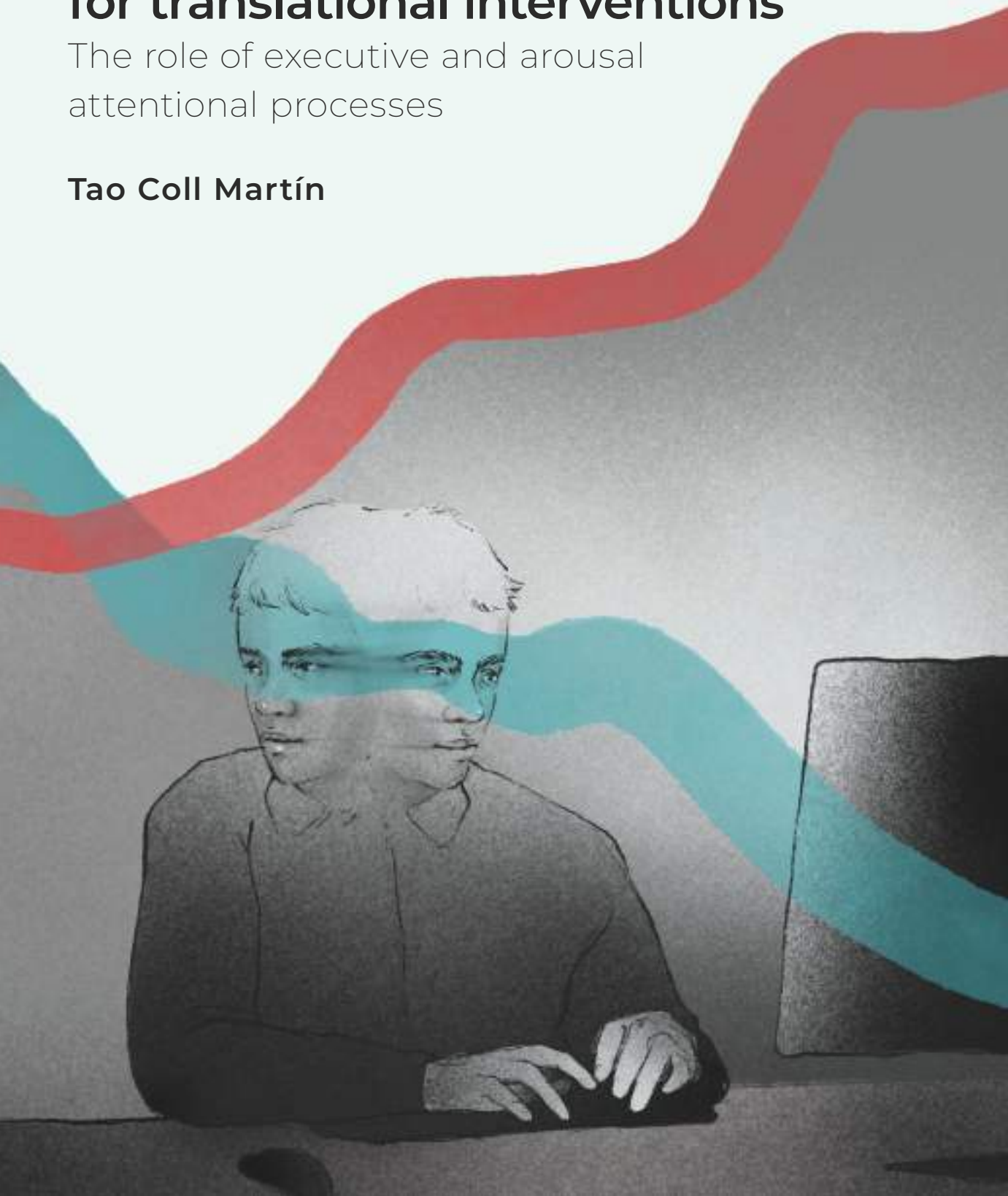
Dimensional perspective in ADHD neurodevelopment as a foundation for translational interventions

The role of executive and arousal attentional processes

Tao Coll Martín



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Dimensional perspective in ADHD neurodevelopment as a foundation for translational interventions:

The role of executive and arousal attentional processes

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A mi Mamaconcha,

¡qué felices somos!

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ABSTRACTS

Abstract

Attention-deficit/hyperactivity disorder (ADHD) is commonly conceptualized as a neurodevelopmental condition characterized by age-inappropriate and impairing levels of inattention and/or hyperactivity–impulsivity. After decades of prolific basic research into the causes and mechanisms underlying the disorder, these findings have failed to translate into interventions with a substantial impact on core ADHD symptoms. To move past this situation, a shift from the medical to the biopsychosocial model of the disorder is crucial. Based on empirical evidence, the biopsychosocial model adopts a dimensional perspective of ADHD as well as other more complex yet less restrictive assumptions than the medical one, holding considerable hope for translational progress.

As part of the dimensional perspective, a fundamental step is the integration between theories aimed at explaining the general neurocognitive functioning and those that account for its variation as a function of ADHD symptom severity. Indeed, the potential alterations in ADHD should be nested within well-established neurocognitive models. In this dissertation, we focused on attention—understood as a general modulatory system of cognition—as a central axis for characterizing two key and dissociable mechanisms in both typical cognitive functioning and ADHD theories: executive and arousal processes. Each of these two domains constitutes an independent mechanism in models of the general functioning of attentional networks (M. I. Posner & Petersen, 1990) and vigilance (Luna et al., 2018). Furthermore, the main theories of ADHD are distinguished between those based on executive dysfunction (Barkley, 1997) and those focused on arousal dysregulation (Sergeant, 2000, 2005). Crucial for the recent debate on the nature of late-onset ADHD, Halperin and Schulz's (2006) neurodevelopmental model proposes a dissociation between arousal and executive mechanisms when accounting for the early onset of ADHD versus its later development.

Based on this dimensional perspective of ADHD within the broader biopsychosocial framework, the aim of the present dissertation was to deepen our understanding of the executive and arousal attentional alterations underlying ADHD symptoms across development, ultimately considering implications for translational interventions. The achievement of this general objective was carried out in five studies grouped into three chapters plus an overarching conceptual analysis that discusses the translational contributions of our findings.

First, we sought to establish a neurocognitive behavioural task capable of (a) feasibly collecting large samples from different contexts, (b) measuring relevant indices of attentional functioning with sufficient reliability, and (c) differentiating between executive and arousal measures. To do so, we conducted an instrumental study focused on the Attentional Networks Test for Interaction and Vigilance—Executive and Arousal Components (ANTI-Vea; Luna et al., 2018). Based on the theoretical models of attention mentioned above, this task assesses the functioning of the three attentional networks (alerting, orienting, and executive control) and two vigilance components (executive and arousal). In this study, we developed a free and online resource to easily run, collect, and analyze large volumes of data from the ANTI-Vea, both in typical lab conditions and online in remote settings: the ANTI-Vea-UGR platform. Consistent with open science principles, the task versions are open source, anonymized ANTI-Vea online participant data is stored on a freely accessible public server, and task analyses can be performed using a custom Shiny app or with its associated R code. In addition, we undertook a narrative review of the more than a dozen studies, encompassing over a thousand participants in total, that have employed ANTI-Vea. We concluded that the reliability of most ANTI-Vea indices was acceptable when the task was used in large samples. Crucially, executive and arousal processes were empirically dissociated using the ANTI-Vea, both through experimental manipulations and within the association pattern of some attention-related constructs.

Second, we conducted an empirical series of three associative studies, all following a very similar procedure: A community sample of university students perform the ANTI-Vea and complete self-reports on the severity of their ADHD symptoms in childhood (retrospectively assessed) and adulthood. The first of these studies (N = 113), of a more exploratory nature, found a neurodevelopmental dissociation consistent with Halperin and Schulz's (2006) neurodevelopmental model. Concretely, arousal indices (i.e., alerting network and arousal vigilance) correlated with ADHD symptoms in childhood, whereas the decrement in executive vigilance was linked to higher symptoms in adulthood. The second study (N = 292) was a preregistered close replication of the previous one with a greater focus on transparently testing the hypothesized neurodevelopmental dissociation between executive and arousal vigilance in relation to ADHD symptoms. Unexpectedly, neither preregistered nor multiverse analyses supported the predictions of the neurodevelopmental model. The third study (N = 492) combined the samples from the two previous studies with a third sample in which participants performed the ANTI-Vea task multiple times to shed light on the neurocognitive characterization of late-onset ADHD. This final pattern of results did not support a neurodevelopmental dissociation either. On the contrary, both executive and arousal alterations were associated with ADHD symptoms in childhood, adulthood, and late onset (i.e., symptoms in adulthood after controlling for those in childhood).

Third, we presented the protocol of a systematic review and meta-analysis of the effects of nonpharmacological interventions for ADHD on indices of autonomic arousal. Arousal dysregulation in ADHD, often in the form of hypo-arousal, is a potential target and mediator in interventions aimed at improving ADHD symptoms. Arousal measures of the autonomic nervous system include cardiac, electrodermal, and pupil activity, among others. The twofold aim of this study was (a) to examine whether current nonpharmacological interventions for ADHD, translational or not, can enhance the

regulation of arousal mechanisms; and (b) to identify promising arousal-based translational interventions for ADHD. Preliminary results have identified 12 studies. However, due to the low statistical power and high heterogeneity across study designs and intervention types, drawing robust conclusions on the current state of the art in this area was challenging.

Finally, we attempted to integrate our findings with the previous literature to derive proposals that may contribute to the future of translational interventions for ADHD in two key issues:

1. Neurocognitive nature of late-onset ADHD. Evidence mostly suggests that late-onset ADHD is not distinct from conventional ADHD at the neurocognitive level. This implies that translational interventions for ADHD should target the same underlying alterations across different stages of development, regardless of the age of disorder onset. Furthermore, this supports the idea that both child- and adult-onset ADHD can be conceptualized as mere variants of the same disorder. Whether this single disorder is neurodevelopmental or not is an open question that depends on the role (moderating vs. causal) of late-onset ADHD precursors.
2. Mechanisms of translational interventions. As we have shown, both executive and arousal alterations may underlie ADHD symptoms. However, interventions for ADHD have typically been designed to train executive domains (e.g., computer-based cognitive training), while targeting arousal regulation is generally neglected. This contrasts to current models of cognitive training, which propose that the transfer of gains is largely due to mechanisms of cognitive efficiency, which are not primarily executive. Therefore, arousal-based translational interventions for ADHD, such as effort training through learned industriousness or trigeminal nerve stimulation, are postulated as promising therapeutic options.

Overall, although the therapeutic progress derived from ADHD basic research is limited, we believe that the translational logic of targeting neurocognitive processes thought to mediate ADHD pathophysiology to improve core symptoms and related impairment still has viability. While the contributions of this thesis are tentative and somewhat constrained by the design of studies, our adoption of the biopsychosocial/dimensional model and the open science framework is, in our view, essential to building a more sound and translatable ADHD science. Beyond translational interventions, promoting inclusive environments of acceptance and appreciation towards the diverse ways of thinking and behaving of people with ADHD is fundamental to the development of their personal growth, strengths, and empowerment in our society.

