

PREPUBLICATION RELEASE

Digital Media, Genetics and Risk for ADHD Symptoms in Children – A Longitudinal Study

Samson Nivins, PhD; Michael A. Mooney, PhD; Joel Nigg, PhD; Torkel Klingberg, PhD

DOI: 10.1542/pedsos.2025-000922

Journal: *Pediatrics Open Science*

Article Type: Original Research Article

Citation: Nivins S, Mooney MA, Nigg J, Klingberg T. Digital Media, Genetics and Risk for ADHD Symptoms in Children – A Longitudinal Study. *Pediatr Open Sci.* 2025; doi: 10.1542/pedsos.2025-000922

This is a prepublication version of an article that has undergone peer review and been accepted for publication but is not the final version of record. This paper may be cited using the DOI and date of access. This paper may contain information that has errors in facts, figures, and statements, and will be corrected in the final published version. The journal is providing an early version of this article to expedite access to this information. The American Academy of Pediatrics, the editors, and authors are not responsible for inaccurate information and data described in this version.



Copyright © 2025. Nivins S et al. This is an open access article distributed under the terms of the CC-BY-NC-ND license, which permits noncommercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN®



Digital Media, Genetics and Risk for ADHD Symptoms in Children – A Longitudinal Study

Samson Nivins PhD¹, Michael A. Mooney PhD², Joel Nigg PhD³,
Torkel Klingberg PhD¹

Affiliations: ¹Department of Neuroscience, Karolinska Institute, Stockholm, Sweden; ²Division of Bioinformatics and Computational Biology, Oregon Health & Science University, Portland, Oregon, United States; ³Division of Clinical Psychology, Oregon Health & Science University, Portland, Oregon, United States

Address correspondence to Torkel Klingberg, Department of Neuroscience, Karolinska Institutet, Stockholm 17165, Sweden [torkel.klingberg@ki.se]

Conflict of Interest Disclosures: The authors have no conflicts of interest relevant to this article to disclose

Role of Funder/Sponsor: This study was supported by the Swedish Research Council to Torkel Klingberg; and Stiftelsen Frimurare Barnhuset, post-doc grant to Samson Nivins. None of those listed here had any part in data handling, data analysis, or result interpretation.

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; DM, digital media; polygenic risk score for ADHD, PGS-ADHD; SES, socioeconomic status; OR, odds ratio; ABCD, Adolescent Brain Cognitive Development; CBCL, Child Behaviour Checklist; DSM, Diagnostic and Statistical Manual of Mental Disorders.

Article summary

Explores associations between types of digital media use and ADHD symptoms in children, emphasising distinct effects and implications for understanding and managing ADHD risk factors.

What's Known on This Subject

ADHD is a common, highly heritable neurodevelopmental disorder. Its rising prevalence suggests environmental influences. Digital media use has been associated with ADHD symptoms, though findings remain mixed. Most research is cross-sectional, and lacks focus on specific media or genetic predisposition.

What This Study Adds

Social media use, unlike other digital media activities, was associated with increased risk of inattention symptoms. Given its widespread use among children, the findings underscore the need for stricter age regulations and consideration of platform design to support healthy development.

Contributors Statement Page

Dr Samson Nivins had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Dr Samson Nivins conceptualized, designed the study, carried out the formal analysis, drafted the initial manuscript, and critically reviewed and revised the manuscript

Prof Torkel Klingberg conceptualized, designed the study, drafted the manuscript, critically reviewed and revised the manuscript

Dr Michael Mooney, Prof Joel Nigg computed the polygenic scores for ADHD, critically reviewed and revised the manuscript

Abstract**Background**

Children spend significant amount of time using digital media (DM), and longer exposure may increase attention-deficit/hyperactivity disorder (ADHD)-related symptoms, although findings are mixed. We investigated longitudinal association between different types of DM use and ADHD-related symptoms in school-aged children, accounting for genetic predisposition and socioeconomic status.

Methods

This study included children from the Adolescent Brain and Cognitive Development Study, followed annually for four years. Estimated time spent on social media, video games, and television/videos was self-reported using Youth Screen Time Survey. ADHD-related symptoms were assessed at each visit with the parent-reported Child Behaviour Checklist. Genetic predisposition was estimated using a polygenic risk score for ADHD (PGS-ADHD).

Results

The study included 8324 children (53% boys; mean age: 9.9 years). On average, children spent 2.3 hours/day watching television/videos, 1.4 hours/day on social media, and 1.5 hours/day playing video games. Average social media use was associated with increased inattention symptoms over time (β [SE], 0.03 [0.01]; $P < 0.001$), with a cumulative four-year effect of $\beta = 0.15$ [SE] = 0.03; $P < 0.001$). No associations were found between playing video games or watching television/videos and ADHD-related symptoms. The association between social media use and inattention symptoms was not moderated by sex, ADHD diagnosis, PGS-ADHD, or ADHD medication status. Inattention symptoms were not associated with increased social media use over time.

Conclusion

Social media use was associated with an increase in inattention symptoms in children over time. Although the observed effect size was small, it could have significant consequences if behavior changes occur at the population level.

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a childhood-onset neurodevelopmental disorder with a heritability estimated between 60 and 80%.¹⁻³ Despite this high heritability, the prevalence of ADHD diagnoses in children has risen significantly. According to U.S. National Survey of Children's Health, parent-reported ADHD prevalence increased from 9.5% in 2003-2007 to 11.3% in 2020-2022.^{4,5} This rise may reflect past underdiagnosis, greater public awareness, current overdiagnosis, or impact of environmental factors. Multiple environmental factors including maternal stress, lead exposure, and perinatal factors can contribute to symptomatology.⁶ Further, there could also be an interaction between genetic predisposition and environmental exposures.^{7,8}

An environmental factor that lately has drawn increasing interest is the potential negative impact of using digital media (DM), including social media (e.g., Facebook or Instagram), playing video games, or watching television/videos. A meta-analysis of longitudinal and cross-sectional studies from 1987 to 2011 found a small but statistically significant association between DM use and ADHD-related symptoms (effect size, $b=0.12$).⁹ However, cross-sectional studies are subjected to self-selection bias, meaning that ADHD symptoms may lead to increased DM use,¹⁰ rather than the other way around. Among longitudinal studies, digital multi-tasking was associated with increased inattention symptoms in younger adolescents ($b=0.16$), but not in older.¹¹ Another study also found that DM use in adolescents predicted later ADHD symptoms (odds ratio, $OR=1.11$).¹² Watching television and playing video games showed a weak association with hyperactivity symptoms two years later in 10-year-olds ($b=0.04$), but not in six-year-olds.¹³ Conversely, a study in 10-year-olds found no significant association between DM use and inattention symptoms 6-12 months later when correcting for baseline symptomatology.¹⁴

In summary, there are indications of associations between different types of DM use and ADHD symptoms, but the literature is inconsistent.¹⁵ Differences in study outcomes could be due to differences in the definition of “Screen Time”, different lengths of follow-up, and differences among study populations. None of these studies accounted for genetic predisposition, which could explain differences in outcomes if populations differ systematically. To our knowledge, one study found that children with a high-genetic predisposition for ADHD measured using polygenic risk scores (PGS-ADHD) were more likely to spend increased time using DM.¹⁶ It is theoretically possible that children with higher PGS-ADHD scores may be more sensitive to DM exposure, which could trigger or exacerbate ADHD symptoms.

The present study aimed to examine longitudinal associations between different types of DM use and ADHD-related symptoms in school-aged children, incorporating genetic predisposition to ADHD through PGS-ADHD. We hypothesized that, if DM use influences ADHD symptoms, children with above-average users exhibit a greater increase in ADHD symptoms over time.

Methods

Study Design and Participants

The behavioural data were obtained from the Adolescent Brain and Cognitive Development (ABCD) Study (<https://abcdstudy.org/>; data release 5.0),¹⁷ a longitudinal cohort comprising 11875 children born between 2005 and 2009. These children were enrolled at ages 9-10 years from 21 data collection sites across the U.S. between 2016 and 2018.¹⁸ Children were excluded: if they were born extremely preterm (<28 weeks'), or had birthweight (<1200 g), were not proficient in English, had any neurological problems, had a history of seizures, or had a contraindication to undergo brain MRI scans. We included only one child per family at random to mitigate familial-related effects in the analysis, leaving 8324 children for the baseline (T₀) analysis. All children and their parents/guardians provided informed written assent/consent for participation, and the central Institutional Review Board at the University of California, San Diego approved the study protocols.¹⁹ This study followed the Strengthening the Reporting of Observational Studies in Epidemiology ([STROBE](#)) reporting guideline.

Children were enrolled at ages 9-10 years (baseline, T₀) and followed annually for up to four years (T₁ - T₄), with follow-up rates exceeding 85% through the third year and 43% at year four. Detailed information on ADHD-related symptoms and DM use survey is provided in (**eMethods in Supplement**). Brief descriptions are provided below.

ADHD-Related Symptoms

ADHD-related symptoms were assessed at all study visits using the parent-reported Child Behaviour Checklist (CBCL), which consist of 118 items rated on a 3-point Likert scale (0=not true, 1=somewhat or sometimes true, and 2=very true or often true). We used combined ADHD-related symptoms as continuous variables based on the Diagnostic and Statistical Manual of Mental Disorders (DSM)-5-defined ADHD CBCL subscale constructed by experts

(i.e., `cbcl_scr_dsm5_adhd`), with higher scores indicating greater ADHD symptomology. Coefficient alpha was satisfactory at all time points with α 's > 0.72.

To examine specific-symptom domains, ADHD symptoms were further categorized into 'inattention' and 'hyperactivity-impulsivity', based on DSM-5 criteria, using CBCL items only. The nine CBCL items (questions) corresponding to each domain were analyzed in a factor analysis.²⁰ Items with a factor loading larger than 0.3 were retained,²¹ resulting in four inattention items, and five hyperactivity/impulsivity items (**eMethods in Supplement**). Although CBCL includes a broader set of questions labeled "inattention," our factor-derived inattention score was highly correlated with the CBCL summary inattention score at T₀ ($r=0.93$). Higher scores on specific-symptom domain scores indicate greater severity of inattention or hyperactivity-impulsivity symptoms.

Exposure

Digital Media Use

The estimated time spent on social media use, playing video games, or watching television/videos was assessed using the Youth Screen Time Survey across multiple visits: T₀, T₁, T₂, T₃, and T₄. Children provided reports on the number of hours they spent on weekdays and weekends excluding school-related work on the following: (1) watching television or movies, (2) watching videos (e.g., YouTube), (3) playing video games on a computer, console, phone, or another device (e.g., Xbox), (4) Texting on a cell phone, tablet, or computer (e.g., Google Chat, WhatsApp), (5) Visiting social networking sites (e.g., Facebook, Instagram), and (6) Using video chat (e.g., Skype, FaceTime). DM use was categorized based on our previous publications,^{22,23} as follows: (a) social media use (4+5+6), (b), playing video games (3), or (c) watching television/videos (1+2). The average daily hours spent on individual DM use were calculated as $[(\text{total weekday hours} * 5) + (\text{total weekend hours} * 2)]/7$.

The Youth Screen Time Survey asks children to report hours spent on each activity for a ‘typical’ weekday and a ‘typical’ weekend day, rather than for a specific prior day, week, or month.²⁴ This approach is intended to capture habitual patterns of media use, reflecting each child’s average daily behaviour on usual days rather than relying on short-term recall over a fixed reference period.

We relied on self-reported surveys by children,^{25,26} considering that parents might not have full awareness, especially among children aged 9-10 and older who frequently use DM unsupervised.

