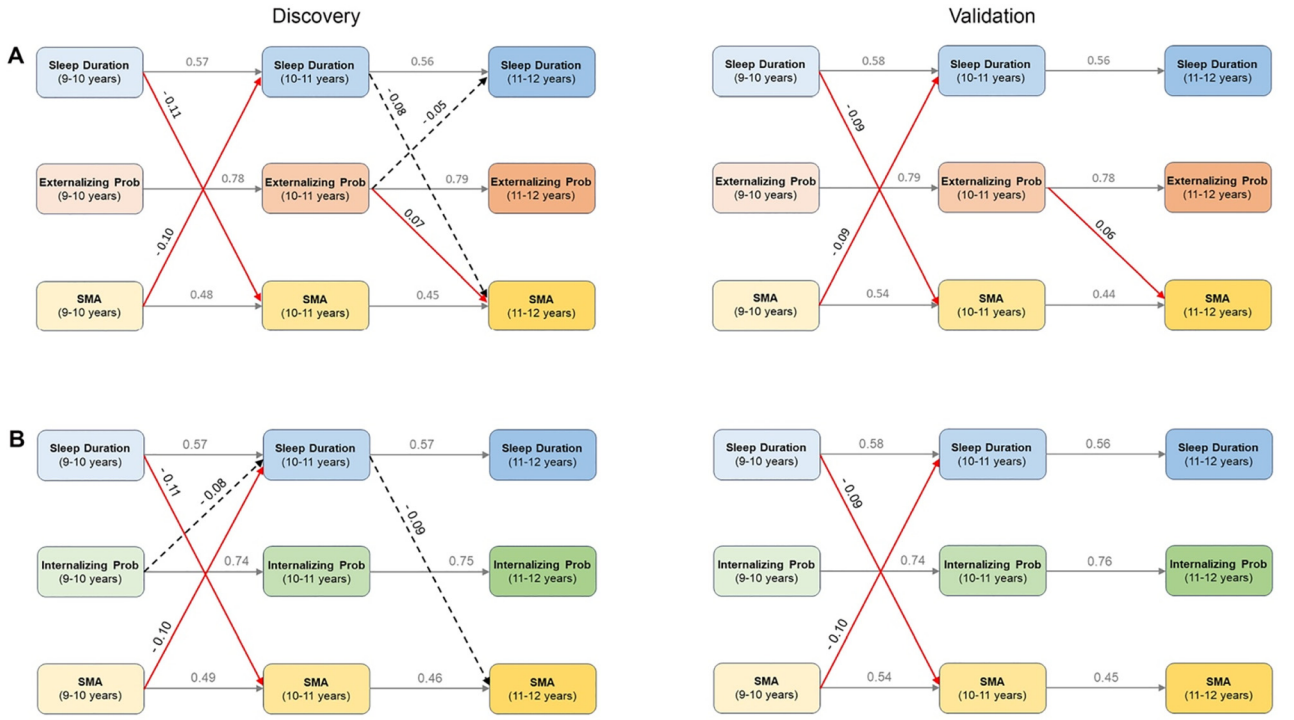

Fig. 1. CLPMs examining connections between sleep duration, SMA, and externalizing (**A**) or internalizing (**B**) problems. Standardized coefficients are presented as numbers. Red solid lines represent significant paths in both discovery and validation datasets, and black dotted lines indicating significant in either discovery or validation datasets. SMA: screen media activity; DIMS: Difficulties Initiating and Maintaining Sleep; CLPM: Cross-lagged Panel Model

