Towards pathophysiology-based interventions for children with ADHD and increased screen time utilisation

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Attention-Deficit/Hyperactivity Disorder (ADHD) is the most common neurodevelopmental condition, affecting around 5-7% of school-aged children. Compared to individuals without ADHD, those with ADHD are at higher risk of mental health disorders (such as anxiety or depression), physical conditions (including obesity and asthma), and maladaptive/risky behaviours (e.g., unintentional physical injuries or suicidal behaviours). An emerging body of evidence suggests that ADHD is also significantly associated with increased screen time utilisation (STU), which can add to the burden of ADHD. However, the relationship between ADHD PRS and STU is a matter of debate. The study by Yang et al. furthers our understanding of the relationship between ADHD symptoms and STU, paving the way for possible pathophysiology-based interventions. The authors focused on polygenic risk scores (PRS), i.e., the sum of the effects of individual genes that provides an overall estimate of genetic lability for a given disorder. Using a multimodal approach with a cross-sectional and longitudinal approach, the authors were able to identify the link between ADHD and STU in individuals with ADHD. The study by Yang et al. furthers our understanding of the relationship between ADHD symptoms and STU, paving the way for possible pathophysiology-based interventions. The authors focused on polygenic risk scores (PRS), i.e., the sum of the effects of individual genes that provides an overall estimate of genetic lability for a given disorder. Using a multimodal approach with a cross-sectional and longitudinal approach, the authors were able to build and test a comprehensive, coherent model linking ADHD and increased STU, spanning from genes to behaviour via brain measures (estimated white matter integrity). In turn, this points to possible intervention strategies. Indeed, there may be several reasons underlying the link between ADHD and STU. For instance, increased STU might stem from the behavioural impulsivity of individuals with ADHD, or increased STU could contribute ADHD symptoms, in particular inattention. However, the study by Yang et al. suggests that both ADHD and long STU share common genetic underpinning and are characterised by possible dysfunctions in white matter tracts related to visual functions. This may result in impaired executive control of visual functions, with enhanced sensitivity to and distractibility by visual stimuli leading to increased STU. If replicated in other studies, the findings by Yang et al. would provide a compelling rationale to assess interventions aimed at restoring or improving these impaired visual functions. To date, the paradigm “gene dysfunction
leading to brain imaging alterations, in turn underpinning behavioural manifestations” has not led to relevant innovations in the care of children with ADHD. For instance, meta-analytic evidence7 based on trials of cognitive training, aimed at changing the functioning of brain areas thought to mediate the relationship between genes and behaviour, has not supported the efficacy of this intervention in terms of improvement of ADHD core symptoms. This is unfortunate because, whilst stimulants have been shown to be highly efficacious - at least in the short term for the core symptoms of ADHD8, their effects in terms of improving associated neuropsychological dysfunctions have been found to be less strong9. One possible reason is that individuals with ADHD included in such trials were highly heterogeneous in terms of underlying pathophysiological correlates. By adopting a precision psychiatry approach10 and selecting more homogeneous subgroups, such as children with high levels of ADHD symptoms and STU, the field might be more successful in designing and implementing novel pathophysiology-based interventions.

Declaration of interests
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