Due to COVID-19 lockdown, children might likely spend more time using DM than anticipated at T₀. A U.S.-based study reported a two-fold increase in DM use during lockdown.²⁷ To account for this change, we used the average estimated time spent on individual DM category (i.e., social media use, playing video games, and watching television/videos) across visits for longitudinal analyses, rather than relying solely on T₀ or T₄. Each category was analyzed separately in longitudinal models.

Genotyping

Saliva samples were collected at T₀ visit, and genotyping was performed at Rutgers University Cell and DNA Repository using Affymetrix NIDA SmokeScreen array, as previously described.^{28,29}

Processed genotypes were obtained from NIMH Data Archive (dx.doi.org/10.15154/1503209), and standard quality control procedures were applied. Samples and Single Nucleotide Polymorphisms (SNPs) with low call rates were excluded, and imputation was conducted using IMPUTE2 with 1000 Genomes (phase 3) reference panel after pre-phasing with SHAPEIT.^{30,31} Only those SNPs imputed with high confidence (INFO>0.8) were retained.

Polygenic scores for ADHD (PGS-ADHD) were derived from the 2016-2017 PGC + iPSYCH ADHD GWAS meta-analysis (20183 cases, 35191 controls) using the LDpred method.^{32,33} The PGS-ADHD score was standardized (mean=0, SD=1) and included in all models along with first ten genetic principal components (PCs) to account for potential genetic confounding **(eMethods in Supplement)**.

Covariates

All covariates were selected *a priori* based on known associations with DM use and/or ADHD-related symptoms.^{34,35} Analyses included age at T₀ visit, sex assigned at birth, socioeconomic status (SES), study sites, PGS-ADHD, and first 10 genetic PCs. Age was considered because DM use typically increases as children grow older.³⁶ Sex was included given well-documented differences in both DM use and ADHD prevalence.³⁷ SES, derived from parental education, household income, and neighborhood quality using principal component analysis, was included since lower SES has been associated with higher DM use.³⁸ Study site was included to account for site-specific variations. Age and sex were obtained from caregiver-reported developmental history questionnaires **(eMethods in Supplement)**.

Statistical analysis

Characteristics are presented as frequencies and percentages for categorical variables, and as mean with SDs for continuous variables. Prior to main analyses, associations between PGS-ADHD, individual DM use at T₀, and ADHD-related symptoms at T₀ were examined using Pearson correlation.

Longitudinal associations between average DM use and ADHD-related symptoms were analyzed using separate linear mixed models with random intercept and slopes for each DM category, examining combined and individual presentations of inattention and hyperactivity-impulsivity symptoms.³⁹ Fixed effects included DM use, time, age at T₀, sex, SES, PGS-

ADHD, and first ten genetic PCs, with study sites as random effects. Two-way interaction (DM use x time) captured longitudinal effects (i.e., between-person) and three-way interaction (DM use x time x PGS-ADHD) tested whether genetic predisposition moderated these effects. Models were fitted using ‘lmer’ function in lme4 package with restricted maximum likelihood.^{40,41} Statistical significance was set at $P < 0.05$, with Bonferroni correction for multiple comparisons ($0.05/9 = 0.005$). Standardized yearly effect sizes of $\beta > 0.05$ or cumulative $\beta > 0.10$ were considered meaningful.⁴²⁻⁴⁴

For significant longitudinal associations ($P < 0.005$, uncorrected), moderation models were fitted separately to test DM use x time interactions individually by sex, ADHD diagnosis at T_0 , and ADHD medication status at T_0 . ADHD diagnosis and medication use were determined from caregiver reports using the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS) and modified Medication Inventory survey. Cumulative effects of DM use on symptom change were estimated by regressing total change in ADHD-related symptoms ($T_4 - T_0$) on average DM use, adjusting for predefined covariates (**eMethods in Supplement**).

To assess the robustness of association between social media use and inattention symptoms, we conducted five additional analyses: (1) limiting the cohort to children born at-term (≥ 37 weeks’) to control for preterm birth; (2) restricting to typically developing children by excluding those with ADHD and co-occurring comorbidities (i.e., intellectual disabilities, conduct disorder, oppositional defiant disorder, or generalized anxiety disorder) identified via NIH Toolbox WISC-V and caregiver KSADS reports. In a previous ABCD study, $\sim 3.5\%$ of children met criteria for ADHD, and $\sim 70\%$ had at least one comorbidity.²⁸ Excluding these children reduced diagnostic heterogeneity and tested whether the observed negative association extended to children without underlying neurodevelopmental or psychiatric diagnoses; (3) including only children with behavioural data at all time points; (4) restricting analysis to T_3

due to lower T₄ follow-up; and 5) exploring whether average inattention symptoms predicted longer social media use (**eMethods in Supplement**).

We also conducted Cross-Lagged Panel Models to confirm the directionality of associations between social media use and inattention symptoms (**Figure 3**) (**eMethods in Supplement**).

All analyses were conducted using R, version 3.5 (RStudio, Boston, USA).

Results

Sample characteristics

Of the 11875 children recruited into the ABCD study cohort, 8324 children (mean [SD] age, 9.9 [0.6]; boys, n (%)=4408 (53.0) fulfilled our inclusion criteria at T₀) (**Table 1**). Throughout four follow-up waves, children spent an average of 1.4 hours/day on social media, 1.5 hours/day playing video games, and 2.3 hours/day watching television/videos. The cohort was ethnically diverse: White, 4356 (52.3%); Black, 1358 (16.3%); Asian, 496 (6.0%); Pacific Islander, 2 (0.02%); Native American, 183 (2.2%); Hispanic, 1763 (21.2%); and Other, 106 (1.3%). PGS-ADHD was positively associated with both ADHD symptoms and DM use at T₀ (**Figure 1**).

DM use and ADHD-related symptoms

The average use of playing video games (video games x time: β [SE], -0.05 [0.01]; $P < 0.001$) or watching television/videos (watching television/videos x time: β [SE], -0.05 [0.01]; $P < 0.001$) was associated with a decrease in hyperactivity-impulsivity symptoms over time (**Table 2**). In contrast, the average use of social media use was associated with an increase in inattention symptoms over time (social media x time: β [SE], 0.03 [0.01]; $P < 0.001$). To illustrate this effect, average social media use was divided into four quartiles and plotted against inattention symptoms over time (**Figure 2a**). A significant three-way interaction was observed between social media use, time, and PGS-ADHD with combined ADHD symptoms (social media x time x PGS-ADHD: β [SE], -0.04 [0.01]; $P = 0.002$). This indicates that the association between social media use and changes in combined ADHD symptoms over time differed depending on PGS-ADHD.

Additional variables, including sex, ADHD diagnosis, or medication status at T₀, did not moderate the association between DM use and ADHD-related symptoms over time (**eTable 1-3**).

Cumulative effect size of DM use on ADHD-related symptoms (T₄–T₀)

Average social media use was positively associated with an increase in inattention symptoms (β [SE], 0.15 [0.03]; $P < 0.001$) over the study period. To illustrate this effect, average social media use was divided into four quartiles and plotted against changes in inattention symptoms (**Figure 2b**). In contrast, neither the association between average time spent watching television/videos and total change in hyperactivity-impulsivity symptoms (β [SE], -0.06 [0.02]; $P = 0.004$), nor that between playing video games and total change in hyperactivity-impulsivity symptom change (β [SE], -0.05 [0.03]; $P = 0.06$), met our pre-defined threshold of $\beta = 0.10$ for a population or clinically relevant cumulative effect size.

Additional analyses

Additional analyses supported the robustness of association between social media use and inattention. Restricting the sample to children born at-term or to typically developing children yielded consistent effect sizes, indicating results were not driven by preterm birth or pre-existing conditions. Including only children with complete behavioural data or limiting follow-up to three years did not alter results. Potential recursive effects of inattention on later social media use were statistically significant but negligible (β [SE], -0.01 [0.001]; $P < 0.001$; total effect (T₄-T₀)=-0.01) (**Figure 2c**) (**eTable 4-8**).

Cross-lagged panel models confirmed a unidirectional association of social media use predicted increases in inattention symptom over time (β [SE], 0.03 [0.01]; $p = 0.004$), whereas inattention did not predict increased social media use (β [SE], -0.002 [0.004]; $p = 0.64$) (**Figure 3**).

Discussion

We investigated the long-term association between different types of DM use and changes in ADHD-related symptoms over four years. Consistent with our hypothesis, there was a positive interaction between average social media use and time, such that children with above-average social media use showed a greater increase in inattention symptoms compared to others. This interaction was specific to social media and not observed for either watching television/videos or playing video games. The associations remained robust across all additional analyses, indicating that findings were not driven by specific subgroups. Importantly, there was no evidence of reverse association, as average inattention symptoms did not predict increased social media use (**Figure 2c**), and cross-lagged panel models further supported one-directional relationship, with higher social media use preceding increases in inattention symptoms (**Figure 3**). Together, these results strengthen the interpretation of potentially causal link between social media use and changes in inattention symptoms.

In this study, social media use was specifically associated with a gradual increase in inattention symptoms over four years. Although yearly effect size was small (**Table 2**), cumulative effect over the study period was 0.15 (**Figure 2b**), showing a linear increase over time (**Figure 2a**). This finding aligns with prior research showing associations between digital multitasking or DM use and later inattention symptoms with comparable effect sizes ($d=0.16$ and $OR=1.11$, respectively).^{11,12} Importantly, the association persisted after accounting for genetic predisposition, and point to a higher degree of specificity, as similar patterns were not observed for playing video games or watching television/videos.

Although genetic liability for ADHD explained a significant portion of average ADHD symptomatology, it did not moderate the association between DM use and symptom change. Surprisingly, PGS-ADHD scores were strongly correlated with DM use (**Figure 1**), which may partly explain the cross-sectional associations between DM use and ADHD-related symptoms.

However, despite this shared genetic influence across all forms of DM use, only social media use was prospectively associated with gradual increase in inattention symptoms.

An effect size of 0.15 has small consequences for an individual's risk of meeting diagnostic criteria and likely minimal functional impact in daily life. However, at population level, such an effect could have substantial consequences. For example, assume a diagnosis of ADHD-inattentive type is met when inattention supersedes 1.65SD above population mean, corresponding to 5% of children. If social media use increases by 1SD across the population, a conservative estimate of behavioral change over the past decade and is associated with 0.15SD increase in inattention, the proportion of children exceeding this threshold would increase by about 35%. This relative increase translates to prevalence rising from 5% to ~6.8%. Using a more recent prevalence estimate of 11.3%, the same 35% increase would imply a rise to ~14.4%. Although hypothetical, these calculations illustrate how even small average shifts in symptoms can have meaningful consequences at population level.

We can only speculate about the mechanisms underlying the association between social media use and increased inattention symptoms. Social media platforms often involve constant messaging and notifications, which can disrupt attention and interfere with current activities. Experimental studies have shown that such interruptions, or even the mere presence of mobile phone nearby without using it, can impair attention and learning on psychological tests.⁴⁵ This can be contrasted with playing video games, which requires sustained attention, and both experimental and longitudinal studies,^{46,47} have been associated with improvements in cognitive function.

We found a steady increase in social media use from around 0.5hours/day at age 9 to 2.5 hours by age 13, despite most platforms such as Facebook and TikTok, requiring users to be at least 13. This early and increasing social media use underscores the need for stricter age verification and clearer guidelines for tech companies. Policymakers should reinforce regulations to limit

access for younger children and ensure platforms are age appropriate to support healthy development.