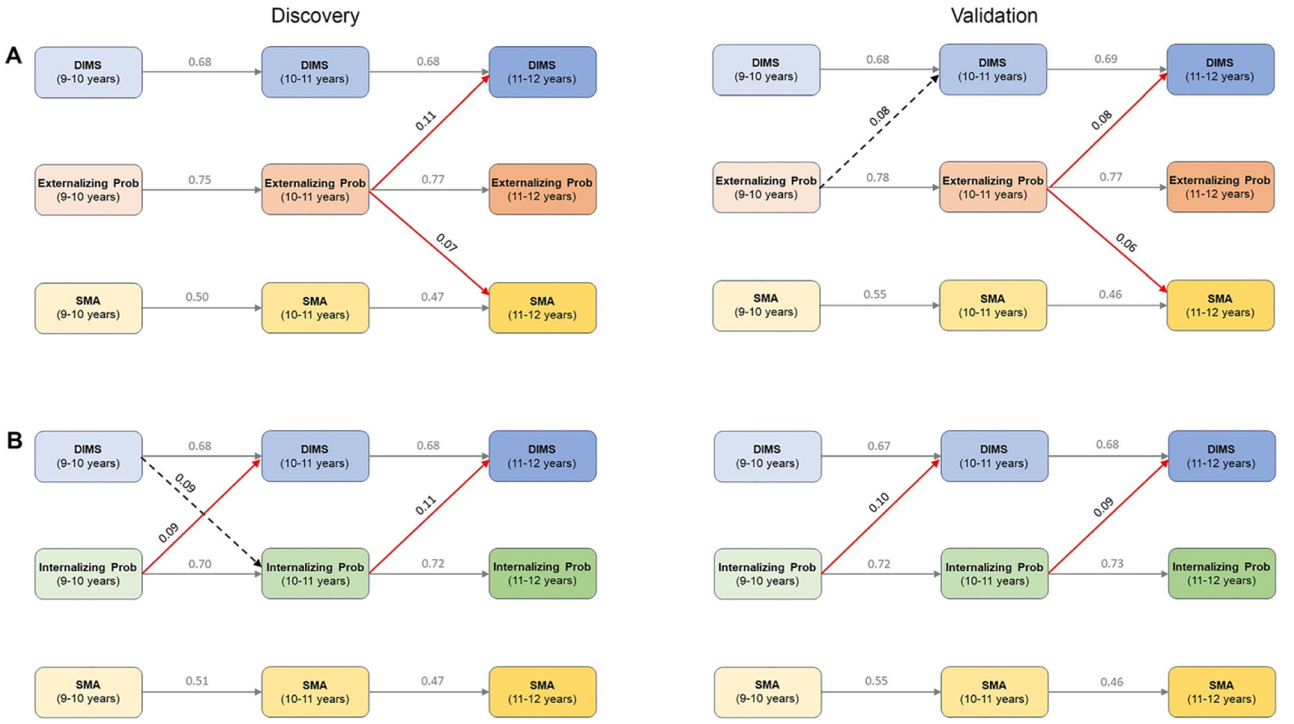

Fig. 2. CLPMs examining connections between DIMS, SMA, and externalizing (**A**) or internalizing (**B**) problems. Standardized coefficients are presented as numbers. Red solid lines represent significant paths in both discovery and validation datasets, and black dotted lines indicating significant in either discovery or validation datasets. SMA: screen media activity; DIMS: Difficulties Initiating and Maintaining Sleep; CLPM: Cross-lagged Panel Model