Resumen

El trastorno por déficit de atención e hiperactividad (TDAH) se conceptualiza comúnmente como una condición del neurodesarrollo caracterizada por unos niveles inapropiados para la edad y deteriorantes de falta de atención y/o hiperactividad–impulsividad. Tras décadas de prolífica investigación básica en las causas y mecanismos subyacentes al trastorno, estos hallazgos no han logrado trasladarse a intervenciones que tengan un impacto sustancial en los síntomas centrales del TDAH. Para superar esta situación, un cambio del modelo médico al modelo biopsicosocial del trastorno es crucial. Basado en evidencia empírica, el modelo biopsicosocial adopta una perspectiva dimensional del TDAH, además de otras suposiciones más complejas pero menos restrictivas que el médico, ofreciendo así esperanzas considerables para el progreso traslacional.

Como parte de la perspectiva dimensional, un paso fundamental es la integración entre teorías dirigidas a explicar el funcionamiento neurocognitivo general y aquellas que explican su variación en función de la severidad de los síntomas del TDAH. De hecho, las potenciales alteraciones en el TDAH deben anidarse dentro de modelos neurocognitivos bien establecidos. En esta disertación, nos centramos en la atención, entendida como un sistema modulador general de la cognición, como eje central para caracterizar dos mecanismos clave y disociables tanto en el funcionamiento cognitivo típico como en las teorías del TDAH: los procesos ejecutivos y de activación (también conocido como *arousal*). Cada uno de estos dos dominios constituye un mecanismo independiente en los modelos del funcionamiento general de las redes atencionales (M. I. Posner & Petersen, 1990) y vigilancia (Luna et al., 2018). Además, las principales teorías del TDAH se distinguen entre las basadas en disfunción ejecutiva Barkley (1997) y las enfocadas en la disregulación de la activación (Sergeant, 2000, 2005). Clave para

el reciente debate sobre la naturaleza del TDAH de inicio tardío, el modelo del neurodesarrollo de Halperin y Schulz (2006) propone una disociación entre mecanismos de activación y ejecutivos al explicar el inicio temprano del TDAH frente a su desarrollo posterior.

Basándonos en esta perspectiva dimensional del TDAH dentro del marco biopsicosocial más amplio, el objetivo de la presente disertación era profundizar en nuestra comprensión de las alteraciones atencionales ejecutivas y de activación subyacentes a los síntomas del TDAH a lo largo del desarrollo, considerando en última instancia sus implicaciones para las intervenciones traslacionales. La consecución de este objetivo general se llevó a cabo en cinco estudios agrupados en tres capítulos más un análisis conceptual general que discute las contribuciones traslacionales de nuestros hallazgos.

En primer lugar, buscamos establecer una tarea neurocognitivo comportamental capaz de (a) recolectar muestras grandes de manera factible en diferentes contextos, (b) medir índices relevantes del funcionamiento atencional con suficiente fiabilidad y (c) diferenciar entre procesos ejecutivos y de activación. Para ello, realizamos un estudio instrumental enfocado en el Test de Redes Atencionales para Interacciones y Vigilancia—Componentes Ejecutivos y de Activación (ANTI-Vea; Luna et al., 2018). Basada en los modelos teóricos de atención mencionados anteriormente, esta tarea evalúa el funcionamiento de las tres redes atencionales (alerta, orientación y control ejecutivo) y dos componentes de vigilancia (ejecutiva y de activación). En este estudio, desarrollamos un recurso gratuito en línea para ejecutar, recopilar y analizar fácilmente grandes volúmenes de datos de la ANTI-Vea, ya sea en condiciones típicas de laboratorio o en línea en entornos remotos: la plataforma ANTI-Vea-UGR. En consonancia con los principios de la ciencia abierta, las versiones de la tarea son de código abierto, los datos en línea anonimizados de los participantes de la ANTI-Vea se almacenan en un servidor

público de libre acceso y el análisis de la tarea se puede realizar mediante una aplicación Shiny personalizada o con su código R asociado. Además, realizamos una revisión narrativa de más de una docena de estudios, incluyendo más de un millar de participantes en total, que han empleado la ANTI-Vea. Concluimos que la fiabilidad de la mayoría de los índices de la ANTI-Vea fue aceptable cuando se utilizaba en muestras grandes. Crucialmente, los procesos ejecutivos y de activación fueron empíricamente disociados en la ANTI-Vea, tanto mediante manipulaciones experimentales como en el patrón de asociaciones de algunos constructos relacionados con la atención.

En segundo lugar, llevamos a cabo una serie empírica de tres estudios asociativos, todos siguiendo un procedimiento muy similar: Una muestra comunitaria de estudiantes universitarios realiza la ANTI-Vea y completa autoinformes sobre la severidad de sus síntomas del TDAH en la infancia (evaluados retrospectivamente) y en la adultez. El primero de estos estudios (N = 113), de naturaleza más exploratoria, encontró una disociación en el neurodesarrollo consistente con el modelo del neurodesarrollo de Halperin y Schulz (2006). Concretamente, los índices de activación (es decir, la red de alerta y la vigilancia de activación) correlacionaron con los síntomas del TDAH en la infancia, mientras que el decremento en la vigilancia ejecutiva se vinculó con niveles de síntomas más altos en la adultez. El segundo estudio (N = 292) es una réplica cercana preregistrada del anterior con un mayor foco en testar de manera transparente una hipotetizada disociación en el neurodesarrollo entre la vigilancia ejecutiva y de activación en relación con los síntomas del TDAH. Inesperadamente, ni los análisis preregistrados ni los análisis multiverso apoyaron las predicciones del modelo del neurodesarrollo. El tercer estudio (N = 492) combina las muestras de los dos estudios anteriores con una tercera muestra en la que los participantes realizaron la tarea ANTI-Vea en múltiples ocasiones para arrojar luz sobre la caracterización neurocognitiva del TDAH de inicio tardío. Este patrón final de resultados tampoco respaldó una disociación en el

neurodesarrollo. Por el contrario, tanto las alteraciones ejecutivas como de activación se asociaron con los síntomas del TDAH en la infancia, en la adultez y de inicio tardío (i. e., síntomas en la adultez después de controlar por los síntomas de la infancia).

En tercer lugar, presentamos el protocolo de una revisión sistemática y un metanálisis sobre los efectos de las intervenciones no farmacológicas para el TDAH en los índices de activación autonómica. La disregulación de la activación en el TDAH, a menudo en forma de hipoactivación, es un posible objetivo y mediador en las intervenciones dirigidas a mejorar los síntomas del TDAH. Las medidas de activación del sistema nervioso autónomo incluyen las actividades cardíaca, electrodérmica y pupilar, entre otras. El doble objetivo de este estudio fue (a) examinar si las intervenciones actuales para el TDAH, traslacionales o no, pueden mejorar la regulación de los mecanismos de activación; y (b) identificar intervenciones traslacionales prometedoras basadas en la activación para el TDAH. Los resultados preliminares han identificado 12 estudios. Sin embargo, dada la baja potencia estadística y la alta heterogeneidad entre los diseños de los estudios y entre los tipos de intervención, fue difícil extraer conclusiones sólidas sobre el estado del arte actual en esta área.

Por último, intentamos integrar nuestros hallazgos con la literatura previa para derivar propuestas que puedan contribuir al futuro de las intervenciones traslacionales para el TDAH en dos cuestiones clave:

1. Naturaleza neurocognitiva del TDAH de inicio tardío. La evidencia sugiere que el TDAH de inicio tardío no es distinto del TDAH convencional a nivel neurocognitivo. Esto implica que las intervenciones traslacionales para el TDAH deben dirigirse a las mismas alteraciones subyacentes en las diferentes etapas del desarrollo, independientemente de la edad de inicio del trastorno. Además, esto apoya la idea de que tanto el TDAH infantil como el de inicio tardío pueden conceptualizarse como meras variantes del mismo trastorno. Si este trastorno único es o no del

neurodesarrollo es una cuestión abierta que depende del rol (moderador versus causal) de los precursores del TDAH de inicio tardío.

2. Mecanismos de las intervenciones traslacionales. Como hemos demostrado, tanto las alteraciones ejecutivas como las de la activación pueden subyacer a los síntomas del TDAH. Sin embargo, las intervenciones para el TDAH han sido típicamente diseñadas para entrenar los dominios ejecutivos (p. ej., el entrenamiento cognitivo computarizado), mientras que enfocarse en la regulación de la activación generalmente ha sido ignorado. Esto contrasta con modelos actuales de entrenamiento cognitivo, que proponen que la transferencia de ganancias se debe en gran medida a mecanismos de eficiencia cognitiva, que no son primariamente ejecutivos. Por tanto, las intervenciones traslacionales para el TDAH basadas en la activación, como el entrenamiento del esfuerzo mediante la laboriosidad aprendida o la estimulación del nervio trigémino, se postulan como opciones terapéuticas prometedoras.

En general, a pesar de que el progreso terapéutico derivado de la investigación básica del TDAH es limitado, creemos que la lógica traslacional de dirigirse a los procesos neurocognitivos que supuestamente median en la fisiopatología del TDAH para mejorar los síntomas centrales y el deterioro relacionado aún tiene viabilidad. Aunque las contribuciones de esta tesis son tentativas y algo constreñidas por el diseño de sus estudios, nuestra adopción del modelo biopsicosocial/dimensional y el marco de la ciencia abierta son, en nuestra opinión, esenciales para construir una ciencia del TDAH más sólida y prometedora. Más allá de las intervenciones traslacionales, promover entornos inclusivos de aceptación y valoración hacia las diversas formas de pensar y comportarse en las personas con TDAH es fundamental para el desarrollo de su crecimiento personal, sus fortalezas y su empoderamiento en nuestra sociedad.

CHAPTER I: INTRODUCTION

ADHD: A disorder in the search for translational interventions

ADHD overview and impact

Attention-deficit/hyperactivity disorder (*ADHD*) is commonly conceptualized as a neurodevelopmental condition characterized by developmentally inappropriate levels of inattention and/or hyperactivity–impulsivity. According to the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association [APA], 2013), for the clinical diagnosis of ADHD, symptoms must be (a) numerous, meeting at least six (five from 17 years of age onwards) out of nine symptoms within at least one of the two clusters (i.e., inattention and hyperactivity–impulsivity) that determine the type of presentation; (b) persistent, lasting at least six months; (c) generalized, present in at least two contexts (e.g., home and school); (d) child-onset, with at least several symptoms present prior to age 12 years; (e) primary, not better explained by another diagnosable mental disorder, rule-breaking behaviours (e.g., oppositionality, defiance, or hostility), or failure to understand instructions; and (f) interfering, affecting the individual's functional performance. When made by a licensed clinician who interviews the parent or caregiver and/or patient, ADHD is considered a well-defined and valid diagnosis (Faraone et al., 2021).

While it can be initiated in the preschool years, ADHD is often diagnosed in school-aged children and remains relatively stable through early adolescence (APA, 2013). In adolescence and young adulthood, many individuals with childhood ADHD continue to be affected by the disorder, although they often show reduced hyperactivity and impulsivity (Faraone, Biederman, & Mick, 2006). The disorder is estimated to be present in about 5%

of children and adolescents (Polanczyk et al., 2007), a percentage that does not seem to have varied in recent decades or by country (Polanczyk et al., 2014), and 2.5% of adults (Simon et al., 2009). In contrast to these epidemiological studies, the percentage of people who have received a diagnosis of ADHD has increased in recent years, due to changes in administrative and clinical practices (Rydell et al., 2018; Song et al., 2019; Xu et al., 2018). ADHD is approximately twice as common in boys as in girls, a difference that becomes more balanced with age (Faheem et al., 2022; Franke et al., 2018; Willcutt, 2012).