This study has several strengths, including its large sample size, longitudinal design, inclusion of genetic information, and detailed categorization of DM use. However, some limitations should be noted. DM use was self-reported rather than objectively measured. Although children and adolescents self-report of DM use is likely more accurate than parent report, it remains possible that children with more ADHD symptoms may misestimate their DM use, as difficulties with time estimation are well-known in ADHD. To address this, we conducted an additional analysis excluding children with ADHD and co-occurring comorbidities, and the findings persisted, suggestion robustness. Further, detailed information on specific activities, such as which games were played, which social media platforms were used, and the time of day, would have added valuable insight. Measuring screen time alone may be insufficient, as recent research suggests that the impact of DM depends more on the nature of engagement than on duration. ADHD assessments were relied on caregiver/parent reports from the KSADS and CBCL; teacher reports, which are often recommended for a more comprehensive evaluation, were not consistently available. Similarly, information on ADHD medication use was only available at T₀ and not tracked longitudinally, and families were not systematically asked about professional ADHD diagnoses across follow-ups. Finally, because ABCD study excluded children with sensory, or major neurological conditions, severe intellectual disabilities, moderate-to-severe autism spectrum disorder, or extremely-preterm birth, the findings may not be generalizable to these populations. Despite these limitations, it is unlikely that they would introduce systematic errors large enough to explain the observed association between social media use and inattention symptoms, or the differing trends seen between social media and gaming.

Conclusion

We identified an association between social media use and increased inattention symptoms, interpreted here as a likely causal effect. Although the effect size is small at individual level, it could have significant consequences if behavior changes across population level. These findings suggest that social media use may contribute to rising incidence of ADHD diagnoses. Future research should investigate the underlying mechanisms, examine the impact of specific social media behaviors, and evaluate strategies to mitigate potential risks.

Acknowledgements

The authors are grateful to the ABCD study members and their families for their participation. Data used in the preparation of this article were obtained from the Adolescent Brain Cognitive Development (ABCD) Study (<https://abcdstudy.org>), held in the NIMH Data Archive (NDA). This is a multisite, longitudinal study designed to recruit more than 10,000 children age 9–10 and follow them over 10 years into early adulthood. The ABCD Study® is supported by the National Institutes of Health and additional federal partners under award numbers U01DA041048, U01DA050989, U01DA051016, U01DA041022, U01DA051018, U01DA051037, U01DA050987, U01DA041174, U01DA041106, U01DA041117, U01DA041028, U01DA041134, U01DA050988, U01DA051039, U01DA041156, U01DA041025, U01DA041120, U01DA051038, U01DA041148, U01DA041093, U01DA041089, U24DA041123, U24DA041147. A full list of supporters is available at <https://abcdstudy.org/federal-partners.html>. A listing of participating sites and a complete listing of the study investigators can be found at https://abcdstudy.org/consortium_members/. ABCD consortium investigators designed and implemented the study and/or provided data but did not necessarily participate in the analysis or writing of this report. This manuscript reflects the views of the authors and may not reflect the opinions or views of the NIH or ABCD consortium investigators. The ABCD data repository grows and changes over time. The ABCD data used in this report came from the ABCD Data Release 5.0 (data accessed- 2023; DAR ID: 14933). We also would like to thank Professor Ulrika Ådén for providing feedback on the manuscript.

Data Availability statement

The data used for the analyses presented in this paper are from the Adolescent Brain Cognitive Development (ABCD) Study [<https://abcdstudy.org>; NIMH Data Archive (NDA)]. Data can be accessed by directly applying to the NDA.

Reference

1. Chen Q, Brikell I, Lichtenstein P, et al. Familial aggregation of attention-deficit/hyperactivity disorder. *Journal of Child Psychology and Psychiatry*. 2017;58(3):231-239.
2. Grimm O, Kittel-Schneider S, Reif A. Recent developments in the genetics of attention-deficit hyperactivity disorder. *Psychiatry and clinical neurosciences*. 2018;72(9):654-672.
3. Chang Z, Lichtenstein P, Asherson PJ, Larsson H. Developmental twin study of attention problems: high heritabilities throughout development. *JAMA psychiatry*. 2013;70(3):311-318.
4. Reuben C, Elgaddal N. Attention-Deficit/Hyperactivity Disorder in Children Ages 5-17 Years: United States, 2020-2022. *NCHS Data Brief*. Mar 2024;(499):1-9.
5. Visser SN, Danielson ML, Bitsko RH, et al. Trends in the parent-report of health care provider-diagnosed and medicated attention-deficit/hyperactivity disorder: United States, 2003-2011. *J Am Acad Child Adolesc Psychiatry*. Jan 2014;53(1):34-46.e2. doi:10.1016/j.jaac.2013.09.001
6. Kim JH, Kim JY, Lee J, et al. Environmental risk factors, protective factors, and peripheral biomarkers for ADHD: an umbrella review. *The Lancet Psychiatry*. 2020;7(11):955-970.
7. Nigg J, Nikolas M, Burt SA. Measured gene-by-environment interaction in relation to attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2010;49(9):863-873.
8. Martel MM, Nikolas M, Jernigan K, Friderici K, Waldman I, Nigg JT. The dopamine receptor D4 gene (DRD4) moderates family environmental effects on ADHD. *Journal of abnormal child psychology*. 2011;39:1-10.
9. Nikkelen SW, Valkenburg PM, Huizinga M, Bushman BJ. Media use and ADHD-related behaviors in children and adolescents: A meta-analysis. *Developmental psychology*. 2014;50(9):2228.
10. Masi L, Abadie P, Herba C, Emond M, Gingras MP, Amor LB. Video Games in ADHD and Non-ADHD Children: Modalities of Use and Association With ADHD Symptoms. *Front Pediatr*. 2021;9:632272. doi:10.3389/fped.2021.632272
11. Baumgartner SE, van der Schuur WA, Lemmens JS, te Poel F. The relationship between media multitasking and attention problems in adolescents: Results of two longitudinal studies. *Human Communication Research*. 2018;44(1):3-30.
12. Ra CK, Cho J, Stone MD, et al. Association of digital media use with subsequent symptoms of attention-deficit/hyperactivity disorder among adolescents. *Jama*. 2018;320(3):255-263.
13. Allen MS, Vella SA. Screen-based sedentary behaviour and psychosocial well-being in childhood: cross-sectional and longitudinal associations. *Mental Health and Physical Activity*. 2015;9:41-47.
14. Barlett ND, Gentile DA, Barlett CP, Eisenmann JC, Walsh DA. Sleep as a mediator of screen time effects on US children's health outcomes: A prospective study. *Journal of Children and Media*. 2012;6(1):37-50.
15. Thorell LB, Burén J, Ström Wiman J, Sandberg D, Nutley SB. Longitudinal associations between digital media use and ADHD symptoms in children and adolescents: a systematic literature review. *European Child & Adolescent Psychiatry*. 2022:1-24.
16. Yang A, Rolls ET, Dong G, et al. Longer screen time utilization is associated with the polygenic risk for Attention-deficit/hyperactivity disorder with mediation by brain white matter microstructure. *EBioMedicine*. Jun 2022;80:104039. doi:10.1016/j.ebiom.2022.104039
17. Adolescent Brain Cognitive Development (ABCD) Study. Accessed 27-08-2023, 2023. <https://wiki.abcdstudy.org/release-notes/start-page.html>.

18. Casey BJ, Cannonier T, Conley MI, et al. The Adolescent Brain Cognitive Development (ABCD) study: Imaging acquisition across 21 sites. *Dev Cogn Neurosci*. 08 2018;32:43-54. doi:10.1016/j.dcn.2018.03.001
19. Auchter AM, Hernandez Mejia M, Heyser CJ, et al. A description of the ABCD organizational structure and communication framework. *Developmental Cognitive Neuroscience*. 2018/08/01/ 2018;32:8-15. doi:<https://doi.org/10.1016/j.dcn.2018.04.003>
20. Bauermeister JJ, Barkley RA, Bauermeister JA, Martínez JV, McBurnett K. Validity of the sluggish cognitive tempo, inattention, and hyperactivity symptom dimensions: neuropsychological and psychosocial correlates. *J Abnorm Child Psychol*. Jul 2012;40(5):683-97. doi:10.1007/s10802-011-9602-7
21. Hair Jr JF, Black WC, Babin BJ, Anderson RE. Multivariate data analysis. *Multivariate data analysis*. 2010:785-785.
22. Sauce B, Liebherr M, Judd N, Klingberg T. The impact of digital media on children's intelligence while controlling for genetic differences in cognition and socioeconomic background. *Scientific reports*. 2022;12(1):7720-7720. doi:10.1038/s41598-022-11341-2
23. Nivins S, Sauce B, Liebherr M, Judd N, Klingberg T. Long-term impact of digital media on brain development in children. *Scientific Reports*. 2024/06/06 2024;14(1):13030. doi:10.1038/s41598-024-63566-y
24. Bagot KS, Tomko RL, Marshall AT, et al. Youth screen use in the ABCD® study. *Developmental Cognitive Neuroscience*. 2022/10/01/ 2022;57:101150. doi:<https://doi.org/10.1016/j.dcn.2022.101150>
25. Brener ND, Billy JO, Grady WR. Assessment of factors affecting the validity of self-reported health-risk behavior among adolescents: evidence from the scientific literature. *J Adolesc Health*. Dec 2003;33(6):436-57. doi:10.1016/s1054-139x(03)00052-1
26. Riley AW. Evidence that school-age children can self-report on their health. *Ambulatory Pediatrics*. 2004;4(4):371-376.
27. Nagata JM, Cortez CA, Cattle CJ, et al. Screen Time Use Among US Adolescents During the COVID-19 Pandemic: Findings From the Adolescent Brain Cognitive Development (ABCD) Study. *JAMA Pediatrics*. 2022;176(1):94-96. doi:10.1001/jamapediatrics.2021.4334
28. Cordova MM, Antovich DM, Ryabinin P, et al. Attention-Deficit/Hyperactivity Disorder: Restricted Phenotypes Prevalence, Comorbidity, and Polygenic Risk Sensitivity in the ABCD Baseline Cohort. *J Am Acad Child Adolesc Psychiatry*. Oct 2022;61(10):1273-1284. doi:10.1016/j.jaac.2022.03.030
29. Baurley JW, Edlund CK, Pardamean CI, Conti DV, Bergen AW. Smokescreen: a targeted genotyping array for addiction research. *BMC genomics*. 2016;17(128):145-145. doi:10.1186/s12864-016-2495-7
30. Howie BN, Donnelly P, Marchini J. A flexible and accurate genotype imputation method for the next generation of genome-wide association studies. *PLoS genetics*. 2009;5(6):e1000529.
31. Delaneau O, Zagury J-F, Marchini J. Improved whole-chromosome phasing for disease and population genetic studies. *Nature methods*. 2013;10(1):5-6.
32. Demontis D, Walters RK, Martin J, et al. Discovery of the first genome-wide significant risk loci for attention deficit/hyperactivity disorder. *Nature genetics*. 2019;51(1):63-75.
33. Vilhjálmsson BJ, Yang J, Finucane HK, et al. Modeling linkage disequilibrium increases accuracy of polygenic risk scores. *The american journal of human genetics*. 2015;97(4):576-592.
34. Green A, Baroud E, DiSalvo M, Faraone SV, Biederman J. Examining the impact of ADHD polygenic risk scores on ADHD and associated outcomes: A systematic review and meta-analysis. *J Psychiatr Res*. Nov 2022;155:49-67. doi:10.1016/j.jpsychires.2022.07.032