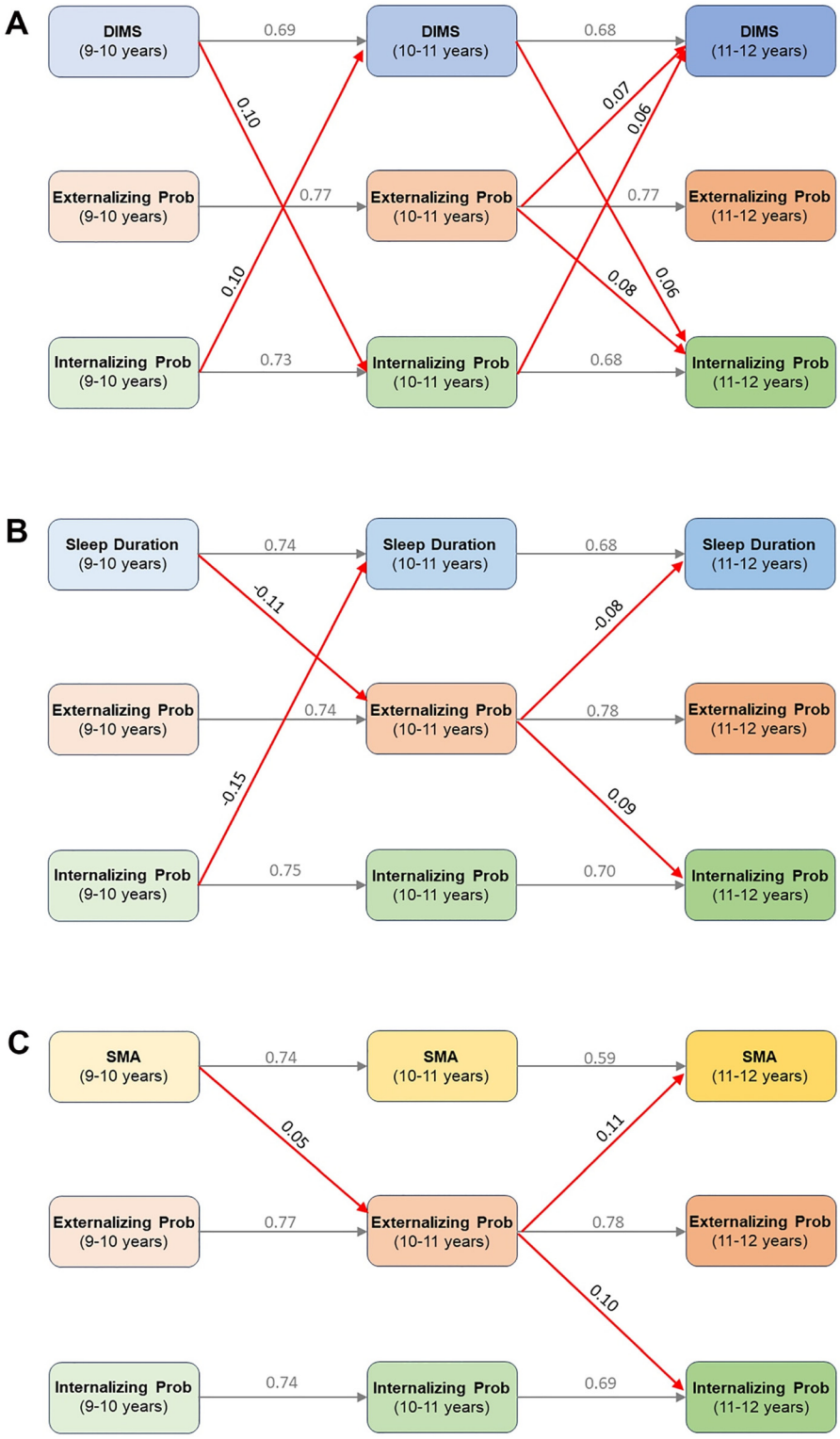

Fig. 3. Concurrent CLPMs evaluating sleep DIMS score (**A**), sleep duration (**B**), or SMA (**C**) along with externalizing and internalizing problems. Standardized coefficients are represented by the numbers, estimated using the discovery data. The figure displays paths that remained significant in both discovery and validation datasets. SMA: screen media activity; DIMS: Difficulties Initiating and Maintaining Sleep; CLPM: Cross-lagged Panel Model

Externalizing behavior partially mediated the effect of structural covariation pattern on SMA

JIVE analyses identified a structural covariation pattern highlighting the covariation among GMV and surface area in regions involved in our hypothesized Thalamus-PFC-Brainstem circuit. Regions with the largest loading magnitudes are depicted in eFig. 1A. This covariation pattern was highly similar in boys and girls and to previous reports (Zhao et al., 2021, 2022), as evidenced by the component loadings correlation of at least 0.98.