ADHD is associated with multiple clinical conditions. To begin with, ADHD often co-occurs with other mental disorders, especially autism spectrum disorders, oppositional defiant disorder, conduct disorder, substance use disorders, anxiety disorders, depression, bipolar disorder, and eating disorders (Chen et al., 2018; Franke et al., 2018; Nazar et al., 2016). Although part of this comorbidity is due to shared genetic variance (Gidziela et al., 2023), the environment frequently evoked by ADHD symptoms (e.g., negative parenting, peer rejection, academic problems) can lead to the development of secondary disorders across the lifespan (Hinshaw, 2018; Nigg et al., 2020; Sonuga-Barke et al., 2023). These relationships may be influenced by emotion dysregulation and cognitive disengagement syndrome (previously referred to as sluggish cognitive tempo), two psychopathological dimensions closely linked to ADHD (Becker et al., 2023; Soler-Gutiérrez et al., 2023). Regarding medical problems, individuals with ADHD are at increased risk for obesity, asthma, allergies, diabetes mellitus, hypertension, sleep problems, psoriasis, epilepsy, sexually transmitted infections, abnormalities of the eye, immune disorders, and metabolic disorders (Faraone et al., 2021). Ultimately, life quality is lower in children and adults with ADHD (Jonsson et al., 2017; Lensing et al., 2015; Quintero et al., 2019).

The impact of living with ADHD has been linked to many severe adverse outcomes. In this sense, people with the disorder have about a 50% greater risk of unintentional physical injuries and serious transport accidents (Z. Chang et al., 2014; Ruiz-Goikoetxea et al., 2018). In school, it has been found that U.S. students with ADHD were 2.8 times more likely to engage in bullying and twice as likely to not have graduated from high school on time (Benedict et al., 2015; Breslau et al., 2011). In adolescence and adulthood, those with ADHD have been found 40% more likely to be unemployed and 70% more likely to have been incarcerated (Fleming et al., 2017; Mohr-Jensen et al., 2019). Finally, meta-analyses show that the death rate from unnatural causes in people with ADHD is about three times higher than in the general population, and the rate of suicide is six times higher (Catalá-López et al., 2022; Septier et al., 2019). Of note, the persistence of ADHD into adulthood was linked to a 12.7-year reduction in estimated life expectancy (Barkley & Fischer, 2019).

Beyond the person with ADHD, the consequences of suffering from this disorder extend to the surrounding environment and, ultimately, to society. In this sense, substantially lower well-being has been found in parents and siblings of people with ADHD, which were independent of their own ADHD symptoms (Peasgood et al., 2016, 2021). At school, ADHD children are more stressful to teach (Greene et al., 2002). In adulthood, marital and family adjustment showed impairment when one member of the couple has ADHD (Eakin et al., 2004). Regarding community, a systematic review of the global economic burden of ADHD found that the additional costs attributable to ADHD ranged between €220 and €16,880 per person and year (Chhibber et al., 2021).

Taken together, ADHD is an impairing condition that affects a substantial percentage of men and women of all ages worldwide. Individuals with ADHD experience a reduced quality of life and an elevated risk of comorbidities with other disorders, medical issues, and adverse life events and situations. The impact of ADHD also extends to their family

and the broader community. Against this backdrop, developing effective interventions for ADHD is a primordial mission of our society.

First-line interventions for ADHD

Pharmacological interventions are typically considered the first-line treatment for children and adults with ADHD (Faraone et al., 2015; National Institute for Health and Care Excellence [NICE], 2019; J. Posner et al., 2020; Wolraich et al., 2019). They are categorized into psychostimulants (i.e., methylphenidate and amphetamine) and non-stimulants (i.e., atomoxetine, guanfacine, and clonidine), the former usually being the first choice. Compared to placebo, medication is efficacious in reducing ADHD symptoms in the short term, with large effects in children and moderate to large effects in adults (Cortese et al., 2018). However, the more general clinical value of medication has been questioned. In fact, adherence is generally low, partly due to side effects (Charach & Fernandez, 2013; Cortese et al., 2018). Moreover, normalization or long-term effectiveness of medication on functional outcomes is rather limited (Shaw et al., 2012).

The main alternative to pharmacological treatment is behavioural intervention. This is the first-line treatment for preschoolers with ADHD and the most recommended complement to pharmacological treatment in childhood and adulthood (NICE, 2019; Wolraich et al., 2019). This type of therapy is based on learning principles of contingency management to target undesirable behaviours in people with ADHD, along with increasing desirable ones (Evans et al., 2018). In the case of preschool or school-age children, this intervention is typically delivered indirectly, through parents (behavioural parent training) or teachers (behavioural classroom management). Mostly in adolescence and adulthood, behavioural interventions incorporate components of cognitive restructuring, problem-solving and organization skill training, thus receiving the designation of cognitive behavioural therapy (Young et al., 2020).

Despite the well-established status of behavioural intervention in the treatment of ADHD, its efficacy for the core symptoms of the disorder (i.e., inattention and hyperactivity–impulsivity) has been called into question. In this sense, an influential meta-analysis (Sonuga-Barke, 2013) of this therapy for children and adolescents found no effect on ADHD symptoms when the assessment was based on raters probably blinded to the treatment allocation (e.g., teacher rating for home-based interventions). Similarly, a meta-analysis of behavioural interventions for preschoolers observed no improvement on masked measures of ADHD symptoms (Rimestad et al., 2016). In adults, cognitive behavioural interventions yielded a small reduction on symptoms (Knouse et al., 2017).[1] Of note, although the effects of behavioural interventions on ADHD symptoms are disappointing, in children with ADHD this type of therapy improves behavioural problems and parenting (Daley et al., 2014).

Overall, the two primary intervention options for ADHD are medication and behavioural intervention. The former substantially reduces the symptoms of the disorder, yet its overall clinical value, including normalization and impact on functional outcomes, is somewhat limited. While behavioural intervention is useful to address some co-occurring problems in individuals with ADHD, its effect on the core symptoms of the disorder is, at best, minor. Crucially, neither of these therapeutic options could be considered a *translational intervention* for ADHD, as we will describe in the following section.

Translational Interventions for ADHD

Translational interventions aim to link findings from basic research to the development of promising therapeutic innovations. Grounded on the developmental causal modelling framework (Morton & Frith, 1995), in ADHD the translational model

[1] Although Young et al. (2020) found slightly higher improvement, this meta-analysis consisted of three studies to which the fixed-effects model (less robust in this context) was applied.

(**Figure 1**) consists of targeting the underlying neurocognitive processes known to mediate the causal pathways between originating risk and clinical expression (Sonuga-Barke & Cortese, 2018; Sonuga-Barke & Halperin, 2010).[2] The design of well-founded

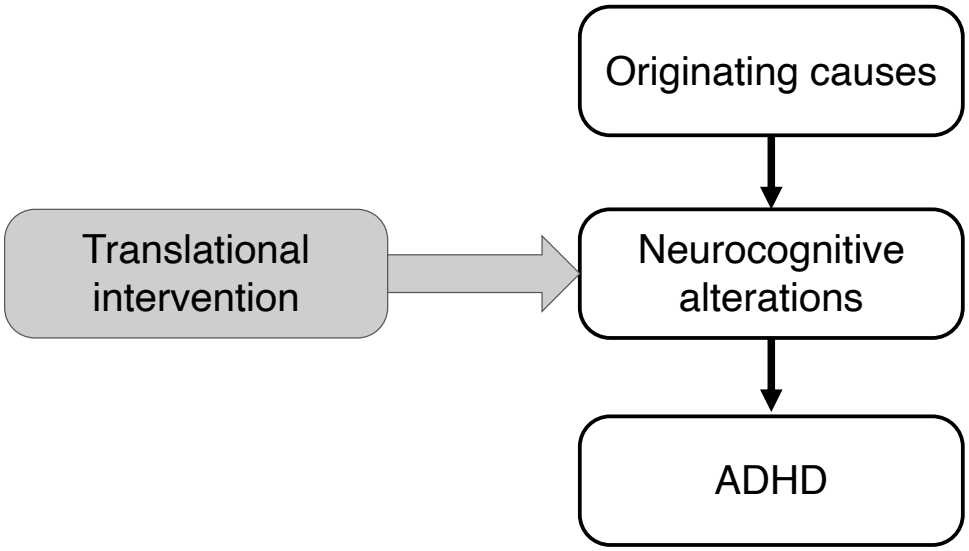

Figure 1. Translational model in ADHD.

translational interventions for ADHD requires a thorough understanding of the nature of the disorder, especially its pathophysiology.

Despite the presumed potential of this approach, the main treatments to address ADHD are not built on, or developed in response to, advances in ADHD science concerning the causes and mediators of the condition (Sonuga-Barke & Cortese, 2018). This is the case of the two first-line therapies described in the previous section: pharmacological treatments and behavioural interventions. The former was initially discovered through clinical trial and error and its subsequent innovation has had more to do with advances in knowledge about drug pharmacodynamics and pharmacokinetics (e.g., extended-release formulations of methylphenidate) rather than an understanding of ADHD psychopharmacology (Swanson et al., 2003; Volkow & Swanson, 2003). Critically,

[2] There are other relevant theoretical proposals that could ground alternative translational models (e.g., Borsboom et al., 2019; Coghill, Hayward, et al., 2014). However, to the best of my knowledge, there are no interventions for ADHD formally based on these models. Additionally, although promising, these alternative theoretical proposals include a set of limitations whose discussion exceeds the scope of this thesis.

neurocognitive improvements produced by medication do not correlate with clinical improvement in ADHD symptoms (Lee et al., 2022). Regarding behavioural interventions, this treatment was developed to address general or disruptive behavioural problems, instead of specifically targeting ADHD-related neurocognitive alterations.

The most widely researched translational intervention in ADHD is computer-based cognitive training (Sonuga-Barke & Cortese, 2018). Leveraging the brain's plasticity, the treatment aims at strengthening the neurocognitive processes that are assumed to underlie ADHD symptoms (e.g., working memory, attentional control, inhibition, multiple executive functions). To do so, this intervention consists of structured and repeated exposures to cognitively demanding tasks. Despite its theoretical plausibility, a recent meta-analysis showed minimal or no effects of computerized cognitive training on ADHD symptoms when assessed by probably blinded raters (Westwood et al., 2023).

Grounded on a neurodevelopmental perspective, Sonuga-Barke and Halperin (2010) proposed a novel format of cognitive training delivered by parents. This intervention, aimed at preschoolers, consists of non-computerized play-like activities that are integrated into daily parent-child interaction. Unlike behavioural parent training, in this translational intervention parents are taught diverse structured plays targeting multiple neurocognitive domains (e.g., "Simon Says" to train response inhibition), along with strategies to progressively adapt them to the child level. Therefore, parents direct and regularly practice these activities with their child. Given the potential of this structured-play-based parent training, we (i.e., my supervisors and I) conducted a meta-analysis of randomized controlled trials about the effects of this translational therapy in preschoolers (Coll-Martín et al., 2019). The results (**Figure 2**) showed that the intervention improved ADHD symptoms only in unmasked but not in masked outcomes, which is a similar pattern to behavioural parent training.