35. Russell AE, Ford T, Williams R, Russell G. The Association Between Socioeconomic Disadvantage and Attention Deficit/Hyperactivity Disorder (ADHD): A Systematic Review. *Child Psychiatry Hum Dev*. Jun 2016;47(3):440-58. doi:10.1007/s10578-015-0578-3
36. Sina E, Buck C, Veidebaum T, et al. Media use trajectories and risk of metabolic syndrome in European children and adolescents: the IDEFICS/I.Family cohort. *International Journal of Behavioral Nutrition and Physical Activity*. 2021/10/18 2021;18(1):134. doi:10.1186/s12966-021-01186-9
37. Twenge JM, Martin GN. Gender differences in associations between digital media use and psychological well-being: Evidence from three large datasets. *J Adolesc*. Feb 2020;79:91-102. doi:10.1016/j.adolescence.2019.12.018
38. Ke Y, Chen S, Hong J, Liang Y, Liu Y. Associations between socioeconomic status and screen time among children and adolescents in China: A cross-sectional study. *PLoS One*. 2023;18(3):e0280248. doi:10.1371/journal.pone.0280248
39. Cnaan A, Laird NM, Slasor P. Using the general linear mixed model to analyse unbalanced repeated measures and longitudinal data. *Statistics in medicine*. 1997;16(20):2349-2380.
40. Bates D, Mächler M, Bolker B, Walker S. Fitting linear mixed-effects models using lme4. *arXiv preprint arXiv:1406.5823*. 2014;
41. R Core Team R. R: A language and environment for statistical computing. 2013;
42. Kraft MA. Interpreting effect sizes of education interventions. *Educational researcher*. 2020;49(4):241-253.
43. Abelson RP. A variance explanation paradox: When a little is a lot. *Psychological bulletin*. 1985;97(1):129.
44. Pogrow S. How effect size (practical significance) misleads clinical practice: The case for switching to practical benefit to assess applied research findings. *The American Statistician*. 2019;73(sup1):223-234.
45. Van Der Schuur WA, Baumgartner SE, Sumter SR, Valkenburg PM. The consequences of media multitasking for youth: A review. *Computers in Human Behavior*. 2015;53:204-215.
46. Bediou B, Adams DM, Mayer RE, Tipton E, Green CS, Bavelier D. Meta-analysis of action video game impact on perceptual, attentional, and cognitive skills. *Psychological bulletin*. 2018;144(1):77.
47. Sauce B, Liebherr M, Judd N, Klingberg T. The impact of digital media on children's intelligence while controlling for genetic differences in cognition and socioeconomic background. *Scientific Reports*. 2022/05/11 2022;12(1):7720. doi:10.1038/s41598-022-11341-2

Figure 1 Association between polygenic scores, digital media use at T₀, and attention deficit hyperactivity disorder symptoms at T₀. Abbreviations: TV, television, ADHD, attention deficit hyperactivity disorder; PGS, polygenic risk scores.

Figure 2 (a) Average social media use and inattention symptoms plotted over time, where average social media use (hours/day) was segregated into different quartiles (Q1, 0.61; Q2, ≥ 0.61 to < 1.22 ; Q3, ≥ 1.22 to < 1.97 ; Q4, ≥ 1.97). Children in the highest quartiles of average social media use showed a significant increase in inattention symptom scores over time; (b) Change in inattention symptom scores over four years, plotted for each quartile of average social media use; and (c) Change in social media use over four years, plotted for each quartile for average inattention symptoms

Figure 3 Cross-Lagged Panel Models showing associations between social media use and inattention symptoms. All coefficients are standardized β weights. Abbreviations: I, Inattention symptoms; and S, social media use. 0 to 4 represent the follow-up timepoints. ** $p < 0.01$; *** $p < 0.001$

Tables

Table 1 Characteristics of the study population (N=8324)

Characteristics	Overall cohort
Age at T ₀ (years)	9.9 (0.6)
Sex, male	4408 (53.0%)
Race or Ethnicity	
White	4356 (52.3%)
Black	1358 (16.3%)
Asian	496 (6.0%)
Pacific Islander	2 (0.02%)
Native American	183 (2.2%)
Hispanic	1763 (21.2%)
Other	106 (1.3%)
Maternal education	
High school or less	121 (1.5%)
high school	411 (4.9%)
High school graduate	833 (10.0%)
Bachelor's degree	2369 (28.5%)
Some college/associate degree	2413 (29.0%)
Master's degree	1639 (19.7%)
Professional degree	528 (6.3%)
Parental income	
≤ 49,999	2206 (26.5%)
50,000 – 74,999	1056 (12.7%)
75,000 – 99,999	1132 (13.6%)
100,000 – 199,999	2359 (28.3%)
≥ 200,000	886 (10.6%)
Socioeconomic status	0.03 (1.4)
Raw polygenic risk score for ADHD	-7.5 (0.3)
Social media use (hours/day)	
T ₀	0.5 (1.1)
T ₁	0.9 (1.6)
T ₂	1.3 (1.4)
T ₃	1.9 (1.5)
T ₄	2.5 (1.4)
Playing video games (hours/day)	
T ₀	1.0 (1.1)
T ₁	1.2 (1.2)
T ₂	1.5 (1.4)
T ₃	1.9 (1.5)
T ₄	2.0 (1.6)
Watching television/videos (hours/day)	
T ₀	2.2 (1.8)
T ₁	2.4 (1.8)
T ₂	2.3 (1.3)
Total digital media use by children at T ₀ (hours/day)	3.7 (3.0)
Average estimated time spent by children over four years (hours/day)	
Social media use	1.4 (1.0)
Playing video games	1.5 (1.0)
Watching television/videos	2.3 (1.3)

Total digital media use by children as reported by parents at T ₀ (hours/day)	3.0 (2.4)
ADHD stimulant medications	778 (0.9)
ADHD-related Symptoms at T ₀	
Combined ADHD	2.7 (3.0)
Inattention	1.7 (1.9)
Hyperactivity-impulsivity	1.3 (1.8)

Data are given as mean (SD) or n (%) unless otherwise indicated.

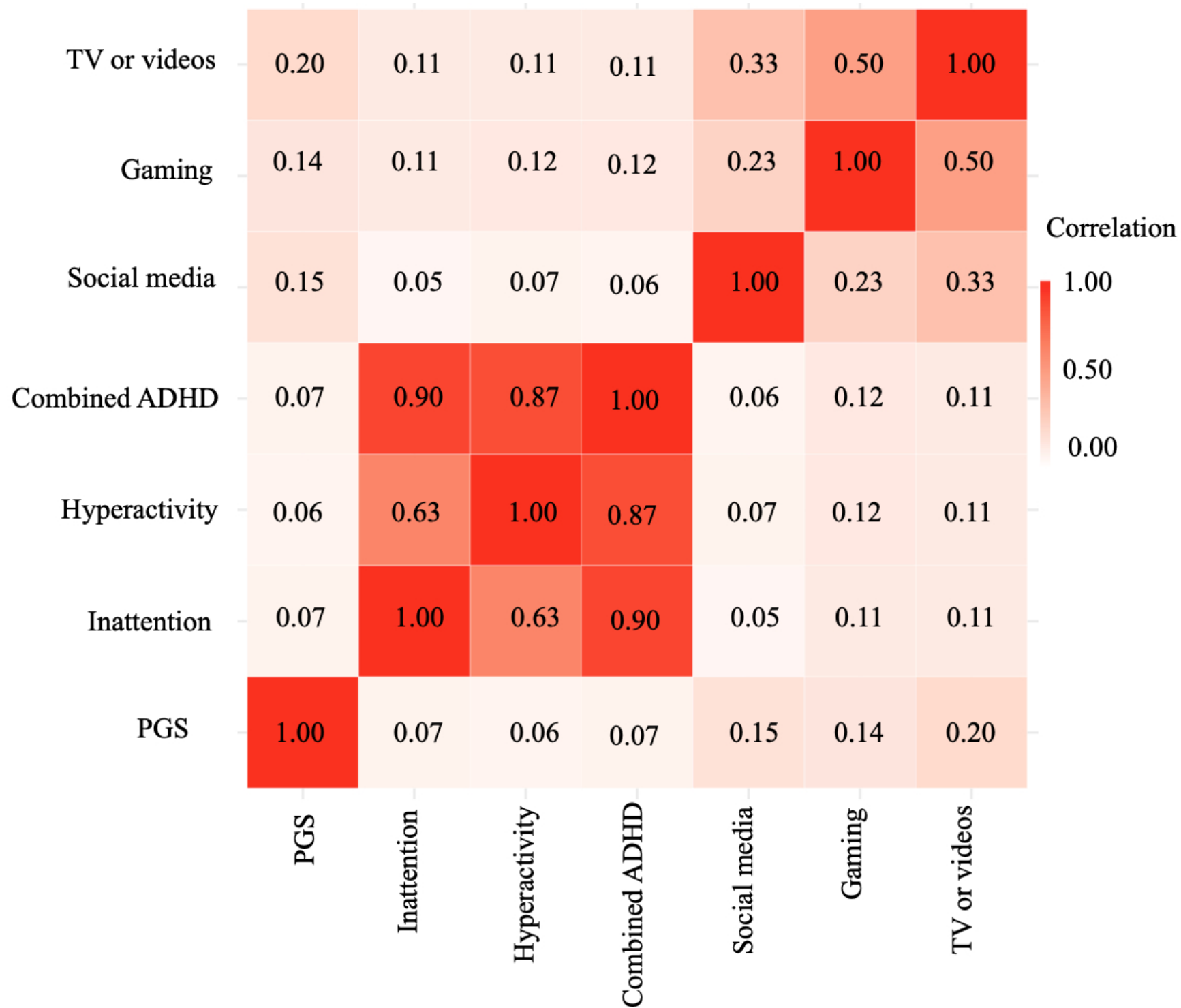
Abbreviations: ADHD, attention-deficit hyperactivity disorder; T₀, baseline; T₁, one-year follow-up; T₂, two-year follow-up; T₃, three-year follow-up; and T₄, four-year follow-up.

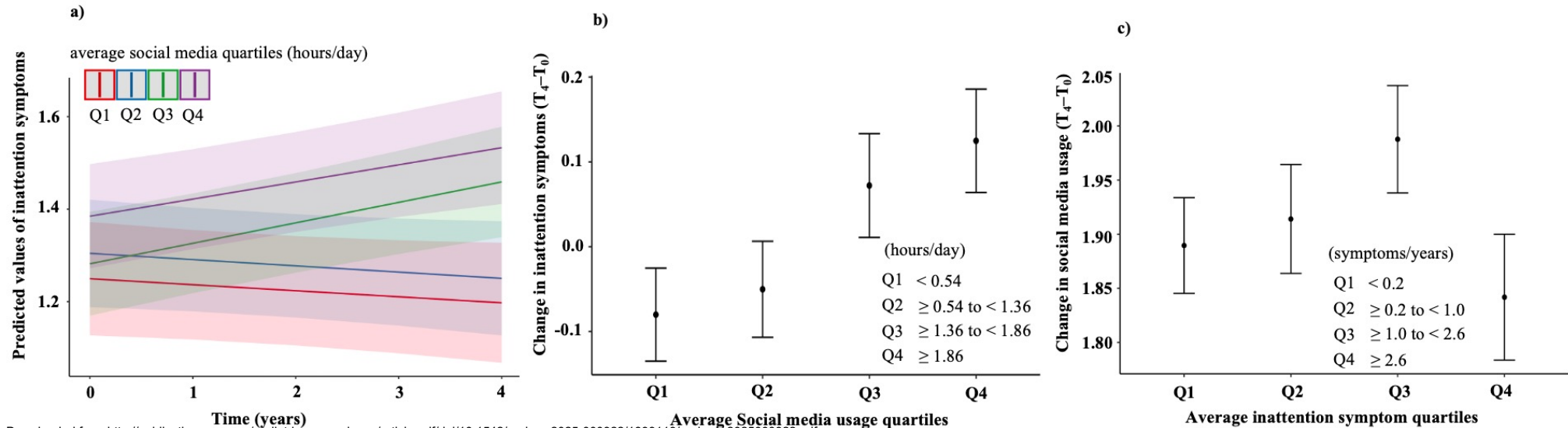
Table 2 Association between digital media use over four years and ADHD-related symptoms in the overall cohort (N=8324)