Controlling for the covariates and the baseline outcome measure, we found higher structural covariation component scores (indicating smaller GMV and surface area in the regions highlighted in eFig. 1A) at 9–10 years were associated with higher externalizing scores at 10–11 years (discovery: $\beta = 1.516$, SE = 0.706, $p = 0.032$; validation: $\beta = 2.01$, SE = 0.708, $p = 0.005$), which in turn led to higher total screentime at 11–12 years (discovery: $\beta = 0.0.030$, SE = 0.006, $p < 0.001$; validation: $\beta = 0.0.022$, SE = 0.005, $p < 0.001$). Despite a significant mediating role of externalizing behavior (discovery: indirect effect = 0.032, [95%CI: 0.003, 0.067], p-value = 0.030; validation: indirect effect = 0.030 [95%CI: 0.007, 0.061], p-value = 0.004; eFig. 1B), we note that there might be multiple pathways (both positive and negative) explaining effects of the baseline structural covariation pattern on SMA at 11–12 years, as evidenced by the inconsistent mediation model (i.e., positive indirect effect but negative direct effect) (MacKinnon, Fairchild, & Fritz, 2007) in the validation set.

Sensitivity analysis. Results from various sensitivity analyses are summarized in Table S3 in Supplementary material. Findings from the main analyses were robustly replicated in the planned sensitivity analyses.

DISCUSSION

The primary goal of our study was to examine relationships among sleep problems, SMA, and psychopathology symptoms in adolescents and yielded five main results. First, there was considerable temporal consistency in these variables over time. Second, a reciprocal relationship was found between sleep duration and SMA in late childhood, but this effect diminished in emerging/early adolescence. Third, externalizing symptoms at ages 10–11 were predictive of higher DIMS, SMA, and internalizing symptoms at ages 11–12. Fourth, internalizing symptoms also predicted future DIMS, although the reciprocal effect was less robust. Fifth, structural brain variations at ages 9–10 were associated with higher externalizing scores at ages 10–11, which partially mediated the relationship with total screen time at ages 11–12. Taken together, these findings suggest that sleep difficulties, screen time, and psychopathology are interrelated and relatively stable over time, and that externalizing symptoms serve as a significant predictor for multiple outcomes.

Bidirectional relationships between SMA and sleep duration in late childhood

Our study demonstrated a reciprocal SMA-sleep duration relationship from ages 9–10 to 10–11 years. The negative impacts of SMA on sleep duration may be partially attributed to exposure to nighttime blue-green light, which diminishes melatonin levels in the body (Hersh, Sisti, Richiutti, & Schernhammer, 2015) and stimulates alertness in the brain (Chaput et al., 2023). Additionally, excessive SMA may correlate with reduced outdoor activities, potentially affecting both sleep quality and circadian rhythms (Wams et al., 2017). Moreover, the content and nature of screen-based activities can be mentally engaging and stimulating, promoting wakefulness (Chaput et al., 2023; Hale et al., 2018). Sleep deprivation could impair function of the PFC (Goldstein & Walker, 2014; Muzur, Pace-Schott, & Hobson, 2002), a region crucial for decision-making and self-control, making youth more susceptible to self-regulation issues. Some youth may turn to screens as a coping mechanism (Kardefelt-Winther, 2014) to manage negative mood states such as anger, irritability, or anxiety induced by insufficient sleep (Shanahan, Copeland, Angold, Bondy, & Costello, 2014; Sivertsen et al., 2015). Furthermore, youth who sleep less may resort to SMA simply because there is less else to do (Paulus et al., 2023).

Interestingly, data suggested these bidirectional effects attenuated by ages 11–12 years. Although studies have reported persistent SMA-sleep duration links that continue beyond ages 12 years (Parent et al., 2016; Richardson et al., 2021), these studies did not consider potential confounding effect due to pubertal development. We speculate that the attenuation may mirror typical developmental shifts in sleep patterns (e.g., shorter sleep duration) during puberty (Colrain & Baker, 2011). Additionally, heightened academic demands, changes in circadian rhythm, and greater social media use (Song et al., 2023) may contribute to the observed weakening of the relationship. Overall, our findings highlight late childhood as a critical period when SMA and sleep duration mutually impact one another. These findings underscore the intricate interplay between technological engagement and sleep, highlighting the need for targeted interventions that address both behavioral and neurobiological mechanisms to improve sleep and self-regulation in adolescents.

