

Guest Editorial

Genes and screens: attention-deficit hyperactivity disorder in the digital age

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This editorial examines the rise of attention-deficit hyperactivity disorder (ADHD) in the digital age, suggesting that excessive digital media use may mimic or exacerbate ADHD symptoms. We propose examining ADHD through the lens of a spectrum condition, highlighting the importance of considering both genetic and environmental factors in its diagnosis and treatment.

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The diagnosis of ADHD has increased markedly over the past two decades, particularly among adolescents and adults. While social factors such as increased awareness and expanded diagnostic criteria may have contributed, another consideration is changes in capacity to maintain attention. We examine the possibility that excessive digital media exposure, with its frequent attentional shifts and immediate rewards, may mimic or exacerbate ADHD symptoms. Building on established literature regarding the dimensional nature of ADHD, we propose examining ADHD through a spectrum framework with two hypothetical subtypes at the extreme poles: a ‘classical’ neurodevelopmental largely genetically driven form (Type I) and an acquired environmentally influenced form (Type II).

Longitudinal research investigating digital media use and attention difficulties has documented associations between exposure patterns and attention regulation over time, although the directionality of these relationships remains an important area of investigation. A 2018 cohort study of 2587 high-school students who did not initially meet clinical criteria for ADHD found that high levels of digital media use were associated with the emergence of symptoms meeting criteria for ADHD over a subsequent 2-year period.¹ While the study did not control for baseline subthreshold symptoms, limiting causal inference, it provided important evidence for the relationship between digital media use and emerging ADHD symptoms. Indeed, a subsequent large-scale 5-year longitudinal study of nearly 4000 high-school students provided a more nuanced perspective on these relationships.² The study demonstrated that social media engagement was associated with increased impulsivity over time, which subsequently predicted greater ADHD symptoms. These longitudinal findings suggest that digital media use may significantly influence attention regulation and executive function – a relationship that takes on particular significance when examining the association between media multitasking and cognitive control.

Media multitasking shows a relationship with attentional problems, possibly mediated by impact on cognitive control. For example, a landmark study of heavy media multitaskers showed worse ability to filter out distractions during working memory and continuous performance tasks, and greater difficulty with task-switching compared to light media multitaskers.³ These data suggest that the habit of rapidly switching between different media streams may be habituating the brain to expect constant stimulation and reduce the capacity for ‘top-down’, internally driven sustained focus.

There is also a growing body of neurobiological research examining associations between digital media use and impaired

cognitive control. In one functional magnetic resonance imaging (fMRI) study of 149 youths, media multitaskers not only performed worse on a task requiring focused attention in the presence of a distractor, but also displayed increased activity in prefrontal brain regions involved in attentional and inhibitory control, suggesting increase in use of cognitive resources needed to maintain focus.⁴ This concept is supported by a structural magnetic resonance imaging (MRI) study involving 75 participants wherein heavy media multitasking was associated with reduced grey matter density in the anterior cingulate cortex, an area crucial for cognitive control.⁵ Notably, a [¹⁸F]-PET (positron emission tomography) study of 22 healthy adults (ADHD was an exclusion criterion) demonstrated a significant negative correlation between social media app use, as measured by day-to-day logs, and dopamine synthesis capacity in the bilateral putamen.⁶ This is particularly noteworthy given that ADHD is known to be associated with lower dopamine synthesis capacity in this region.

A systematic review examining evidence from 28 neuroimaging studies (nine structural MRI, six resting state fMRI and 13 task-based fMRI) identified several consistent patterns. Social media overuse is associated with the following:

- smaller volumes in limbic system structures, including the ventral striatum, amygdala, subgenual anterior cingulate and orbitofrontal cortex;
- increased ventral striatum activity in response to social media cues such as viewing own photos that had received many ‘likes’; and
- functional connectivity changes involving the dorsal attention network, which is involved in top-down attention processes.⁷

These findings are consistent with those of a scoping review of neuroimaging correlates of screentime in adolescents that summarised 16 studies published between 2010 and 2020.⁸ The authors found that frequent and longer duration of screentime was associated with reduced efficiency of cognitive control processes, including via impacts on the default mode network and central executive network. They noted that ‘online activities act as strong rewards to the brain and repeated screen time augments the tendency to seek short-term gratifications’.⁸