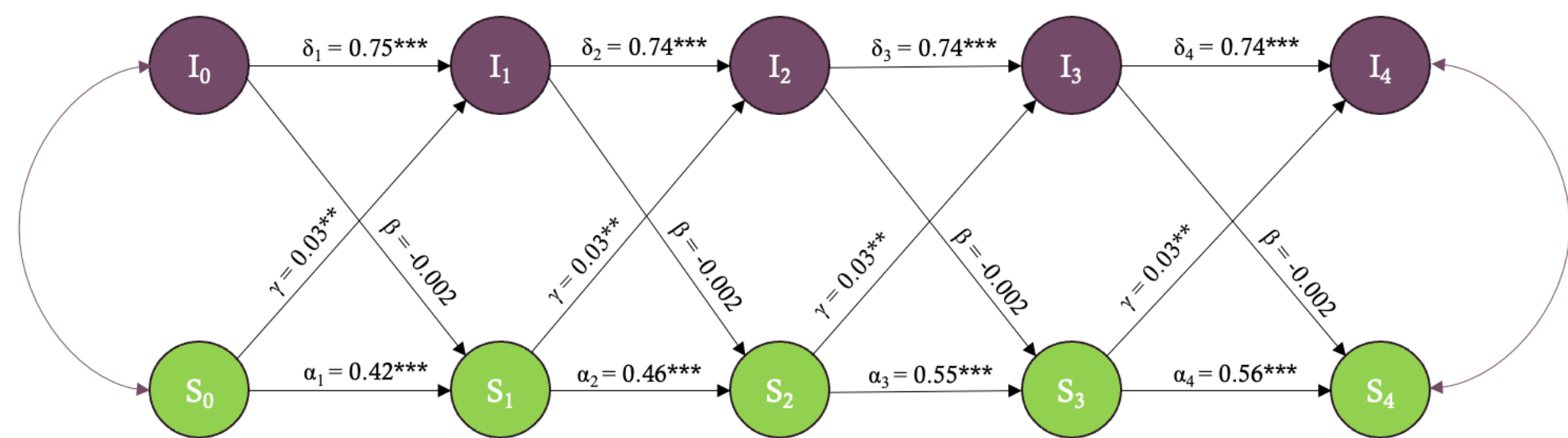
Symptoms	Social media use	SES	PGS-ADHD	Social media use x Time	Social media use x Time x PGS-ADHD
Combined ADHD	0.11 (0.04)	-0.22 (0.04)***	0.54 (0.05)***	0.03 (0.01)	-0.04 (0.01)**
Inattention	0.05 (0.02)	-0.14 (0.02)***	0.29 (0.02)***	0.03 (0.01)***	-0.01 (0.01)
Hyperactivity-impulsivity	0.21 (0.04)***	-0.25 (0.04)***	0.50 (0.05)***	-0.03 (0.01)	-0.01 (0.01)
	Playing video games	SES	PGS-ADHD	Playing video games x Time	Playing video games x Time x PGS-ADHD
Combined ADHD	0.30 (0.04)***	-0.17 (0.04)***	0.51 (0.05)***	-0.01 (0.01)	-0.01 (0.01)
Inattention	0.21 (0.02)***	-0.11 (0.02)***	0.27 (0.02)***	-0.01 (0.01)	-0.01 (0.01)
Hyperactivity-impulsivity	0.33 (0.04)***	-0.22 (0.04)***	0.48 (0.05)***	-0.05 (0.01)***	-0.01 (0.01)
	Watching television or videos	SES	PGS-ADHD	Watching television or videos x Time	Watching television or videos x Time x PGS-ADHD
Combined ADHD	0.39 (0.04)***	-0.14 (0.04)**	0.49 (0.05)***	-0.02 (0.01)	-0.02 (0.01)
Inattention	0.23 (0.02)***	-0.10 (0.02)***	0.26 (0.02)***	-0.01 (0.01)	-0.01 (0.01)
Hyperactivity-impulsivity	0.46 (0.04)***	-0.17 (0.04)***	0.46 (0.05)***	-0.05 (0.01)***	-0.01 (0.01)

Data are presented as standardized beta (standard error). Abbreviations: ADHD, attention-deficit/hyperactivity disorder; SES, socioeconomic status; and PGS, polygenic risk scores.

Significance levels are presented as ***<0.001, **<0.005 (Bonferroni corrected).







Supplemental Information

Digital media, Genetics and change in ADHD Symptoms in Children – a Longitudinal Study

Samson Nivins PhD¹, Michael A. Mooney PhD², Joel Nigg PhD³,
Torkel Klingberg PhD¹

Affiliations: ¹Department of Neuroscience, Karolinska Institute, Stockholm, Sweden; ²Division of Bioinformatics and Computational Biology, Oregon Health & Science University, Portland, Oregon, United States; ³Division of Clinical Psychology, Oregon Health & Science University, Portland, Oregon, United States

Address correspondence to Torkel Klingberg, Department of Neuroscience, Karolinska Institutet, Stockholm 17165, Sweden [torkel.klingberg@ki.se]

Table of contents**Methods**

Sampling design.....	4
Digital media usage.....	4
Self-reported survey.....	4
Parent-reported survey.....	5
ADHD-related symptoms	6
ADHD diagnosis.....	6
Exclude children with other comorbid conditions	7
Intellectual developmental disorder	7
Conduct disorder.....	7
Oppositional defiant disorder.....	7
Generalized anxiety disorder	7
Medication status	7
Covariates	8
Socioeconomic status.....	8
Genotyping.....	8
Statistics	9
Table S1 Sex-specific effect on the association between digital media usage over four years and ADHD-related symptoms in the overall cohort (N=8324)	11
Table S2 Effect of ADHD diagnosis status on the association between digital media usage over four years and ADHD-related symptoms in the overall cohort (N=8324).....	12
Table S3 Effect of ADHD medication status on the association between digital media usage over four years and ADHD-related symptoms in the overall cohort (N=8324).....	13
Table S4 Association between social media usage and ADHD-related symptoms in children born at term (n=6986)	14
Table S5 Association between social media usage and ADHD-related symptoms in children without any neurodevelopmental conditions (n=6631)	15
Table S6 Association between social media usage and ADHD-related symptoms in children with behavioural data available across all follow-ups (N=3414)	16
Table S7 Association between social media usage and ADHD-related symptoms in children with three years of follow-ups (N=7215)	17
Table S8 Association between average inattention symptoms and social media usage in children over four years	18
Figure S1 Correlation between individual DM usage across four different waves of follow-up	19
Figure S2 Confirmatory Factor Analysis of ADHD symptoms. Rectangles represent information directly measured (observed), and each rectangle in the model represents an	

individual item. Circles represent latent variables or unobserved variables that are not directly measured. Factor loadings are shown for observed variables on the latent factors.....	20
References.....	21

Methods

Sampling design

The ABCD study sample comprised 11875 children (9780 singleton births; 47.8% male; 52.1% white) aged 9-10 years, along with their parents/guardians, at baseline. They were recruited across 21 data collection sites and will be followed for at least 10 years.¹ This recruitment cohort closely matches the sociodemographic composition of the U.S. population of 9-10-year-old children. At each data-collection site, the majority of the children were enrolled through local elementary and charter schools, while a minority were recruited through non-school-based community outreach and word-of-mouth referrals. Twins were identified and recruited from birth registries.²

Digital media usage

The estimated time spent on individual digital media (DM) use, including social media use, playing video games, or watching television/videos, was assessed at all visits (baseline visit (T₀), one-year later (T₁), two-year later (T₂), three-year later (T₃), and four-year later (T₄)) using the Youth Screen Time Survey.

Self-reported survey

At each visit, children reported the number of hours they spent on a typical weekday (Monday to Friday during the school years and holiday/school breaks) and weekend (Saturday and Sunday) days by device, media platform, or activity excluding the number of hours spent on school-related work.

These activities included:

- (1) watching television or movies
- (2) watching videos (e.g., YouTube)
- (3) playing video games on a computer, console, phone, or another device (e.g., Xbox, PlayStation, iPad)
- (4) Texting on a cell phone, tablet, or computer (e.g., Google Chat, WhatsApp)
- (5) Visiting social networking sites (e.g., Facebook, Twitter, Instagram)
- (6) Using video chat (e.g., Skype, FaceTime).

DM use was categorized as follows (a) social media use (combining activities 4, 5, and 6); (b), playing video games (activity 3); or (c) watching television/videos (combining activities 1 and 2). The response options included were none - '0', < 30 minutes - '0.25', 30 minutes - '0.5', 1 hour - '1', 2 hours - '2', 3 hours - '3', or > 4 hours - '4'.

To calculate the average hours spent per day for individual DM use, we used the following formula: (total number of hours spent on a weekday * 5 + the total number of hours on a weekend day * 2)/7.

During both the T₀ and T₁ visits, details on the duration of individual DM use were collected using the same categorical scale as described earlier. However, starting from the T₂ visit onwards, slight modifications were made to the Youth Screen Time Survey due to the increasing prevalence of DM use among children. Specifically, the category 'watching television' was changed to 'watching or streaming videos or movies', and 'watching videos (such as YouTube)' was changed to 'watching or streaming videos or live streaming (such as YouTube, Twitch)'. These categories were combined into a single category labeled 'watching television/videos'. Similarly, activities such as 'video chatting, visiting social media apps, and texting cell phone' were merged into a category named 'social media use'. The activities 'editing photos and videos' and 'searching or browsing the internet' were excluded as it does

not co-exist with the T₀ details. In addition, the category ‘playing video games’ was further divided into two sub-categories: ‘time spent on single-player’ and ‘time spent on multi-player’, which were combined into a single category labeled ‘playing video games’.

Furthermore, the response format was changed from categorical to continuous, with response options ranging from 0 minutes, 15 minutes, 30 minutes, 45 minutes, 1 hour, 1.5 hours, 2 hours, 2.5 hours, 3 hours, and every additional hour up until 24 hours. To keep the data consistent across all time points, the data from the T₂, T₃, and T₄ visits were harmonized with the T₀ and T₁ visits data. These visits were re-coded into the following categories: none – ‘0’, < 30 minutes – ‘0.25’, 30 minutes – ‘0.5’, 1 hour – ‘1’, 1.15 hours – ‘1.25’, 1.30 hours – ‘1.5’, 2 hours – ‘2’, 2.15 hours – ‘2.25’, 2.30 hours – ‘2.5’, 3 hours – ‘3’, 3.15 hours – ‘3.25’, 3.30 hours – ‘3.5’, and > 4 hours – ‘4’.

There were good test-retest correlations between DM usage across different waves, ranging from 0.20 to 0.57 (**Supplemental Figure S1**).

Parent-reported survey

Caregivers/parents were asked to report the number of hours spent by their child on a typical weekday and weekend days in total on watching television, shows or videos, texting or chatting, playing games, or visiting social networking sites (Facebook, Twitter, Instagram), excluding the number of hours spent on school-related work during T₀ and T₁ visits. Parents reported the total estimated time spent on these activities in hours and minutes for weekdays and weekends. To calculate the average hours spent on screen time per day, we used the following: (total number of hours spent on a weekday * 5 + the total number of hours on a weekend day * 2)/7.

Further, we assessed the agreement between caregivers/parents and child reports for DM use at the T₀ visit using a correlation coefficient and found it to be 0.43, indicating moderate agreement between them.