Externalizing problems at 10–11 years

Externalizing problems typically precede internalizing problems in adolescents, and externalizing behaviors often lead to subsequent sleep disturbance (Flouri et al., 2019; Quach, Nguyen, Williams, & Sciberras, 2018). Our results are consistent with these findings, suggesting that externalizing problems emerging around ages 10-11 play a crucial role in linking earlier sleep and SMA to subsequent internalizing and sleep difficulties. Insufficient sleep and excessive SMA may independently and jointly influence future externalizing behaviors. For example, insufficient sleep may impair PFC function and emotional/behavioral regulation (Goldstein & Walker, 2014; Muzur et al., 2002;

Vandekerckhove & Cluydts, 2010), leading to increased externalizing problems including aggression, impulsivity, and poor decision-making (Becker et al., 2019). Additionally, excessive SMA, especially with inappropriate content, may reinforce externalizing behaviors (Twenge & Campbell, 2018). Both excessive SMA and sleep deficiency have been associated with poorer academic achievement (Adelantado-Renau et al., 2019) and reduced parental monitoring of behavior (Gunn et al., 2019), which may further exacerbate externalizing problems. Although our study provides insights into these intricate relationships among sleep problems, SMA, and behavioral symptoms in children, further research is warranted to comprehensively understand the interconnected underlying mechanisms and developmental pathways involved in these associations.

As an initial investigation, we performed a mediation analysis to examine the potential mediating role of externalizing problems in the relationship between the structural covariation pattern at ages 9–10 and SMA at ages 11–12. Our decision to focus on the baseline structural covariation pattern was informed by our previous work using the ABCD baseline data, which demonstrated significant associations between SMA and the covariation pattern involving thalamic, prefrontal cortical and brainstem regions (Zhao et al., 2022), as well as between SMA and externalizing behavior. Extensive prior research has revealed common neural substrates, including PFC, that are linked to externalizing psychopathology (Koob & Volkow, 2016). Additionally, the covariation pattern involving the thalamus-PFC-brainstem network has been consistently observed across multiple samples and age groups in our prior research (Zhao et al., 2021). Therefore, investigating the potential role of this neural feature in the subsequent externalizing-SMA relationship was empirically grounded. Our mediation analysis showed a significant indirect effect in both training and validation sets (eFig. 1), indicating that alterations in thalamus-PFC-brainstem covariation pattern at 9–10 years might be linked to higher externalizing problems at 10–11 years, which could potentially contribute to increased SMA at 11–12 years. However, it is important to note that although a significant indirect effect was replicated, it accounted for about 9% of the mediated effect and the significant direct effect linking baseline structural covariation pattern to SMA at 11–12 years was only observed in the training data. This suggests that there may be multiple causal pathways linking the baseline covariation pattern to subsequent SMA.

Clinical implications

The study's findings have significant clinical implications for pediatricians managing adolescent health. The observed temporal consistency in sleep patterns, SMA, and psychopathology symptoms underscores the importance of early identification and intervention, as these features appear to be relatively stable over time. The reciprocal relationship between sleep duration and SMA suggests that targeted interventions in one area could yield benefits in the other, offering a multi-faceted approach to treatment. For example, strategies that improve children's self-regulatory capacities (Williams, Berthelsen, Walker, & Nicholson, 2017) and promote healthy sleep habits and mindful screen use may mutually reinforce each other. Moreover, the predictive value of externalizing symptoms for future sleep difficulties and internalizing symptoms highlights the need for comprehensive screening and monitoring, especially around the ages of 10–11. Given that the study also noted socioeconomic and ethnic disparities in missing data, pediatricians should exercise caution in generalizing these findings across diverse populations. Overall, these insights can guide more effective, evidence-based care strategies for adolescents, particularly in the realms of sleep hygiene, screen time management, and mental health.