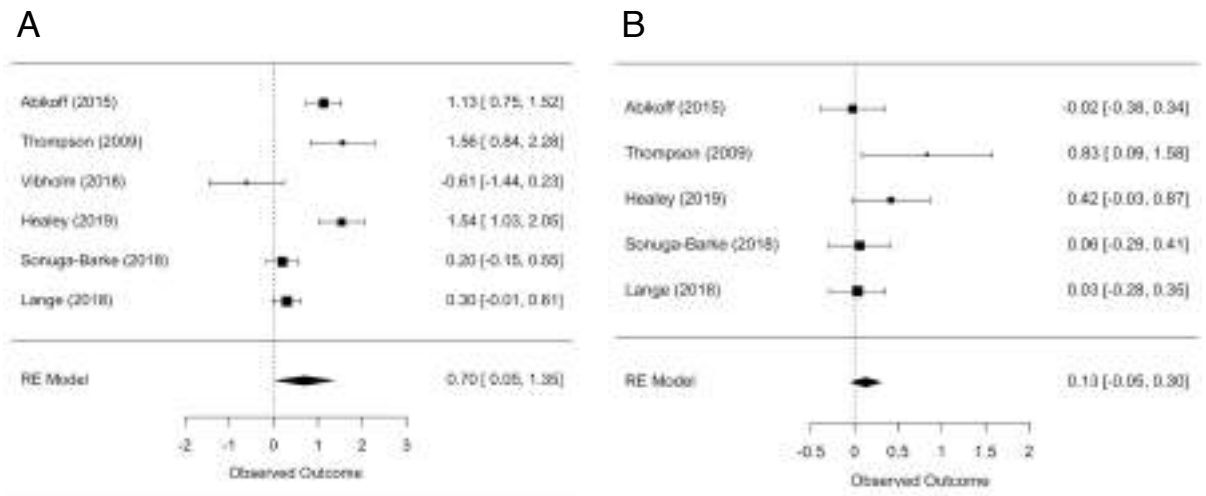

Figure 2. Effects of play-based parent training on the improvement of ADHD symptoms in preschoolers. Panel A: unblinded outcomes. **Panel B**: blinded outcomes. From Coll-Martín et al. (2019).

Beyond cognitive training, translational interventions for ADHD have also been developed in the form of neurotherapeutics. The most extensively researched therapy is neurofeedback, an intervention that teaches participants, particularly those with ADHD, to self-regulate specific neural activity within certain regions or networks presumed to be altered in the disorder. This is achieved through a process of trial and error, using real-time visual or auditory feedback of the corresponding brain activation patterns. Despite its popularity, the efficacy of neurofeedback is limited to a small reduction exclusively on inattention (Riesco-Matías et al., 2021). Other neurotherapeutics such as non-invasive brain stimulation, namely repetitive transcranial magnetic stimulation and transcranial direct current stimulation, has produced limited and somewhat unpromising evidence of having an impact on ADHD symptoms (Rubia, 2022; Westwood et al., 2021).

In sum, the current translationally oriented science-driven interventions for ADHD have not been able to produce substantial improvement in the core symptoms of the disorder, albeit some of these proposals have been scarcely studied. However, its theoretical plausibility, built upon an extensive body of basic research (Halperin et al., 2012; Sonuga-Barke & Cortese, 2018), justifies to keeping exploring alternatives based on this translational approach. Noteworthy, advancements in basic research on ADHD

may pave the way for new and more refined science-driven therapeutic developments. In the next section, we will discuss how the conception of ADHD has evolved over the years and what implications this has for translational research and interventions.

Towards a biopsychosocial model of ADHD: Dimensional perspective, heterogeneity, and multiple neurodevelopmental courses

From the medical to the biopsychosocial model

In the first section of this thesis, we have provided a definition of ADHD in line with what the DSM-5 asserts it to be. From that conceptualization, ADHD is a technical construct representing a practical attempt to characterize and clinically address the following empirical reality (Coghill & Sonuga-Barke, 2012; Sonuga-Barke, 2019): (a) attentional problems, overactivity and impulsive behaviours—clinically referred to as symptoms—cluster together and can be statistically and prognostically differentiated from other clusters of problems, despite a degree of overlap between and heterogeneity within; and (b) developmentally inappropriate and persistent level of this cluster—clinically referred to as syndrome—can be greatly impairing and relate to multiple negative outcomes. In this sense, the term "disorder" is no more than a shorthand for this impairing cluster, along with some specifiers (e.g., persistency, child onset, generalizability) aimed at providing certain coherence and integrity.

Unlike clinical pragmatism, the empirical level of ADHD turns the diagnosis into working models[3]—an approximation to the actual nature of the disorder—to be tested, updated, and refined. Although research on ADHD models may contribute to a more useful diagnosis, their most direct contribution lies in their influence on therapeutic innovation. The two main models in ADHD are the *medical* model and the *biopsychosocial* model

[3] To avoid confusion of terms: The previous section refers to a model of *intervention* in ADHD, with the translational model in **Figure 1** underpinning the present thesis. In this section, we present and discuss the distinct models on the *nature* of ADHD.

(Beauchaine, 2003; Coghill & Sonuga-Barke, 2012; Engel, 1977; Sonuga-Barke, 2013; Sonuga-Barke & Halperin, 2010). A comparative summary of their main characteristics is shown in **Table 1**.

Table 1

Comparison of Characteristics Between the Medical and Biopsychosocial Models of ADHD

Assumption	Medical model	Biopsychosocial model
Philosophy	Essentialism	Nominalism
Differences	Discrete, categorical, cualitative	Dimensional, continuous, cuantiative
Etiology	Bio-genetically determined	Genes and biopsychosocial enviroment
Pathophysiology	Single core deficit	Heterogeneous
Symptoms	Dysfunctional	Not necessarily dysfunctional
Impairment	Symptom-inherent	Context-dependent
Course	Child-onset neurodevelopmental disorder	Any

Note. This table presents the most prototypical and arguably consistent view of the two models, but hybrid variants are also possible. Etiology here refers to both originating causes and factors leading to the remission of the disorder. Pathophysiology refers to the neurocognitive mechanisms that underlie ADHD symptoms. In both models, these neurocognitive alterations act as mediators between etiological factors and symptoms.

The traditional medical model of disease underlies the original concept of ADHD and is shaped by a set of seemingly implicit essentialist assumptions derived from the diagnostic label. From this view, ADHD is a discrete category with nonarbitrary boundaries that distinguish cases from controls. This implies that clinical symptoms are qualitatively different from variations across the normal range of expression in the population due to having distinct causes and underlying mechanisms. Although the etiology of ADHD can be complex and even heterogeneous, the medical model only considers genetic or early biological factors (e.g., low birth weight, prematurity, exposure to neurotoxins). These precursors give rise to a single neurocognitive central deficit (e.g., behavioural inhibition) that serves as a biomarker to distinguish people with and without ADHD on an individual

basis. Consequently, symptoms are the manifestation of a dysfunctional condition that inherently impairs the functioning of the people with the disorder.

In contrast, the biopsychosocial alternative to ADHD is closely aligned with the trait approach. From this *dimensional* perspective, ADHD is viewed as a social construct built upon extreme levels of normal variation within the population, which reflect shared causes and underlying mechanisms throughout a continuum of severity. Indeed, the impairment associated with the disorder is context-dependent, as it arises from a mismatch between that extreme trait expression and the social environment. According to this model, ADHD results from dynamic, complex, and heterogeneous interplays between genes and the biopsychosocial environment (i.e., biological, psychological, and social factors) that, along with altered neurocognitive processes, interact reciprocally across development. The neurocognitive processes underlying ADHD are heterogeneous, that is, multiple pathways converge on equal or similar symptoms. In this regard, at least for some individuals, ADHD symptoms may reflect functional ways of thinking and behaving that are usually maladaptive (e.g., delay avoidance). Ultimately, the etiological and pathophysiological heterogeneity opens up the possibility that the disorder may have multiple developmental trajectories, including late- or adult-onset ADHD.

Compared to the medical model, the biopsychosocial model holds out considerable hope for translational progress. Indeed, its less restrictive assumptions offer a wider range of possibilities for addressing the disorder. Crucially, a growing body of research is challenging the core assumptions of the medical model, favouring instead the biopsychosocial perspective. This shift is leading to a reconceptualization of ADHD. Next, we will review this evidence and discuss its translational implications.

Evidence and translational implications of the biopsychosocial model

Dimensions over categories

Perhaps the most supported biopsychosocial assumption is the dimensional distinction between people with and without ADHD. In this case, converging evidence at behavioural (Haslam et al., 2006), neurocognitive (Frazier, 2007), and genetic (Gjone, 1996) levels supports a continuous rather than a categorical structure of the disorder. For example, there is no difference in patterns of heritability at the extreme of the distribution. Of note, ADHD has been referred to as a paradigmatic case of mental disorder based on a dimensional architecture (Coghill & Sonuga-Barke, 2012). Moreover, a recent meta-analysis found that most psychopathology domains, like other psychological constructs, are latently continuous (Haslam et al., 2020).

The dimensional approach opens up new opportunities for translational research on ADHD. For instance, ADHD theories could be tested and generated based on the neurocognitive correlates of symptom severity in community- or population-based samples (Hilger & Fiebach, 2019; Hilger et al., 2020), which are typically larger in size than those of case-control studies (Norman et al., 2023). For example, impaired vigilance (Craig & Klein, 2019) and higher irrelevant distraction (Forster & Lavie, 2016) positively correlated with ADHD symptoms in nonclinical samples. Moreover, translational intervention for ADHD symptoms could benefit from general theories of cognitive enhancement (e.g., Inzlicht et al., 2018; von Bastian et al., 2022).

Psychosocial environment may also play an etiological role

Finding sound evidence of psychosocial etiological factors in ADHD has been and remains highly challenging**.** To begin with, twin studies have estimated that ADHD is 74% due to heritable factors (i.e., genetic variation), with the rest mostly explained by unique

environmental exposures or random error (Faraone & Larsson, 2019; Gidziela et al., 2023). In these studies, the percentage attributed to the shared environment—the context affecting both siblings, which is, in principle, more subject to being systematically modified—is marginal at most (Burt, 2009; Gidziela et al., 2023). Of note, the psychosocial component (e.g., parenting) is only one part of the environment, the other being biological (e.g., exposure to neurotoxins, low birth weight). Moreover, many of the associations found between environment and ADHD (e.g., household chaos, maternal smoking) do not seem to reflect an environmental cause, but rather gene–environment correlations (Agnew-Blais et al., 2022; Haan et al., 2022).

However, twin studies present certain limitations that could overemphasize the weight of genetics at the expense of environmental factors. In this sense, gene–environment interactions are not measured in the typical twin design (Sonuga-Barke et al., 2023). Indeed, only 20–30% of the whole estimated heritability has been traced in genome-wide studies of common genetic variants (Demontis et al., 2019), while the remainder “missing heritability” may also be accounted for by other elements including gene–environment interactions. Moreover, when controlled for familial and genetic cofounding, a small causal link between child maltreatment and ADHD symptoms has been found (Dinkler et al., 2017). Crucially, extremely depriving institutional environments in early life are likely to be causally associated with a variant of ADHD that persists into adulthood (Kennedy et al., 2016)—a presentation that are not accounted for by malnutrition (Sonuga-Barke et al., 2008).