We used the self-reported survey completed by children over caregivers/parents, since caregivers/parents may not be fully aware of what kind and type of DM are used by these 9-10-year-old children or older children. Children of this age range use DM without being supervised, for example, in their bedrooms at night. Therefore, children may report their estimated time spent on each DM use more precisely than their caregivers/parents. There is also substantial evidence showing that children as young as six years old can reliably report about their own health.³

Due to covid lockdown, it is more likely that these children could spend more time using DM than anticipated at T₀. This was more evident in a US-based study, where they reported a two-fold increase in the estimated time spent on DM use during the COVID lockdown compared to the pre-pandemic period.⁴ Therefore, to account for an increase in estimated time spent using DM amongst children between T₀ and T₄, we used an average estimated time spent for individual DM use rather than just considering only T₀ or T₄ for the longitudinal analyses. The average estimated time spent for individual DM use was calculated by averaging the estimated time spent for individual DM use across all time points. For example, playing video games = (T₀ + T₁ + T₂ + T₃ + T₄)/5.

ADHD-related symptoms

ADHD-related symptoms were assessed by the Child Behaviour Checklist (CBCL) questionnaire from the Achenbach System of Empirically Based Assessment completed by the accompanying caregivers/parents of a child across all visits. It consists of 118 items, and answers are given on a three-point Likert scale (0=not true, 1=somewhat or sometimes true, and 2=very true or often true).

For our analysis, we used combined ADHD-related symptoms based on the Diagnostic and Statistical Manual of Mental Disorders (*DSM*)-5-defined ADHD CBCL subscale constructed by experts (i.e., *cbcl_scr_dsm5_adhd*). Absolute scores were used for all measures. Coefficient alpha was satisfactory at all time points with α 's > 0.72.

We also categorized the presentation of ADHD symptoms into 'inattention' and 'hyperactivity-impulsivity' separately. Items from the CBCL questionnaire were selected based on the *DSM-IV* and *DSM-5* criteria. Inattention items included: "easily distracted, can't concentrate, sustained attention, and poor schoolwork". Hyperactivity-impulsivity items included: "impulsive, talks too much, hyperactive, poor coordination, and loud".

A confirmatory factor analysis (CFA) was conducted to verify the factor structure (comparative fit index=0.96, root mean square error of approximation=0.07, 90% CI [0.07, 0.08], standardized root mean square residual=0.03; factor loadings are provided in **Supplemental Figure S2**). Items with factor loadings below 0.30 were removed. Scores for the retained items were then summed to create inattention and hyperactivity-impulsivity symptom scores, which were treated as continuous variables. Higher scores reflect higher levels of ADHD-related symptoms.

ADHD diagnosis

The presence of ADHD symptoms (either past or current), in the child, was evaluated through the caregivers/parents reports based on the computerized Kiddie-Structured Assessment for Affective Disorders and Schizophrenia (KSADS) at the T₀ visit. This tool is based on a well-studied and validated tool, both in research and clinical settings. Diagnoses of ADHD were made in accordance with *DSM-5* criteria, which require an endorsement of six or more symptoms of inattention or hyperactivity-impulsivity.

At T₀, children were assessed for ADHD diagnosis based on the caregivers/parents reporting through the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS).

Inattention symptom scores were computed as follows.

The scores were counted as one for inattentive items -

$$\begin{aligned} & \text{if } (ksads_14_76_p = 1 \ \& \ ksads_14_77_p = 1) = 1 \\ & \text{if } (ksads_14_80_p = 1 \ \& \ ksads_14_81_p = 1) = 1 \end{aligned}$$

For the remaining inattentive items, the scores were counted as one for each -

$$ksads_14_76_p, ksads_14_80_p, ksads_14_394_p, ksads_14_395_p, ksads_14_396_p, ksads_14_397_p, ksads_14_398_p, ksads_14_399_p, ksads_14_400_p$$

Similarly, for hyperactivity-impulsivity symptom scores were computed as follows:

$$\text{if } (ksads_14_84_p = 1 \ \& \ ksads_14_85_p = 1) \text{ or, if } (ksads_14_88_p = 1) = 1$$

For the remaining hyperactivity-impulsivity items, the scores were counted as one for each -

ksads_14_401_p, ksads_14_402_p, ksads_14_403_p, ksads_14_404_p,
ksads_14_405_p, ksads_14_406_p, ksads_14_407_p,
ksads_14_408_p

Excluded children with other comorbid conditions

Intellectual developmental disorder

We used the NIH Toolbox WISC-V Matrix Reasoning total scale score of less than or equal to three to exclude children with intellectual developmental disorders. This is equivalent to an estimated IQ ≤ 70).

pea_wiscv_tss ≤ 3

To exclude the children with conduct, oppositional defiant disorder, or generalized anxiety disorder, we used the KSADS reported by caregivers/parents.

Conduct disorder

The scores were counted as one for each item:

ksads_16_449_p, ksads_16_463_p, ksads_16_453_p, ksads_16_461_p,
ksads_16_465_p, ksads_16_98_p, ksads_16_104_p + ksads_16_102_p,
ksads_16_457_p + ksads_16_455_p, ksads_16_451_p,
ksads_16_106_p, ksads_16_100_p, ksads_16_447_p,
ksads_16_459_p

Oppositional defiant disorder

The scores were counted as one for each item:

ksads_15_95_p, ksads_15_436_p, ksads_15_435_p, ksads_15_433_p,
ksads_15_93_p, ksads_15_432_p, ksads_15_91_p, ksads_15_437_p,
ksads_15_434_p

Generalized anxiety disorder

The scores were counted as one for each item:

ksads_10_45_p, ksads_10_320_p, ksads_10_324_p, ksads_10_328_p,
ksads_10_326_p, ksads_10_47_p,
ksads_10_322_p

Medication status

Caregivers/parents were asked to bring along the prescribed medication used by their child in the last two weeks during the T₀ visit. They also completed the Medication Inventory survey modified from the PhenX instrument, listing the names and dosages of all medications taken by the child. The details of the ADHD medications are provided below.

ADHD (stimulant and non-stimulant medications)

- a) Methylphenidate Derivative such as Ritalin, Concerta
- b) Amphetamines such as Adderall, Vyvanse
- c) Alpha agonist such as Intuniv, Tenex
- d) Atomoxetine such as Strattera.

Covariates

All the covariates were selected prior to the analysis. Age at T₀ visit, sex assigned at birth (boy or girl), socioeconomic status (SES), scanner sites, PGS for ADHD, and for the top ten ancestries informative genetic principal components (10PCs of genetics). Age and biological sex were retrieved from the Developmental History Questionnaire survey completed by the caregivers/parents.

Socioeconomic status

Data on parental education, household income, and neighbourhood quality were collected by the ABCD study team during the T₀ visit. Parental education was defined as the highest level of education completed by parents or caregivers, categorized as, middle school or less, some high school, high school graduate, some college/associate degree, bachelor's degree, a master's degree, or professional degree. Household income was determined by the combined annual income of all family members over the past 12 months, categorized as less than \$49,999; \$50,000–74,999; \$75,000–99,999; \$100,000–199,999; and greater than \$200,000. Neighborhood quality was assessed using the Area Deprivation Index (ADI) scores (reshist_addr1_adi_wsum), which were based on the children's primary home addresses.⁵ The ADI score provides a measure of socioeconomic disadvantage at the neighborhood level, based on 17 metrics from Census data including poverty, education, employment, and housing quality, with higher scores indicating greater neighborhood deprivation.^{6,7}

All three variables were included in the Principal Component Analysis (PCA), with each loading onto the first component (household income, parental education, ADI; loadings of 0.63, 0.56, and -0.53 respectively), which accounted for over 60% of the variance. This primary component was utilized in all subsequent analyses. The SES scores were normalized with a mean of 0 and a standard deviation of 1.

Genotyping

Saliva samples were collected from all the children at the T₀ visit. The detailed procedure for genotyping has been described previously.⁸

Briefly, the Rutgers University Cell and DNA Repository stored and genotyped all the samples using the Affymetrix NIDA SmokeScreen array.⁹ The processed genotypes were downloaded from the NIMH Data Archive (dx.doi.org/10.15154/1503209), and standard quality control checks were performed.

One batch of samples had a significantly lower call rate (~85%) than others (~98%) as calculated by quality control procedures using GWAS Tools and was removed (N=126 samples). After initial quality control checks, Single Nucleotide Polymorphisms (SNPs) with an adequate call rate of > 94% were retained. SNP allele frequencies, as calculated by GWAS Tools, were examined for differences between batches, and no significant batch effects were found. To control for possible population stratification, PCA was conducted using the PC-Air method in the GENESIS Bioconductor package.¹⁰ SNPs that were not initially genotyped were imputed with IMPUTE2 software using 1000 genomes (1KG phase 3) as the reference panel (https://mathgen.stats.ox.ac.uk/impute/1000GP_Phase3.html).¹¹ Autosomal chromosomes were pre-processed and phased using SHAPEIT,¹² where variant positions and alleles were checked against the 1000 genomes reference panel. Only those SNPs imputed with high confidence (INFO > 0.8) were retained. Genotype probabilities were converted to best-guess genotypes, with the genotype set to missing if the probability < 0.8.

The polygenic risks score (PGS) was constructed using the 2016-2017 PGC + iPSYCH ADHD GWAS meta-analysis,¹⁰ as the discovery data set (20,183 ADHD cases; 35,191 controls), which was calculated using the LDpred method.¹³ Only SNPs with INFO (imputation quality) score > 0.8 in both the PGC meta-analysis and the ABCD data were considered. SNPs were further limited to the ~1.2 million HapMap SNPs as suggested for LDpred. Linkage disequilibrium was estimated using all unrelated individuals in the ABCD cohort, and the PGS was created with the proportion of causal SNPs set to 0.3, given that ADHD is known to be highly polygenic. PGS-ADHD was then standardized in our sample to mean=0 and SD=1. We also used the first 10 genomic PCs as covariates in our analyses.

Statistics

To investigate the longitudinal association of DM usage on combined and individual presentation of ADHD symptoms the following model was used:

$$\begin{aligned} \text{ADHD-related symptoms} \sim & \beta_0 + \beta_1 (\text{average individual DM use}) + \beta_2 (\text{Time}) + \beta_3 (\text{average} \\ & \text{individual DM use} \times \text{Time}) + \beta_4 (\text{SES}) + \beta_5 (\text{Sex}) + \beta_6 (\text{PGS-ADHD}) + \\ & \beta_7 (\text{average individual DM use} \times \text{Time} \times \text{PGS-ADHD}) + \beta_8 (10\text{PCs}) + \\ & \beta_9 (\text{Age at } T_0) + b_{0j} + b_{1j} (\text{Time}) + e_i \end{aligned}$$

b_{0j} and b_{1j} are the random intercept and slope; e_i represents the residual error term.