These neuroimaging findings inform our understanding of ADHD in the digital age. The observed patterns in brain structure and function associated with digital media use parallel those seen in ADHD, suggesting possible interactions between digital media use and attention regulation. Of particular interest is the impact on the dopaminergic system, as evidenced by the PET study showing

reduced dopamine synthesis capacity with increased social media use. This aligns with the dopamine deficit hypothesis of ADHD and raises questions about whether chronic digital media exposure could lead to a form of acquired dopamine dysregulation. These changes in the dopaminergic system are just one aspect of broader neurobiological alterations seen in digital media users. For example, the alterations in limbic system structures and functional connectivity changes in attention networks highlight the potential for digital media to reshape neural circuits involved in attention, reward processing and executive function.

Thus, multiple converging lines of evidence, including epidemiological,^{1,2} experimental³ and neurobiological,^{4–8} point to a relationship between digital media use and cognitive control deficits manifesting as ADHD. Recent research has demonstrated that while ADHD prevalence has increased, the relative contribution of genetic and environmental factors has remained stable over time. This stability in genetic influence alongside rising prevalence suggests that changing environmental contexts – particularly our increasingly digital environment – may be interacting with genetic vulnerability to influence symptom expression and presentation.


These findings highlight that symptoms of ADHD may arise from varying aetiologies influenced by both genetic and environmental factors, and support our examination of ADHD through a spectrum framework with two presentations at the extreme poles. Similar to how Type I and Type II diabetes present with similar clinical features despite distinct underlying mechanisms, these ADHD presentations may manifest with comparable symptoms while arising from different pathways. Type I ADHD, a ‘classical’ neurodevelopmental, largely genetically driven form, may be characterised by early onset, robust response to stimulant medications, high heritability and lower prevalence. Type II ADHD, an acquired, environmentally influenced form, may be characterised by later symptom onset and driven by environmental factors, including excessive exposure to digital media – a particularly salient factor given the increasing pervasiveness of digital media in society. Support for Type II ADHD comes from longitudinal studies examining temporal associations between digital media use and attention difficulties, complemented by neuroimaging findings that show parallel patterns in dopamine synthesis, limbic system structure and attention network connectivity between heavy digital media users and individuals with ADHD.

It is necessary, however, to acknowledge the spectrum of multifactorial ADHD between these two poles. Digital media exposure may not only be a key factor in Type II ADHD but may also exacerbate symptoms in genetically susceptible individuals, potentially leading to more severe or treatment-resistant presentations.

This spectrum model underscores the need for personalised treatment strategies. Concerns have arisen about increased stimulant prescriptions; for example, the use of most behavioural health medications in the USA remained relatively unchanged during the COVID-19 pandemic, while the use of ADHD stimulant medications increased significantly.⁹ How much of this was transient and related to stress and excessive digital media use? Furthermore, the long-term effectiveness and appropriateness of stimulant medications might be expected to vary significantly in patients falling along different points of the proposed spectrum. For individuals with Type II ADHD, where environmental factors such as excessive digital media use play a more significant role, psychotherapeutic and behavioural interventions – such as cognitive-behavioural therapy or digital media reduction strategies –

may prove more effective than stimulants in addressing the underlying causes of attentional difficulties. These variations could help explain the inconsistent literature on stimulant ‘drug holidays’, with some studies suggesting benefits from taking breaks, while others indicate the opposite.

In conclusion, our examination suggests excessive digital media exposure may, in addition to social factors, play a role in the significant rise in ADHD diagnoses in recent years. Building on the established understanding of ADHD’s dimensional structure, we propose a framework that examines how contemporary environmental factors, particularly digital media exposure, may interact with genetic vulnerability along this continuum. This framework emphasises the role of digital media exposure in Type II ADHD and its potential to exacerbate symptoms of ‘classical’ Type I ADHD. While compelling evidence links digital media use to attention difficulties, rigorous empirical testing will be crucial to refine, validate or refute specific aspects of this framework and determine its clinical utility. The ultimate value of this perspective lies in its ability to generate testable hypotheses that can advance our understanding of how modern environmental factors influence attention regulation and ADHD expression, potentially leading to more effective, personalised interventions for individuals presenting with ADHD symptoms in our increasingly digital world.

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Declaration of interest

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