Together, extreme environmental factors may provide a primary explanation for ADHD, though the effect of common exposures is small and less studied. Additionally, based on more basic research into neurocognitive principles, it seems plausible that environmental enrichment can prevent ADHD (Halperin et al., 2012), while high digital exposure could mechanistically exacerbate the risk of developing it (Sonuga-Barke &

Kostyrka-Allchorne, 2023). Therefore, the possibility that environmental factors, including psychosocial ones, play a role in the etiology of ADHD provides a rationale for believing that environments created by translational interventions (e.g., cognitive training, neurotherapeutics) may have a substantial impact on core symptoms. However, further causal studies are still needed to consolidate this conclusion.

The idea of a single central deficit is arguably untenable

Initially, ADHD was thought to have a core neurocognitive deficit, either executive (Barkley, 1997), arousal/energetic (Sergeant, 2005), or motivational (Sonuga-Barke et al., 1992). Despite extensive research on the subject, the search for this central deficit as a diagnostic biomarker has been unsuccessful: no specific neurocognitive alteration—or a combination thereof—has been found to distinguish between an individual with and without the disorder (Astle & Fletcher-Watson, 2020; Cortese et al., 2023; Nigg et al., 2005). In fact, individuals with ADHD can be classified into dissociable but potentially overlapping neurocognitive subtypes, depending on the profile of neuropsychological alterations they present (De Zeeuw et al., 2012; Sjöwall et al., 2013; Sonuga-Barke, Bitsakou, & Thompson, 2010; Sonuga-Barke, 2005). Interestingly, even studies analyzing up to six different neurocognitive subtypes find a group with no observable neurocognitive impairment (Coghill, Seth, & Matthews, 2014).

Against this backdrop, translational research on ADHD pathophysiology should measure multiple neurocognitive domains, including both executive and nonexecutive domains (Castellanos et al., 2006). This heterogeneity is somehow consistent with the negligible or small ADHD-related effects reported in large-scale neurocognitive studies (Aduen et al., 2020; Bernanke et al., 2022; Norman et al., 2023), in which upward-biased estimates are less common. On a practical level, translational interventions should also target nonexecutive processes instead of focusing only on the executive ones, as has mainly been done so far (Sonuga-Barke & Cortese, 2018; Westwood et al., 2023).

Symptoms are not necessarily dysfunctional or impairing

Given the variety of causal pathways leading to the development of ADHD, it has been argued that at least in some of these pathways the symptoms are not the product of an underlying dysfunction. An example is an ADHD variant that could reflect aversion to delay. In this case, impulsivity is conceived as a functional behaviour aimed at escaping a situation of delayed gratification, while inattention and hyperactivity are behaviours emitted to reduce the perception of time in situations where delay is unavoidable (for a review of evidence for this proposal, see Sonuga-Barke, Wiersema, et al., 2010). In an environment characterized by the attributes of our current context (e.g., compulsory formal education; Hinshaw, 2018), these behaviours are functional but typically maladaptive, leading to the impairment associated with ADHD.

The ideological extension of the above type of accounts has led to the neurodiversity concept (Sonuga-Barke & Thapar, 2021). From this view, ADHD is part of a wider spectrum of naturally occurring variations in the style of thinking and behaviour. Thus, interventions should focus on creating environments of acceptance and accommodation as well as promoting personal agency by uncovering the strengths and talents of neurodivergent people (Franke, 2023; Schippers et al., 2022; Sonuga-Barke et al., 2023). However, the whole neurodiversity movement is currently more a socio-cultural rights-based ideology than an empirical-based approach. Even so, the idea that impairment—and, to some extent, the symptoms themselves—is context-dependent has been supported elsewhere (Cheesman et al., 2021; Sonuga-Barke & Fearon, 2019) and is one of the main positions to account for the phenomenon of late-onset ADHD in the following section.

ADHD can be a late-onset condition

ADHD has been conceptualized as a child-onset neurodevelopmental condition. Indeed, the DSM-5 (APA, 2013) states that the disorder begins in childhood and, consequently, sets as a diagnostic criterion that several symptoms must have been present prior to age 12. However, this neurodevelopmental notion of the disorder has been recently challenged by longitudinal studies from different countries suggesting that a substantial proportion of adults with ADHD did not meet the diagnostic—nor "several" symptoms—in childhood (Agnew-Blais et al., 2016; Caye et al., 2016; Cooper et al., 2018; Moffitt et al., 2015). This so-called late-onset ADHD, estimated to constitute around 1%–2% of the population and half of all adults with ADHD, presents a life impairment that demands clinical attention (Asherson & Agnew-Blais, 2019).

While late-onset ADHD is becoming more scientifically recognized, the controversy centers around the extent to which these symptomatic manifestations reflect the same etiopathophysiology as the conventional form of the disorder (Asherson & Agnew-Blais, 2019; Polanczyk et al., 2019). From a translational perspective, determining whether late-onset ADHD symptoms are the result of neurocognitive alterations other than those of childhood could lead to the development of specific interventions for each variant of the disorder. This open question is also crucial to the conceptualization of ADHD as a neurodevelopmental condition. This issue is more deeply developed in **Appendix** (in Spanish) and will be taken up in the general discussion of this dissertation.

In conclusion, the empirical concept of ADHD is evolving rapidly, as the shift from a medical model to a biopsychosocial perspective gathers pace promoted by scientific progress. Since the biopsychosocial model holds a dimensional assumption, a crucial step in this transition is the integration between frameworks aimed at explaining the general neurocognitive functioning and those that account for its variation related to ADHD symptoms. Indeed, it has been argued that the potential alterations in ADHD should be nested within well-established neurocognitive frameworks (Bush, 2010; Castellanos &

Tannock, 2002). In the following section, we will illustrate how *attention*—understood as a general modulatory system of cognition—may constitute a central axis for characterizing two key dissociable mechanisms in both typical cognitive functioning and ADHD theories: *executive* and *arousal* processes.

Executive and arousal attentional processes

General functioning and assessment

Attentional networks

Different theories have emphasized distinct aspects of attention, giving rise to a diversity of attentional phenomena that have even led some authors to question the very existence of attention as an explanatory concept (Hommel et al., 2019). One of the most renowned alternatives to this pessimistic view of attention is the integrative model of the attentional networks proposed by M. I. Posner and colleagues (Petersen & Posner, 2012; M. I. Posner & Petersen, 1990). According to this view, attention should be considered as a system exerting three independent, albeit interactive, attentional functions with an important role in the coordination of behaviour. These attentional functions are implemented in three neural networks, anatomically separated from the attentionally-modulated processing systems.

First, the alerting network regulates the level of arousal and activation for both momentary readiness for imminent events (phasic alertness) and sustained performance over long time periods (tonic alertness or vigilance). This network involves noradrenergic innervations from the locus coeruleus to the frontal and parietal lobes of the right hemisphere. The second subsystem is the orienting network, responsible for prioritizing sensory inputs by selecting a modality, spatial location, or object. It comprises cortical regions such as parietal cortices and frontal eye fields and the subcortical structures of pulvinar nuclei and superior colliculi. Finally, the executive network is in charge of monitoring performance and prioritizing goal-oriented responses in conflict situations. This third subsystem includes the anterior cingulate and prefrontal regions.

Several tasks have been developed to simultaneously measure these three components of attention, the most common being the Attention Network Test (ANT; Fan et al., 2002; see de Souza et al., 2021, for a review). This computerized task and other variants like the ANTI (ANT for interactions; Callejas et al., 2004) present a sequence of visual stimuli that combines a spatial cueing (M. I. Posner, 1980) and warning signal task with a flanker paradigm (Eriksen & Eriksen, 1974). Subtractions between the task conditions resulting from specific manipulations of warning, cueing, and flankers provide the effects of alerting, orienting, and congruency (an index of the executive network), respectively. Different from the ANT, the use of a different cue for measuring alertness and orienting in the ANTI also allows the measure of the interaction between the three attentional networks.

Vigilance

Despite the comprehensiveness of the attentional networks model, it does not characterize, at least explicitly, a crucial dissociation in the vigilance domain. The construct of vigilance, central to the phenotypic and neurocognitive characterization of ADHD (Huang-Pollock et al., 2012; Wilding, 2005; Willcutt et al., 2005), is generally defined as the attentional capacity to maintain performance over time. Given the diversity of terms and measures linked to vigilance, some authors deem it as a nonunitary concept (Langner & Eickhoff, 2013; Luna et al., 2018; Sturm et al., 1999). In this vein, Luna et al. distinguish two components of vigilance: executive vigilance and arousal vigilance.

Executive vigilance is the ability to detect infrequent but critical signals among nonsignal stimuli. It is measured with tasks derived from the continuous performance test (CPT) paradigm such as the AX-CPT (Rosvold et al., 1956) or the Test of Variables of Attention (TOVA; Greenberg & Waldman, 1993). This type of task seems to involve executive mechanisms of sustained attention for stimuli discrimination and goal-oriented response selection. Notably, response-inhibition CPTs (e.g., Conners's CPT [Conners,

2000], Sustained Attention to Response Task [SART; Robertson et al., 1997]), the most common CPT variant in ADHD research, also require motor suppression of the preponderant response in the presence of the target stimulus. Key measures of executive vigilance are hits (inverse of omission errors) and false alarms, both tending to decrease with time on task (for a discussion about false alarms, see Thomson et al., 2016).

Arousal vigilance is the capacity to sustain a rapid reactivity to any environmental stimulus without implementing any control over the selection of the response executed. This form of vigilance is measured in simple reaction time (RT) tasks, such as the Psychomotor Vigilance Test (PVT; Lim & Dinges, 2008) and the WAFA test of the Vienna Test System (Schuhfried, 2013). These tasks seem to record an arousal component of vigilance, a mechanism that could be more related to physiological levels of excitability. The main indices of arousal vigilance are the mean and variability of the RT and the attentional lapses. Contrary to executive vigilance, this vigilance decrement manifests as an increase in the indices during the task.

To simultaneously integrate both components of vigilance into the assessment of the three attentional networks, Luna et al. (2018) designed the *ANT for Interaction and Vigilance—Executive and Arousal Components* (ANTI-Vea). This task is mainly based on the ANTI, which constitutes the bulk of ANTI-Vea trials. To assess executive vigilance, the flanker task is embedded in a CPT structure where participants must detect an infrequent target (i.e., the vertical displacement of the central arrow). For its part, arousal vigilance is measured with a salient stimulus (i.e., a red down counter) that the participant must stop as fast as possible. Worthy of note, the length of the task (~33 min) enables the analysis of the decrement of both types of vigilance across the six blocks of the task (Luna, Roca, et al., 2021).

ADHD alterations

As mentioned above, pathophysiological heterogeneity means that there are different neurocognitive pathways that can give rise to similar ADHD symptoms, which is known as equifinality. This implies that there may be different subgroups of people with ADHD depending on the type of cognitive pathway they have impaired (e.g., executive functions, reward processing, timing, arousal regulation, mind-wandering). At the same time, neurodevelopment and the reciprocal interaction with the environment may give rise to new secondary neurocognitive impairments. For example, primary executive deficits may generate alterations in reward processing, and vice versa (Sonuga-Barke, 2005). In case–control or correlational research, this heterogeneity may be observed as ADHD-related small effects in a wide range of processes (e.g., response inhibition, working memory, attentional control, delay discounting, alertness; Coghill, Seth, & Matthews, 2014; Willcutt et al., 2005) but not in all (e.g., orienting network; Arora et al., 2020).