For moderation analysis following model was used:

For sex:

$$\begin{aligned} \text{ADHD-related symptoms} \sim & \beta_0 + \beta_1 (\text{average individual DM use}) + \beta_2 (\text{Time}) + \beta_3 (\text{average} \\ & \text{individual DM use} \times \text{Time}) + \beta_4 (\text{SES}) + \beta_5 (\text{Sex}) + \beta_6 (\text{PGS-ADHD}) + \\ & \beta_7 (\text{average individual DM use} \times \text{Time} \times \text{PGS-ADHD}) + \beta_8 (10\text{PCs}) + \\ & \beta_9 (\text{average individual DM use} \times \text{Time} \times \text{Sex}) + \\ & \beta_{10} (\text{Age at } T_0) + b_{0j} + b_{1j} (\text{Time}) + e_i \end{aligned}$$

For ADHD diagnosis at T_0 :

$$\begin{aligned} \text{ADHD-related symptoms} \sim & \beta_0 + \beta_1 (\text{average individual DM use}) + \\ & \beta_2 (\text{Time}) + \beta_3 (\text{average individual DM use} \times \text{Time}) + \beta_4 (\text{SES}) + \\ & \beta_5 (\text{Sex}) + \beta_6 (\text{PGS-ADHD}) + \beta_7 (\text{average individual DM use} \times \text{Time} \times \text{PGS-ADHD}) + \\ & \beta_8 (10\text{PCs}) + \beta_9 (\text{average individual DM use} \times \text{Time} \times \text{ADHD diagnosis at } T_0) + \\ & \beta_{10} (\text{Age at } T_0) + b_{0j} + b_{1j} (\text{Time}) + e_i \end{aligned}$$

For ADHD medication status at T_0 :

$$\begin{aligned} \text{ADHD-related symptoms} \sim & \beta_0 + \beta_1 (\text{average individual DM use}) + \beta_2 (\text{Time}) + \\ & \beta_3 (\text{average individual DM use} \times \text{Time}) + \beta_4 (\text{SES}) + \beta_5 (\text{Sex}) + \\ & \beta_6 (\text{PGS-ADHD}) + \beta_7 (\text{average individual DM use} \times \text{Time} \times \text{PGS-ADHD}) + \\ & \beta_8 (10\text{PCs}) + \beta_9 (\text{average individual DM use} \times \text{Time} \times \text{ADHD medication status at } T_0) + \\ & \beta_{10} (\text{Age at } T_0) + b_{0j} + b_{1j} (\text{Time}) + e_i \end{aligned}$$

To investigate the association between DM usage and total change in ADHD-related symptoms ($T_4 - T_0$) to compute the cumulative effect size the following model was used:

$$\begin{aligned} \text{ADHD-related symptoms } (T_4 - T_0) \sim & \beta_0 + \beta_1 (\text{average individual DM use}) + \beta_2 (\text{SES}) + \beta_3 \\ & (\text{Sex}) + \beta_4 (\text{PGS-ADHD}) + \beta_5 (10\text{PCs}) + \beta_6 (\text{Age at } T_0) + e_i \end{aligned}$$

Recursive analysis:

$$\begin{aligned} \text{Social media use} \sim & \beta_0 + \beta_1 (\text{average inattention symptoms}) + \beta_2 (\text{Time}) + \\ & \beta_3 (\text{average inattention symptoms} \times \text{Time}) + \beta_4 (\text{SES}) + \\ & \beta_5 (\text{Sex}) + \beta_6 (\text{PGS-ADHD}) + \beta_7 (\text{average inattention symptoms} \times \text{Time} \times \text{PGS-ADHD}) + \\ & \beta_8 (10\text{PCs}) + \beta_9 (\text{Age at } T_0) + b_{0j} + b_{1j} (\text{Time}) + e_i \end{aligned}$$

An additional test was conducted using a Cross-Lagged Panel Models (lavaan package version 0.6.16 in R), to confirm the directionality of the association between social media use and inattention symptoms using the following model (**Figure 3**):

$$\begin{aligned} S_{t+1} &= \alpha_1 S_t + \beta I_t + \text{covariates} + e_i \\ I_{t+1} &= \delta_1 I_t + \gamma S_t + \text{covariates} + e_i \end{aligned}$$

where S_t is the social media use at time-point t ; I_t is the inattention symptoms at time t ; α_1 and δ_1 are the estimates of autoregressive path, i.e., impact of prior measurement on the next measurement of the same construct; β and γ are the estimate of cross-lagged path between construct; e_i error term. Covariates included in estimate of first timepoint was: age at T_0 visit, sex, SES, PGS-ADHD, and top ten genetic PCs. A more complex model, not constraining β and γ to be fixed across time-points, did not result in a better model fit (evaluated with change in AIC). Including the covariates at each time-point did not change the results. Only children with complete data at all five time points were included.

Supplementary tables

Table S1 Sex-specific effect on the association between digital media use over four years and ADHD-related symptoms in the overall cohort (N=8324)

Symptoms	Social media use	SES	PGS-ADHD	Social media use x Time	Social media use x Time x Sex
Inattention	0.01 (0.03)	-0.14 (0.02)***	0.29 (0.02)***	0.03 (0.01)***	-0.02 (0.01)
	Playing video games	SES	PGS-ADHD	Playing video games x Time	Playing video games x Time x Sex
Hyperactivity-impulsivity	0.41 (0.04)***	-0.22 (0.04)***	0.48 (0.05)***	-0.08 (0.02)***	0.05 (0.03)
	Watching television or videos	SES	PGS-ADHD	Watching television or videos x Time	Watching television or videos x Time x Sex
Hyperactivity-impulsivity	0.48 (0.06)***	-0.17 (0.04)***	0.46 (0.05)***	-0.07 (0.02)***	0.05 (0.02)

Data are presented as standardized beta (standard error). Abbreviations: ADHD, attention-deficit/hyperactivity disorder; SES, socioeconomic status; and PGS, polygenic risk scores. Significance levels are presented as ***<0.001.

Table S2 Effect of ADHD diagnosis status on the association between digital media use over four years and ADHD-related symptoms in the overall cohort (N=8324)

Symptoms	Social media use	SES	PGS-ADHD	Social media use x Time	Social media use x Time x ADHD diagnosis
Inattention	0.09 (0.03) ^{**}	-0.13 (0.02) ^{***}	0.24 (0.02) ^{***}	0.03 (0.01) ^{***}	-0.01 (0.01)
	Playing video games	SES	PGS-ADHD	Playing video games x Time	Playing video games x Time x ADHD diagnosis
Hyperactivity-impulsivity	0.19 (0.06) ^{***}	-0.22 (0.04) ^{***}	0.42 (0.04) ^{***}	-0.07 (0.02) ^{***}	0.05 (0.02)
	Watching television or videos	SES	PGS-ADHD	Watching television or videos x Time	Watching television or videos x Time x ADHD diagnosis
Hyperactivity-impulsivity	0.39 (0.06) ^{***}	-0.17 (0.04) ^{***}	0.40 (0.04) ^{***}	-0.06 (0.02) ^{***}	0.03 (0.02)

Data are presented as standardized beta (standard error). Abbreviations: ADHD, attention-deficit/hyperactivity disorder; SES, socioeconomic status; and PGS, polygenic risk scores. Significance levels are presented as ***<0.001; **<0.01.

Table S3 Effect of ADHD medication status on the association between digital media use over four years and ADHD-related symptoms in the overall cohort (N=8324)

Symptoms	Social media use	SES	PGS-ADHD	Social media use x Time	Social media use x Time x ADHD medication status
Inattention	0.05 (0.02)*	-0.13 (0.02)***	0.28 (0.02)***	0.03 (0.01)***	-0.03 (0.02)
	Playing video games	SES	PGS-ADHD	Playing video games x Time	Playing video games x Time x ADHD medication status
Hyperactivity-impulsivity	0.29 (0.05)***	-0.21 (0.04)***	0.46 (0.05)***	-0.05 (0.01)***	-0.03 (0.04)
	Watching television or videos	SES	PGS-ADHD	Watching television or videos x Time	Watching television or videos x Time x ADHD medication status
Hyperactivity-impulsivity	0.45 (0.04)***	-0.17 (0.04)***	0.44 (0.05)***	-0.05 (0.01)***	0.05 (0.04)

Data are presented as standardized beta (standard error). Abbreviations: ADHD, attention-deficit/hyperactivity disorder; SES, socioeconomic status; and PGS, polygenic risk scores. Significance levels are presented as ***<0.001; *<0.05.

Table S4 Association between social media use and ADHD-related symptoms in children born at term (n=6986)

Symptoms	Social media use	SES	PGS-ADHD	Social media use x Time	Social media use x Time x PGS-ADHD
Inattention	0.06 (0.04)*	-0.14 (0.03)***	0.28 (0.03)***	0.03 (0.01)***	-0.01 (0.01)

Data are presented as standardized beta (standard error). Abbreviations: ADHD, attention-deficit/hyperactivity disorder; SES, socioeconomic status; and PGS, polygenic risk scores. Significance levels are presented as ***<0.001; *<0.05.

Model: *Inattention symptoms* $\sim \beta_0 + \beta_1$ (average social media use) $+ \beta_2$ (Time) $+ \beta_3$ (average social media use x Time) $+ \beta_4$ (SES) $+ \beta_5$ (Sex) $+ \beta_6$ (PGS-ADHD) $+ \beta_7$ (average social media use x Time x PGS-ADHD) $+ \beta_8$ (10PCs) $+ \beta_9$ (Age at T_0) $+ b_{0j} + b_{1j}$ (Time) $+ e_i$

Table S5 Association between social media use and ADHD-related symptoms in children without any neurodevelopmental conditions (n=6631)

Symptoms	Social media use	SES	PGS-ADHD	Social media use x Time	Social media use x Time x PGS-ADHD
Inattention	0.05 (0.02)*	-0.11 (0.02)***	0.19 (0.02)***	0.03 (0.01)***	-0.01 (0.01)

Data are presented as standardized beta (standard error). Abbreviations: ADHD, attention-deficit/hyperactivity disorder; SES, socioeconomic status; and PGS, polygenic risk scores. Significance levels are presented as ***<0.001; *<0.05.

Model: *Inattention symptoms* $\sim \beta_0 + \beta_1$ (average social media use) $+ \beta_2$ (Time) $+ \beta_3$ (average social media use x Time) $+ \beta_4$ (SES) $+ \beta_5$ (Sex) $+ \beta_6$ (PGS-ADHD) $+ \beta_7$ (average social media use x Time x PGS-ADHD) $+ \beta_8$ (10PCs) $+ \beta_9$ (Age at T_0) $+ b_{0j} + b_{1j}$ (Time) $+ e_i$

Table S6 Association between social media use and ADHD-related symptoms in children with behavioural data available across all follow-ups (N=3414)

Symptoms	Social media use	SES	PGS-ADHD	Social media use x Time	Social media use x Time x PGS-ADHD
Inattention	0.05 (0.04)*	-0.12 (0.03)***	0.32 (0.05)***	0.03 (0.01)***	-0.01 (0.01)

Data are presented as standardized beta (standard error). Abbreviations: ADHD, attention-deficit/hyperactivity disorder; SES, socioeconomic status; and PGS, polygenic risk scores. Significance levels are presented as ***<0.001; *<0.05.

Model: *Inattention symptoms* $\sim \beta_0 + \beta_1$ (average social media use) $+ \beta_2$ (Time) $+ \beta_3$ (average social media use x Time) $+ \beta_4$ (SES) $+ \beta_5$ (Sex) $+ \beta_6$ (PGS-ADHD) $+ \beta_7$ (average social media use x Time x PGS-ADHD) $+ \beta_8$ (10PCs) $+ \beta_9$ (Age at T_0) $+ b_{0j} + b_{1j}$ (Time) $+ e_i$

Table S7 Association between social media use and ADHD-related symptoms in children with three years of follow-ups (N=7215)

Symptoms	Social media use	SES	PGS-ADHD	Social media use x Time	Social media use x Time x PGS-ADHD
Inattention	0.05 (0.02)*	-0.14 (0.02)***	0.30 (0.04)***	0.03 (0.01)***	-0.001 (0.01)

Data are presented as standardized beta (standard error). Abbreviations: ADHD, attention-deficit/hyperactivity disorder; SES, socioeconomic status; and PGS, polygenic risk scores. Significance levels are presented as ***<0.001; *<0.05.