Strengths and limitations

This study has notable strengths, including the use of a large, population-based sample, the complex CLPM approach, the use of discovery and replication samples, and the rigorous sensitivity analyses to ensure that findings are not contingent on analytical approaches (Orben et al., 2019). We used CLPM. Several alternative models, including the Random-Intercept CLPM (see review by (Mund & Nestler, 2019)), have been proposed to examine reciprocal relations in longitudinal settings. These alternative models may provide additional insights into within-person processes and individual differences in the relationships among SMA, sleep, and psychological symptoms. Future research could employ these alternative approaches, particularly when the focus is on examining how these relationships vary across individuals or subgroups, or when the assumptions of time-invariant individual differences are met. Given that internalizing and externalizing symptoms often co-occur, future research could employ alternative modeling approaches to examine potential interactions between these symptom domains in relation to SMA and sleep problems (Ozkok et al., 2022; Speyer, Ushakova, Blakemore, Murray, & Kievit, 2023). Also, it is unfeasible to examine connections among SMA, sleep, and internalizing/externalizing symptoms in one CLPM due to potential estimation problems. The study is also limited by the use of only parent-reported data on sleep difficulties and psychopathology symptoms and self-reported SMA. It will be useful to validate the findings using objective measures of SMA and sleep difficulties. In addition, when comparing participants included in the analysis with those excluded due to missing psychopathology symptoms at ages 11–12, a higher proportion of exclusions originated from families belonging to more disadvantaged groups, and they had consistently allocated more screen time across all assessed time points. Furthermore, the primary SMA outcome, total screentime, is a global and non-specific measure. We acknowledge the importance of assessing nuanced aspects of screen media usage. Factors such as parenting rules on SMA, SMA engagement patterns, the use of self-report versus objective measures of SMA and distinguishing problematic from general SMA could potentially influence the

observed associations. We contend that total daily screen time remains an important and clinically useful metric warranting continued research attention. Several studies reveal consistent associations between higher total screen exposure and adverse sleep outcomes in youth (Hale et al., 2019; Hale & Guan, 2015). Links between increased total usage and mental health issues also exist (Houghton et al., 2018; Zhao et al., 2022). Therefore, quantifying total daily consumption can still provide a meaningful indicator of risk, even without parsing the exact timing, content, and contexts of technology habits. In particular, excessive cumulative screen engagement may displace vital activities for healthy development like sleep, exercise, and face-to-face interactions (Tremblay et al., 2011). From a public health perspective, establishing thresholds for harmful total daily use can inform sensible media consumption guidelines for families and clinicians. Of course, like any complex behavior, more multidimensional profiling of usage patterns should complement this research. For a comprehensive understanding, these areas need further investigation, for example, using the Bronfenbrenner's ecological systems approach applied to SMA (Paulus et al., 2023). Finally, it is important to note that the relationships observed in this study reflect pre-COVID-19-pandemic patterns, as our analysis was based on data collected before December 31, 2019. Given the significant disruptions related to the pandemic, including school closures, increased social isolation, and elevated stress levels, it is possible that the dynamics among SMA, psychopathology, and sleep problems may have shifted in during and after the pandemic.

CONCLUSION

This investigation enhances our understanding of the intricate relationships between SMA, sleep difficulties, and psychopathological symptoms in youth. We found a reciprocal SMA-sleep duration relationship, particularly in late childhood. Externalizing symptoms emerged as pivotal, linking earlier SMA and sleep problems to future internalizing issues. These insights have important clinical implications and call for multidisciplinary approaches to comprehend causal factors affecting youth sleep and psychopathology. Despite limitations like reliance on parent-reported data and potential sampling bias, our study provides a robust framework that underscores the urgency for targeted interventions focused on sleep duration, SMA, and externalizing symptoms, which could potentially yield cascading benefits for youth mental health.

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Conflict of interest: The authors declare no conflicts of interest. Dr. Potenza has consulted for Opiant Therapeutics, Game Day Data, Baria-Tek, and Boehringer Ingelheim,; has been involved in a patent application with Yale University and Novartis; has received research support from Mohegan Sun Casino, Children and Screens and the Connecticut Council on Problem Gambling; has participated in surveys, mailings or telephone consultations related to drug addiction, impulse-control disorders or other health topics; has consulted for and/or advised gambling, non-profit and legal entities on issues related to impulse control, internet use, and addictive disorders; has provided clinical care in a problem gambling services program; has performed grant reviews for research-funding agencies; has edited journals and journal sections; has given academic lectures in grand rounds, CME events and other clinical or scientific venues; and has generated books or book chapters for publishers of mental health texts. Dr Potenza is an Associate Editor to the Journal of Behavioral Addictions. The other authors do not report disclosures.

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SUPPLEMENTARY MATERIAL

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