The models we will present below were originally designed to explain ADHD in terms of central neurocognitive deficits. While they were initially viewed as competing theories, from a biopsychosocial perspective, each of these models might explain a portion of the neurocognitive variance or differences associated with ADHD.

Response inhibition model

Barkley's (1997) response inhibition model, also known as the self-regulation model, proposes that response inhibition is the primary deficit in ADHD. From this view, virtually all behavioural regulation and executive functioning are secondary to this core inhibitory deficit. Specifically, behavioural inhibition would allow suppression of predominant responses to create a delay period within which to implement self-directed responses (i.e., executive functions) while controlling for interference. This executive dysfunction in ADHD has been conceived as fixed, pervasive, and ubiquitous deficits (i.e., largely context- and

state-independent; Castellanos et al., 2006; Sonuga-Barke & Halperin, 2010). In this sense, Willcutt et al. (2005) found that people with ADHD perform worse on a wide range of tasks and executive functions.

State-regulation deficit model

The state-regulation deficit account for ADHD (Sergeant, 2000; van der Meere, 2005) is based on the cognitive-energetic framework (Sanders, 1983, 1998), which proposes that information processing efficiency is primarily a product of elementary cognitive stages and their energetic distribution. The energetics pools encompass the phasic arousal and activation (i.e., tonic arousal) levels and their regulation through effort allocation. Unlike Barkley's (1997) model, here ADHD would have to do with alterations in energetic mechanisms (Sergeant, 2005). Specifically, ADHD is viewed in terms of arousal dysregulation, which becomes evident in contexts with a low (hypoarousal) or high (hyperarousal) degree of stimulation or demands (Sonuga-Barke, Wiersema, et al., 2010). This context dependency in ADHD-linked alterations has been observed in a meta-analysis of cognitive tasks (Metin et al., 2012). Furthermore, a systematic review with measures of the autonomic nervous system reported a dysregulation of arousal linked to ADHD, mainly in the form of hypoarousal (Bellato et al., 2020).

Neurodevelopmental model

Halperin and Schulz's (2006) neurodevelopmental model of ADHD proposes that the onset of ADHD in childhood is due to subcortical brain dysfunctions that remain stable over the lifespan, while the trajectory of symptom severity with age is dependent on the development of the prefrontal cortex. The non-executive subcortical alterations include basic control, timing, and, crucially, arousal processes. For its part, the prefrontal cortex subserves executive processes, which are not central to the early emergence of ADHD in childhood but influence its developmental course into adulthood. Of note, these executive

alterations may explain both the persistence/remission of child-onset ADHD and the emergence of the late-onset form of the disorder. Although a longitudinal study provided preliminary evidence for this model (Halperin et al., 2008), subsequent research has yielded mixed results at best (Luo et al., 2019; van Lieshout et al., 2013).

Integrating frameworks

Based on a broad conceptualization of attention, a meta-model can be elaborated to characterize and distinguish executive and arousal mechanisms, integrating both its general functioning and its alterations linked to ADHD (**Figure 3**). This meta-model is intended to serve as an anchor for the studies of the present thesis, their interpretation, and implications. It is based on a dimensional perspective of ADHD where theories of general attentional functioning and those about its alterations in ADHD not only can but

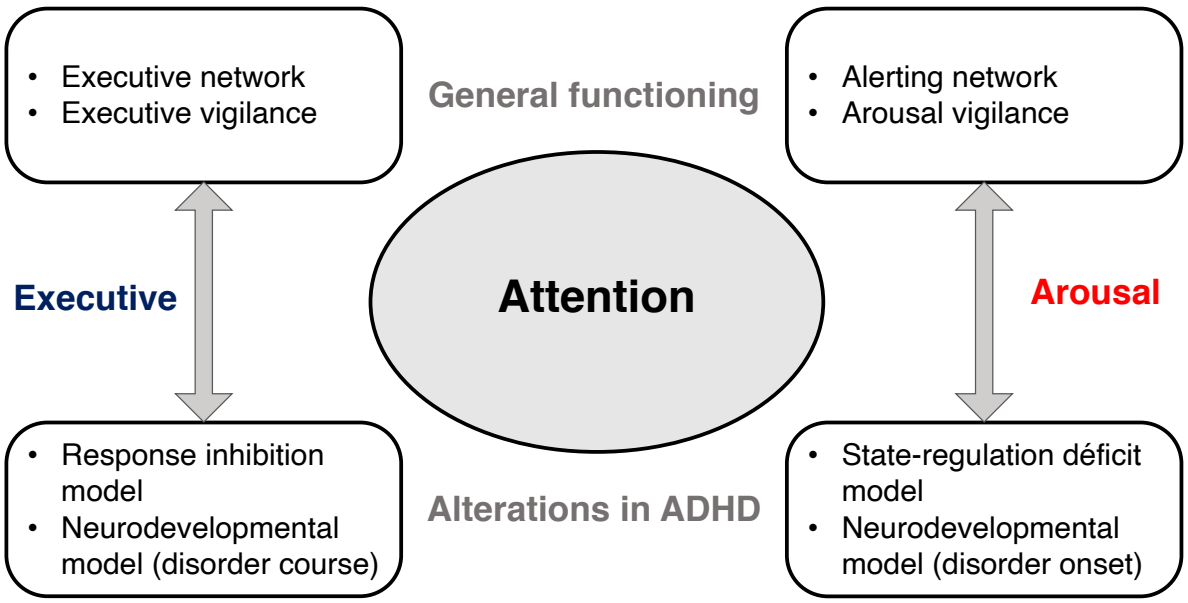

Figure 3. Conceptualization and relevance of the executive and arousal components of attention from an integrative view. Theories of the general functioning of attention include M. I. Posner and Petersen's model (1990), which distinguishes the executive and the alerting networks, as well as Luna et al.'s (2018) conceptualization of vigilance, differentiating between executive and arousal vigilance. ADHD theories have proposed that the central deficit of the disorder resides in alterations in executive (response inhibition model; Barkley, 1997) or arousal (state-regulation deficit model; Sergeant, 2005) processes. In addition, Halperin and Schulz's (2006) neurodevelopmental model considers that ADHD is initiated in childhood by alterations involving arousal processes, while the development of the disorder throughout lifespan depends on executive mechanisms.

should be integrated. This framework encompasses previous proposals that have been made to connect the attentional network model with theories of executive dysfunction and arousal dysregulation (Berger & Posner, 2000; Bush, 2010; Martella et al., 2020). In addition, the present framework extends that dissociation between executive and arousal processes into the study of both vigilance and ADHD neurodevelopment.

Overall, the model serves to explain and predict ADHD-related deficits observed in two well-established attentional components, namely executive and arousal. Crucially, all the elements of the model can be evaluated simultaneously using the ANTI-Vea previously mentioned.

CHAPTER II: AIMS AND OVERVIEW OF RESEARCH

Aims and overview of research

The previous chapter has evidenced a somewhat discouraging picture of ADHD science. After decades of research and investment in understanding the underlying causes and mechanisms of the disorder, this body of scientific knowledge has not yet been able to develop effective interventions. This has led some scholars to believe that ADHD science is in a certain state of crisis (Sonuga-Barke, 2020, 2023). To move past this situation, a shift from the medical to the biopsychosocial perspective of the disorder is fundamental to formulating research questions in ADHD basic science as well as interpreting their translational implications. Unfortunately, until recently the biopsychosocial model has not been the dominant paradigm that has guided basic research (Hilger et al., 2020; Moses et al., 2022; Owens et al., 2021).

In the current dissertation, we face the same far-reaching key question as the medical model: *What are the neurocognitive causes of ADHD and how to target them through translational interventions?* However, we have endeavored to ensure that the assumptions of the biopsychosocial model (**Table 1** in **Introduction**) form the foundation of our study designs. First and foremost, consistent with a dimensional perspective, we have considered the distribution of ADHD symptomatology severity in community samples as a valid approach to the study of the neurocognitive mechanisms of the condition. Second, assuming that impairment in ADHD is context-dependent, we have focused on the neurocognitive nature and correlates of the symptoms of the disorder. Third, we have examined the possibility that the neurocognitive mechanisms of late-onset ADHD—and, therefore, the design of translational interventions—are different from those associated with conventional ADHD. Fourth, in line with the heterogeneous pathophysiology of the disorder, we have evaluated two key neurocognitive domains of distinct nature and theoretical tradition, namely executive and arousal attentional processes. Finally,

assuming the potential causality of postnatal environmental factors on the mechanisms underlying the disorder, we have explored the effect of nonpharmacological interventions on neurocognitive measures.

Although executive and arousal processes do not account for all ADHD alterations, their selection for this thesis is based on a balance between their representativeness and feasibility for the research aims. Indeed, theories of ADHD based on executive or arousal processes are typically the most influential and recurrently invoked in the literature (Luo et al., 2019). In addition, theories linking ADHD to motivational alterations, such as the delay aversion model, generally dovetail with the arousal state-regulation deficit model, sharing predictions in most contexts (Sonuga-Barke, Wiersema, et al., 2010; van der Meere et al., 2010). Moreover, the contrast between executive and non-executive processes is critical when dissociating neurocognitive alterations, as in the neurodevelopmental model of ADHD (Halperin & Schulz, 2006). Finally, our laboratory's development of the Attentional Networks Test for Interactions and Vigilance—Executive and Arousal Components (ANTI-Vea), which is a cornerstone of this thesis, allows for operationalizing the distinction between the two processes within the theoretical and empirical framework of experimental models of attention. This distinction is fundamental given that in ADHD literature it is challenging to find attentional measures of arousal that are virtually free of executive load, as conceptualized by Luna et al. (2018).

In parallel with the crisis of ADHD science, this thesis also considers the issue of a broader crisis affecting psychological science as a whole: the credibility or confidence crisis (Alister et al., 2021; Vazire et al., 2022). This crisis was evidenced by the arguably low rate of replicability of findings in the different branches of psychology, including mental health (Open Science Collaboration, 2015; Youyou et al., 2023). To improve replicability in psychology, the implementation of open science practices is crucial (Korbmacher et al., 2023). In this sense, the present dissertation provides open access to all data, codebooks,

materials, and scripts from all studies, when applicable. Additionally, this thesis attempts to address the issue of underpowered studies, a replicability concern notably prevalent in this ADHD field, especially given the typically poor reliability scores of neurocognitive measures (Parsons et at., 2019).

Based on all the considerations above, the aim of the present dissertation was to *deepen our understanding of the executive and arousal attentional alterations underlying ADHD symptoms, grounded on dimensional and neurodevelopmental perspectives from a broader biopsychosocial framework, and ultimately considering implications for translational interventions*. The achievement of this general objective was carried out in five studies grouped into three chapters, each involving different types of research (**Table 1** for a comparison chart), as well as in the overarching conceptual analysis provided in the **General Discussion**. Each of these four sections aimed to attain several more specific aims, as described next.

Table 1

Characteristics of the studies included in the present dissertation

Study	Research	Design	Sample	Attentional components
Chapter III: Study 1	Instrumental	N/A	N/A	Executive and arousal
Chapter IV: Studies 2–4	Empirical	Correlational	Community	Executive and arousal
Chapter V: Study 5	Review	Intervention	Clinical [a]	Arousal

[a] Participants diagnosed with ADHD.