Model: *Inattention symptoms* $\sim \beta_0 + \beta_1$ (average social media use) $+ \beta_2$ (Time) $+ \beta_3$ (average social media use x Time) $+ \beta_4$ (SES) $+ \beta_5$ (Sex) $+ \beta_6$ (PGS-ADHD) $+ \beta_7$ (average social media use x Time x PGS-ADHD) $+ \beta_8$ (10PCs) $+ \beta_9$ (Age at T_0) $+ b_{0j} + b_{1j}$ (Time) $+ e_i$

Table S8 Association between average inattention symptoms and social media use in children over four years

Digital media use	Inattention symptoms	SES	PGS-ADHD	Inattention symptoms x Time	Inattention symptoms x Time x PGS
Social media	0.06 (0.01)***	-0.11 (0.02)***	0.01 (0.03)	-0.01 (0.001)***	-0.002 (0.004)

Data are presented as standardized beta (standard error). Abbreviations: ADHD, attention-deficit/hyperactivity disorder; SES, socioeconomic status; and PGS, polygenic risk scores. Significance levels are presented as ***<0.001.

Model: *Social media use* $\sim \beta_0 + \beta_1$ (average inattention symptoms) $+ \beta_2$ (Time) $+ \beta_3$ (average inattention symptoms x Time) $+ \beta_4$ (SES) $+ \beta_5$ (Sex) $+ \beta_6$ (PGS-ADHD) $+ \beta_7$ (average inattention symptoms x Time x PGS-ADHD) $+ \beta_8$ (10PCs) $+ \beta_9$ (Age at T_0) $+ b_{0j} + b_{1j}$ (Time) $+ e_i$

Supplementary Figures

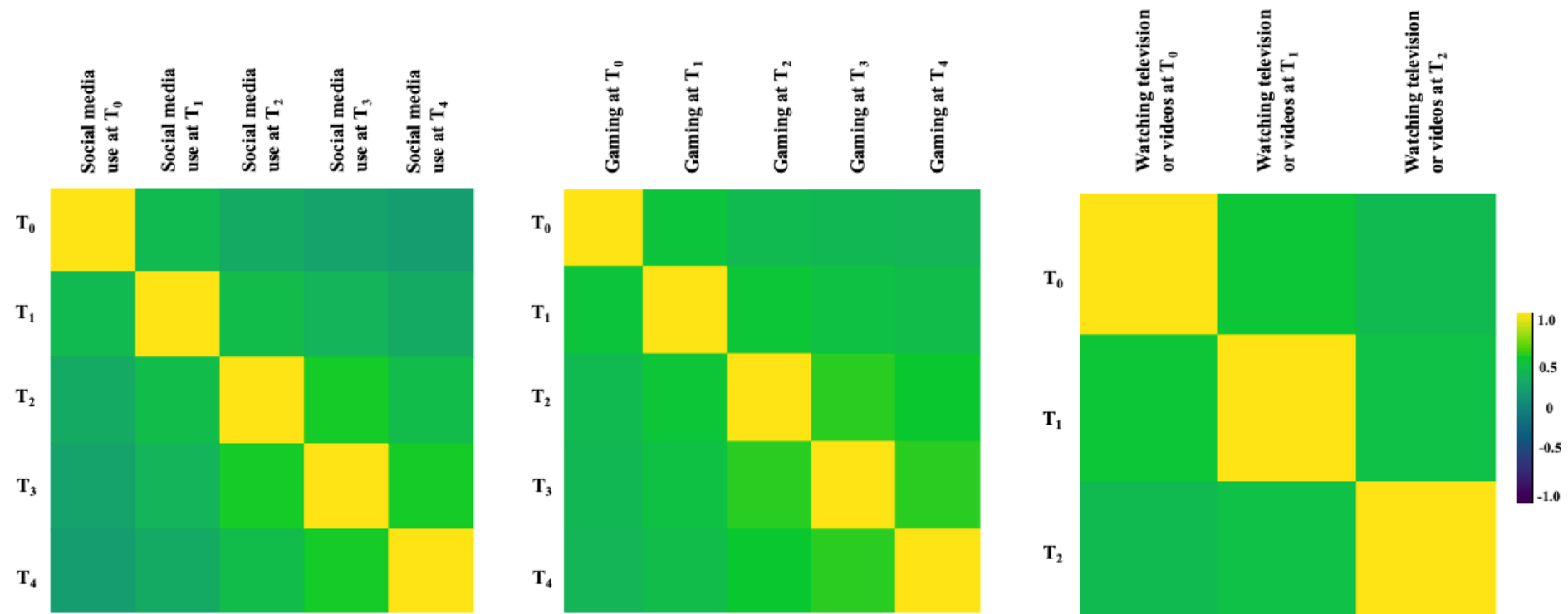


Figure S1 Correlation between individual DM use across four different waves of follow-up

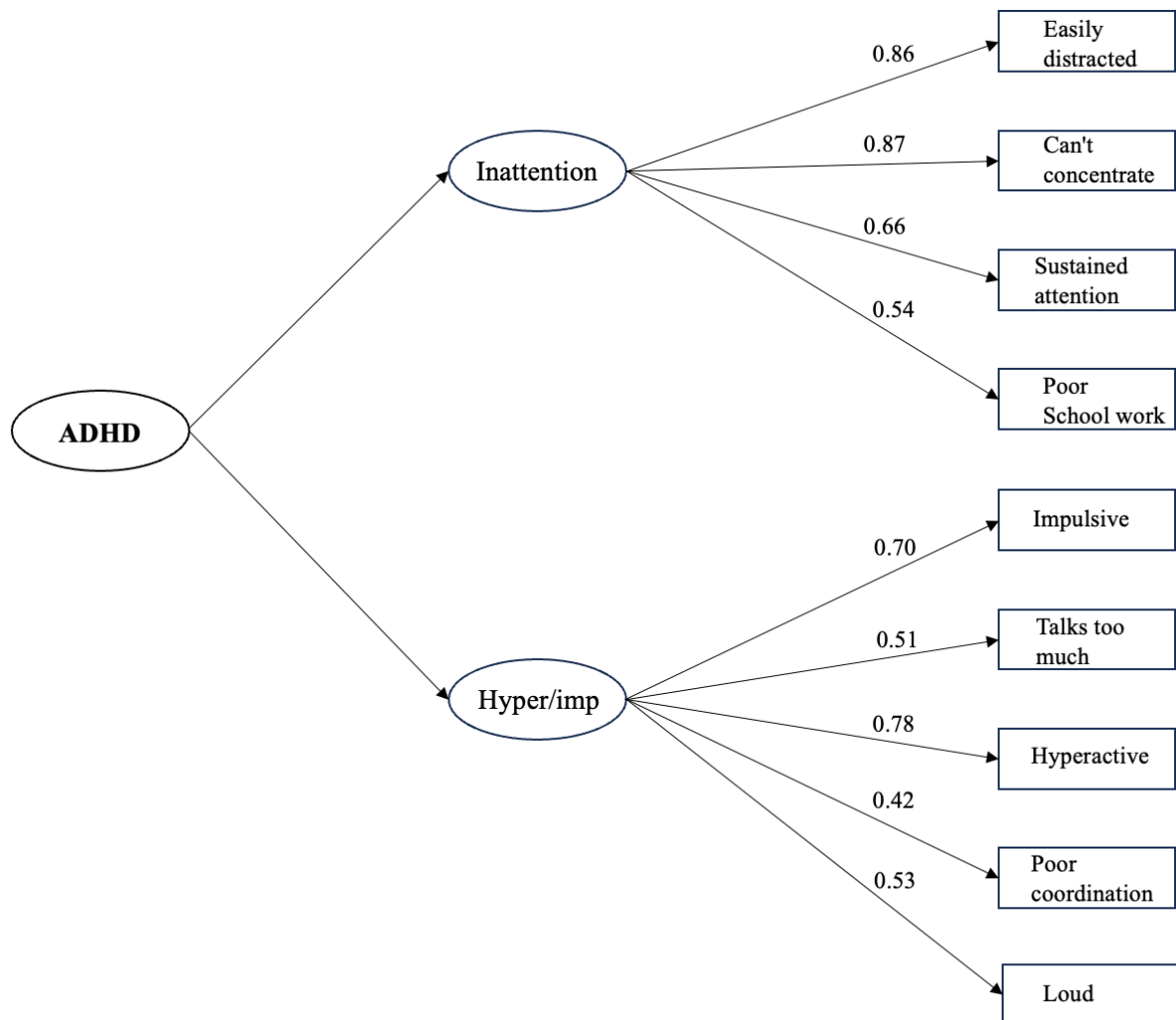


Figure S2 Confirmatory Factor Analysis of ADHD symptoms. Rectangles represent information directly measured (observed), and each rectangle in the model represents an individual item. Circles represent latent variables or unobserved variables that are not directly measured. Factor loadings are shown for observed variables on the latent factors.

References

1. Garavan H, Bartsch H, Conway K, et al. Recruiting the ABCD sample: Design considerations and procedures. *Dev Cogn Neurosci.* Aug 2018;32:16-22. doi:10.1016/j.dcn.2018.04.004
2. Iacono WG, Heath AC, Hewitt JK, et al. The utility of twins in developmental cognitive neuroscience research: How twins strengthen the ABCD research design. *Dev Cogn Neurosci.* Aug 2018;32:30-42. doi:10.1016/j.dcn.2017.09.001
3. Riley AW. Evidence that school-age children can self-report on their health. *Ambulatory Pediatrics.* 2004;4(4):371-376.
4. Nagata JM, Cortez CA, Cattle CJ, et al. Screen Time Use Among US Adolescents During the COVID-19 Pandemic: Findings From the Adolescent Brain Cognitive Development (ABCD) Study. *JAMA Pediatrics.* 2022;176(1):94-96. doi:10.1001/jamapediatrics.2021.4334
5. Fan CC, Marshall A, Smolker H, et al. Adolescent Brain Cognitive Development (ABCD) study Linked External Data (LED): Protocol and practices for geocoding and assignment of environmental data. *Dev Cogn Neurosci.* Dec 2021;52:101030. doi:10.1016/j.dcn.2021.101030
6. Kind AJH, Jencks S, Brock J, et al. Neighborhood socioeconomic disadvantage and 30-day rehospitalization: a retrospective cohort study. *Annals of internal medicine.* 2014;161(11):765-774. doi:10.7326/M13-2946
7. Singh GK. Area deprivation and widening inequalities in US mortality, 1969-1998. *Am J Public Health.* Jul 2003;93(7):1137-43. doi:10.2105/ajph.93.7.1137
8. Cordova MM, Antovich DM, Ryabinin P, et al. Attention-Deficit/Hyperactivity Disorder: Restricted Phenotypes Prevalence, Comorbidity, and Polygenic Risk Sensitivity in the ABCD Baseline Cohort. *J Am Acad Child Adolesc Psychiatry.* Oct 2022;61(10):1273-1284. doi:10.1016/j.jaac.2022.03.030
9. Baurley JW, Edlund CK, Pardamean CI, Conti DV, Bergen AW. Smokescreen: a targeted genotyping array for addiction research. *BMC genomics.* 2016;17(128):145-145. doi:10.1186/s12864-016-2495-7
10. Demontis D, Walters RK, Martin J, et al. Discovery of the first genome-wide significant risk loci for attention deficit/hyperactivity disorder. *Nature genetics.* 2019;51(1):63-75.
11. Howie BN, Donnelly P, Marchini J. A flexible and accurate genotype imputation method for the next generation of genome-wide association studies. *PLoS genetics.* 2009;5(6):e1000529.
12. Delaneau O, Zagury J-F, Marchini J. Improved whole-chromosome phasing for disease and population genetic studies. *Nature methods.* 2013;10(1):5-6.
13. Vilhjálmsdóttir BJ, Yang J, Finucane HK, et al. Modeling linkage disequilibrium increases accuracy of polygenic risk scores. *The american journal of human genetics.* 2015;97(4):576-592.