In **Chapter III** (**Study 1**) we presented a free and online resource to assess the functioning of the three attentional networks (alerting, orienting, and executive control) and two vigilance components (executive and arousal vigilance): the ANTI-Vea-UGR platform (https://anti-vea.ugr.es/). We developed this platform to easily run, collect, and analyze large volumes of data from the ANTI-Vea or its subtasks, either in typical lab conditions or online in remote settings. Consistent with open science principles, the task versions are open source, anonymized ANTI-Vea online participant data is stored on a freely accesible

public server, and task analyses can be performed using a custom Shiny app or its associated R code. In addition, we undertook a narraitve review of studies conducted to date using the ANTI-Vea in different designs, contexts, and populations. This work aimed to address three specific goals of the dissertation, namely establish a neurocognitive behavioural task capable of (a) *feasibly collecting large samples from different contexts*, (b) *measuring relevant indices of attentional functioning with sufficient reliability*, and (c) *conceptually and empirically differentiate between executive and arousal measures*. The manuscript of this study has been published in the *Journal of Intelligence* (Coll-Martín, Román-Caballero, et al., 2023).

Once we established the adequacy of the ANTI-Vea to measure executive and arousal attentional processes, in **Chapter IV** (**Studies 2–4**) we focused on the role of these neurocognitive processes in ADHD symptoms from an associative approach. The procedure for the three studies in this chapter was very similar: A community sample of university students perform the ANTI-Vea, either the lab or online version, and complete self-reports on the severity of their ADHD symptoms during childhood (retrospectively assessed) and adulthood. **Study 2**—published in the *British Journal of Psychology* (Coll-Martín et al., 2021) and disseminated in Spanish in *Ciencia Cognitiva* (Coll-Martín, Carretero-Dios, & Lupiáñez, 2022; see **Appendix**)—is a somewhat exploratory approach to the correlations between ANTI-Vea indices and self-reported ADHD symptoms. **Study 3**—currently under review in *Collabra: Psychology* (Coll-Martín, Carretero-Dios, & Lupiáñez, 2023)—is a preregistered close replication of **Study 2** with a greater focus on transparently testing a hypothesized neurodevelopmental dissociation between executive and arousal vigilance in relation to ADHD symptoms, while strictly controlling statistical error rates and robustness. **Study 4**—currently in preparation (Coll-Martín, Sonuga-Barke, et al., 2023)—combined the samples from the two previous studies with a third sample in which participants performed the ANTI-Vea multiple times to shed light on the

neurocognitive characterization of late-onset ADHD. This empirical series aimed at addressing a main specific goal of the present dissertation, namely to *provide transparent and reliable evidence on the relationship between attentional processes and ADHD symptoms distinguishing between (a) executive and arousal components as well as between (b) childhood, adult, and late-onset symptoms*.

The empirical series suggested that both executive and arousal processes were potential targets for translational interventions addressing ADHD symptoms across the lifespan. However, interventions for ADHD have typically been designed to train executive domains, while targeting arousal regulation is generally neglected (Sonuga-Barke & Cortese, 2018; Westwood et al., 2023). Against this backdrop, in **Chapter V** (**Study 5**), we presented the protocol of a systematic review and meta-analysis on the effects of nonpharmacological interventions for ADHD on indices of autonomic arousal. The specific aim of this chapter for the dissertation was (a) *to examine whether current nonpharmacological interventions for ADHD, translational or not, can enhance the regulation of arousal mechanisms*; and (b) *to identify promising arousal-based translational interventions for ADHD*. This protocol has been registered with PROSPERO (CRD42022372965; Coll-Martín, Evangelista, et al., 2022). Although the final version of the review is still being conducted, preliminary results were included in this thesis.

Finally, in **General Discussion** (**Chapter VI**) we placed our findings in the broader context of the science-driven management of ADHD, focused on its neurocognitive mechanisms. The specific aims of this chapter were to discuss (a) *to what extent our findings can shed light on effective translational innovations for ADHD* and (b) *whether these need to be different for the late-onset form of the disorder*. First, we propose a series of competing causal models for conceiving the nature of ADHD based on the characteristics of the precursors of late-onset ADHD. Based on this framework, we integrate the dissertation findings with previous research and discuss their clinical

implications for late-onset ADHD in particular and ADHD in general. Lastly, drawing on the literature on the mechanisms of cognitive training in the general population, we discuss the failure of current translational interventions applied to ADHD and propose new approaches based on arousal mechanisms.

CHAPTER III:
MEASURING EXECUTIVE AND AROUSAL ATTENTIONAL PROCESSES

Study 1

The ANTI-Vea-UGR platform: A free online resource to measure attentional networks (alertness, orienting, and executive control) functioning and executive/arousal vigilance

This work has been published as:

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Abstract

The Attentional Networks Test for Interactions and Vigilance–executive and arousal components (ANTI-Vea) is a computerized task of 32 min duration in the standard format. The task simultaneously assesses the main effects and interactions of the three attentional networks (i.e., phasic alertness, orienting, and executive control) and two dissociated components of vigilance with reasonable reliability (executive and arousal vigilance). We present this free and publicly accessible resource (ANTI-Vea-UGR; https://anti-vea.ugr.es/) developed to easily run, collect, and analyze data with the ANTI-Vea (or its subtasks measuring some attentional and/or vigilance components embedded in the ANTI-Vea). Available in six different languages, the platform allows for adaptation of stimuli timing and procedure to facilitate data collection from different populations (e.g., clinical patients, children). Collected data can be freely downloaded and easily analyzed with the provided scripts and tools, including a Shiny app. We discuss previous evidence supporting that attention and vigilance components can be assessed in typical lab conditions as well as online and outside the laboratory. We hope this tutorial will help researchers interested in measuring attention and vigilance with a tool useful to collect data from large sample sizes and easy to use in applied contexts.

Attentional networks and Vigilance

What is the ANTI-Vea task?

One of the most widely acclaimed approaches to the understanding of human attention is the integrative model developed by Michel I. Posner (Petersen & Posner, 2012; M. I. Posner & Petersen, 1990). According to this renowned author, attention should be considered as a system exerting three different attentional functions: alertness (or selection in time), orienting (or selection in perception), and executive control (or selection at response levels), all playing an important overall role in behavioural coordination. These attentional functions are modulated by three neural networks (Fernandez-Duque & Posner, 2001): a network involving frontal and parietal regions of the right hemisphere modulated by noradrenergic release for alertness, a posterior network (frontal eye field, parietal cortex, and other subcortical structures) modulated by cholinergic innervations for orienting (Corbetta, 1998), and two anterior circuits (fronto-parietal and cingulo-opercular systems) modulated by dopaminergic activity for executive control.

Following the original Attention Network Test (ANT; Fan et al., 2002), specific adapted versions of the ANT have been developed aiming to improve the assessment of the three attentional functions (for a review, see de Souza Almeida et al., 2021). For example, the ANT for Interactions (ANTI; Callejas et al., 2004) task allows to measure not only the way the networks work but also how they interact with each other. The subsequent version, the ANTI-Vigilance (ANTI-V; Roca et al., 2011) was developed with the aim of adding a direct measure of maintenance of attention over time-on-task, that is, vigilance. Additional adaptations of these tasks have been developed, incorporating new components and adjusting them to specific populations, such as children (e.g., Child ANT, Rueda et al., 2004; ANTI-Birds, Casagrande et al., 2012), or patients with visual impairments (Auditory ANT, Roberts et al., 2006, Johnston et al., 2019). For a more

detailed review of the origins and different evolutions of the task, see de Souza Almedia et al. (2021).

Since the expanded use of all of these tasks in attention research began, our team has been one of the most active in the subject and has greatly contributed to this enterprise. We are currently working on the dissemination of the ultimate version developed during the last five years: the ANT for Interactions and Vigilance–executive and arousal components (ANTI-Vea; Luna et al., 2018). The ANTI-Vea is suitable to assess the performance of the attentional networks and their interactions, while it provides two independent measures of vigilance, in line with other well-known tasks like the Sustained Attention for Response Task (SART; Robertson et al., 1997) and the Mackworth Clock Test (MCT; N. H. Mackworth, 1948) for executive vigilance (EV) or the Psychomotor Vigilance Test (PVT; Dinges & Powell, 1985) for arousal vigilance (AV). The platform we have built for the use of the ANTI-Vea and the embedded subtasks is user-friendly, and the data, despite being complex and providing multiple measures, can be easily analyzed with the provided guide and resources. The present tutorial aims to present a detailed description of the ANTI-Vea-UGR platform, introducing a theoretical and methodological description of the ANTI-Vea and then a step-by-step guidance on the use of the online ANTI-Vea task and its different available resources for data analyses.

ANTI-Vea relevance. Dissociation between executive and arousal vigilance

When measuring vigilance, the behavioural pattern usually observed depicts a decrease in performance across time-on-task (Al-Shargie et al., 2019; Doran et al., 2001; J. F. Mackworth, 1964; Tiwari et al., 2009). Theoretical and empirical research has proposed a dissociation between two well-differentiated components of this ability: (a) executive vigilance, understood as the capacity to monitor and detect critical signals that rarely occur over a long period of time; and (b) arousal vigilance, which refers to the ability to maintain a fast response to any stimulus in the environment (see Luna et al., 2018).

Thus, while the EV decrement has been observed as a gradual loss in the hit rate in the MCT and the SART (See et al., 1995; Thomson et al., 2016), the AV decrement has been instead reported as a progressive increase in the average and variability of reaction time (RT) in the PVT (Basner et al., 2011; Lamond et al., 2008; Loh et al., 2004). These behavioural patterns describe the so-called vigilance decrement.

The relevance of the ANTI-Vea relies on allowing a simultaneous (yet independent) assessment of the EV and AV components in a single experimental session. In this task, the EV component is assessed with a signal-detection task similar to the MCT (N. H. Mackworth, 1948), in which participants have to discriminate the vertical displacement of the central arrow. In turn, the AV component involves a reaction time task akin to the PVT (Dinges & Powell, 1985), where participants must stop a countdown as quickly as possible. While the EV decrement is observed as a decrease in both hits and false alarms (FAs), leading to an increase in response bias rather than a loss of sensitivity,[4] in line with Thomson et al. (2016), the AV decrement is characterized as an increase in mean and variability of RT.

What does ANTI-Vea measure?

ANTI-Vea design

The standard ANTI-Vea combines three classic attentional and vigilance paradigms, in three different types of trials, which allows measuring the functioning of the three attentional networks and their interactions (ANTI trials), while simultaneously testing the decrement in executive and arousal vigilance across time-on-task (EV and AV trials respectively). The three types of trials are randomly presented within each block of trials.

[4] Technically, a decrement in sensitivity across the blocks of the ANTI-Vea has been also observed. However, compared to the change in response bias, that effect is substantially lower and likely due to a floor effect in FAs (Luna, Barttfeld, et al., 2021) or other artifacts (Román-Caballero et al., 2023).

The largest proportion of trials (ANTI trials, 60%) are similar to the ones used in the ANTI, based on a classic flanker task (Eriksen & Eriksen, 1974), but also incorporating attentional orienting (as in a spatial cueing paradigm; M. I. Posner, 1980), and alertness (an auditory tone as a warning signal) manipulations. The remaining trials are evenly distributed between the two vigilance paradigms. As described above, for the assessment of the EV component participants have to discriminate the vertical displacement of the central arrow (EV trials, 20%), while in the case of the AV component participants must stop a countdown as quickly as possible (AV trials, 20%).

The general procedure is represented in **Figure 1**. While participants keep their eyes on a black fixation point (“+”) which remains centered on the screen the whole time, a horizontal string of five black arrows appears for 200 ms either above or below the fixation point. Participants have to respond towards which direction the central arrow (i.e., the target) points to, ignoring the direction pointed at by the surrounding arrows (i.e., the distractors), by pressing the corresponding key. If the target points rightwards, participants have to press the letter “M" on the keyboard with their right hand, and if it points leftwards, participants have to press the letter “C” with their left hand. There are two conditions for the ANTI trials: one condition in which all five arrows point in the same direction (congruent trials, 50%) and another condition in which the central arrow points in the opposite direction (incongruent trials, 50%). RT and percentage of errors of both conditions allow assessing executive control (also referred to as “cognitive control” or “executive attention”). An auditory warning signal (2000 Hz) may appear 500 ms before the target in half of the ANTI trials during 50 ms (tone condition and no tone condition). This warning signal tests phasic alertness. The orienting network is assessed with a nonpredictive visual cue (a black asterisk, “*”) of 50 ms that can appear either above or below the fixation point 100 ms prior to the target. This cue can be presented at the same location as the target (valid trials, a third of ANTI trials) or at the opposite position (invalid condition, a

third of ANTI trials), and is absent for the remaining third of ANTI trials (no cue condition). The presence of this visual cue in the same location as the flankers make it easier for participants to respond to the target direction. Furthermore, the auditory warning signal mentioned above increases this facilitation effect, as shown in various studies (e.g., Callejas et al., 2005; Roca et al., 2011). In contrast, in the invalid trials, participants have to reorient their attention to the flankers and the target. This results in longer RTs compared to the control condition (no cue).

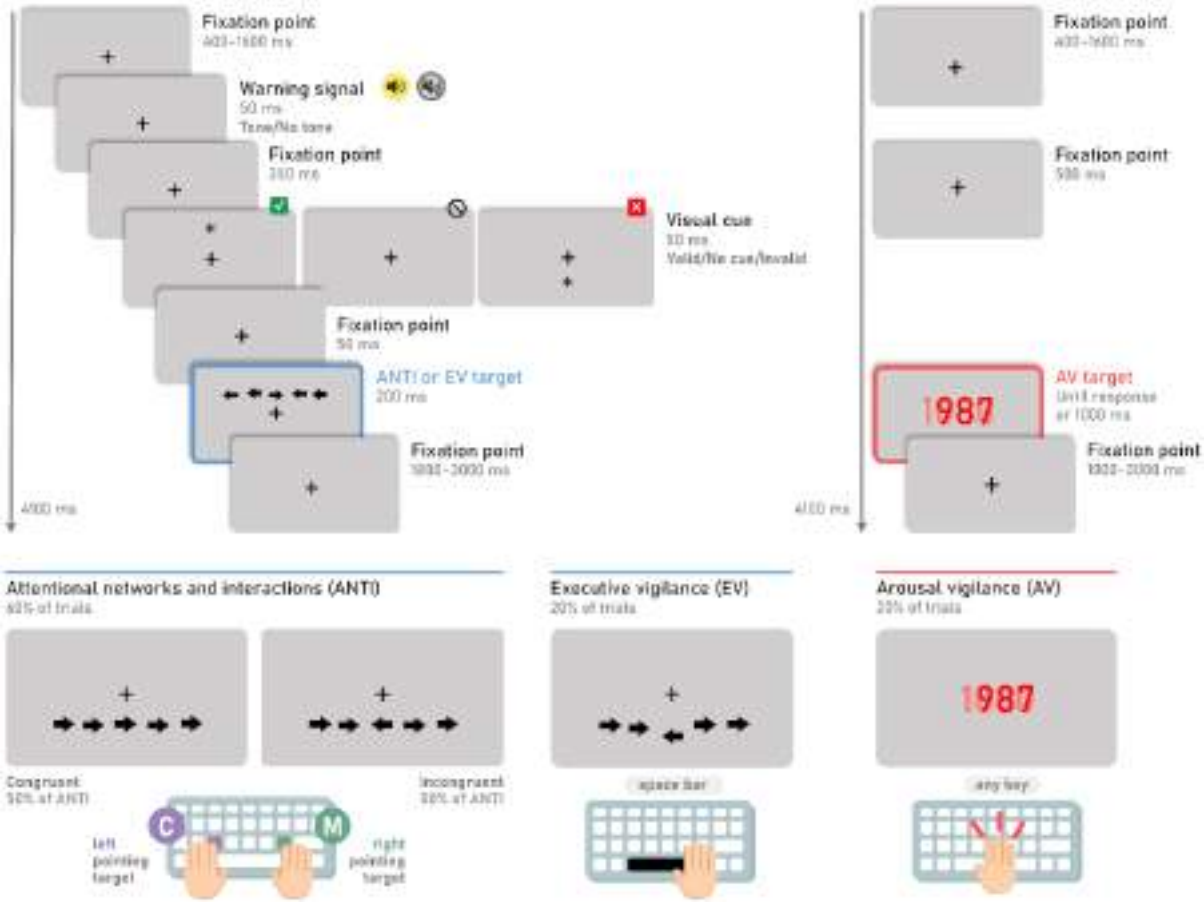


Figure 1. ANTI-Vea trials and correct responses. The top left panel shows the temporal sequence of the ANTI and EV trials. Target and flankers may appear above or below the fixation cross and point to the left or the right side with equal probability. The warning signal appears in half of these trials. The visual cue has an equal chance of appearing in the same location as the target (trial marked with a green checkmark), in the opposite location (marked with a red cross), or not appearing (marked with a barred symbol). The bottom left and middle panels show the correct response based on whether the target is vertically aligned with the flankers (ANTI trials) or displaced (EV trials). The right panels show the temporal sequence and correct response of the AV trials. The duration of each task interval appears next to its corresponding box. Note that, although each trial lasted 4,100 ms, the sequence of events appeared at a variable interval within each trial.

The EV trials (i.e., the trials of the signal-detection subtask of the ANTI-Vea) follow the same procedure and stimuli presentation as the ANTI trials. However, the target appears vertically displaced (either upwards or downwards) from its central position in relation to the alignment with the flankers. In particular, to generate some noise, the five arrows in the ANTI trials and the surrounding arrows in the EV trials can be slightly horizontally and vertically displaced at random by ± 2 px from its central position (see Fig. 1). In the EV trials, the substantial displacement of the target is only vertical and fixed at ± 8 px (see Fig. 1). To complete the EV subtask correctly, participants are instructed to remain vigilant at all times to detect the large vertical displacement of the target and to press the spacebar regardless of the target's direction. Note that the target displacement is considered as the infrequent critical signal of the signal-detection task in the ANTI-Vea. Thus, if participants correctly detect the target's displacement in EV trials, the response is categorized as a hit. If this displacement is not detected (i.e., the spacebar is not pressed), the response is categorized as a miss. Importantly, the ANTI trials serve a dual purpose in the ANTI-Vea: On the one hand, as explained above, they measure the independence and interactions of the classic attentional networks; on the other hand, critically, they serve as the 'noise events' of the signal-detection task of the ANTI-Vea since in these trials the target is not substantially displaced from its central position relative to the distractors. Therefore, if participants press the spacebar in the ANTI trials, the response is categorized as a FA (i.e., an incorrect detection of the infrequent critical signal).

Lastly, for the AV trials (i.e., the trials that mimic the PVT), no warning signal, visual cue, or flankers are presented. Instead, the fixation point remains on the screen until a red millisecond countdown appears at the center of the screen, starting at 1000 and descending to 0 or until a response is executed. Participants are instructed to remain vigilant at all times and to stop the millisecond countdown every time it appears on the

screen as fast as possible by pressing any key on the keyboard. The AV is thus evaluated with the mean and variability of the RT to the countdown.

The three types of trials have the same timing, with each having a total duration of 4,100 ms. Each trial starts with the fixation point on the center of the screen for a random duration between 400 and 1600 ms, continues with a maximum response time of 2000 ms, and ends with the fixation point until the trial duration is reached. This stimulus timing makes participants unable to predict when the target will appear on the screen and which type of trial will be presented. All the stimuli and instructions are presented over a gray background.

The standard ANTI-Vea includes a four-block practice phase, in which instructions and visual feedback are provided so that participants can gradually familiarize themselves with each type of trial. In the first practice block, 16 ANTI trials are presented after the instructions. The second block consists of 32 randomized trials, of which half are EV trials. The third one contains 16 ANTI, 16 EV and 16 AV randomized trials. Finally, the last block includes 40 randomized trials (24 ANTI, 8 EV, and 8 AV) with no visual feedback.

Once participants complete the practice phase, six consecutive experimental blocks are run, without pause and visual feedback. The total time of the experimental blocks is 32 min 48 s for the standard format of six blocks (5 min 28 s per block; 21 min 52 s in the sometimes used four-blocks version). Each experimental block includes 80 pseudo-randomized trials (48 ANTI, 16 EV, and 16 AV). The 48 ANTI trials per block have the following factorial design: Warning signal (no tone/tone) × Visual cue (invalid/no cue/valid) × Congruency (congruent/incongruent) × Target direction (left/right) × Stimuli position regarding the fixation point (up/down). The last two factors are usually not considered for statistical analyses and are only included as control conditions of stimuli presentation. For the EV trials, one factor is added: target displacement direction (upwards/downwards). The 16 EV trials per block are randomly picked out from among the 96 possible ones. For

a better understanding of what this experimental phase looks like, a video is available on the website (direct link at https://videopress.com/v/0hmK7b0Q).

Some versions of the task, or when some parameters are used (see section 5.1. Features and options), other types of trials are added to the task (also randomly within each block of trials). Thus, in the ANTI-Vea-D version of the task, 8 additional trials are added per block in which a salient image of a cartoon character is added to measure distraction by irrelevant but salient information. Similarly, it is possible to add a variable number of thought probes (TP) to each block to measure mind wandering across time on task. In these trials participants have to answer the following question: “Where was your attention just before the appearance of this question?” Participants respond by moving the cursor on a continuous scale ranging from "completely on-task" (extreme left, coded as -1) to "completely off-task" (extreme right, coded as 1). It is possible to select the option of 4, 8 or 12 TPs per block. The presentation of the TP trials is pseudo-randomized, so that there are at least 5 trials of the ANTI-Vea task between TPs.

ANTI-Vea indices

The complex structure and multiple manipulations present in the ANTI-Vea allow for obtaining a wide variety of attentional functioning indices. The core indices of the ANTI-Vea comprise 8 attentional network scores (ANTI) and 10 vigilance scores (EV and AV). These core indices are described in Table 1.

ANTI scores include both mean RT and error rate for the overall ANTI trials, as well as the phasic alertness, orienting, and congruency (i.e., executive control) effects. For RT in ANTI trials, incorrect trials and RTs below 200 ms or above 1500 ms are usually filtered out, which complies with Luna, Roca, et al. (2021). Vigilance scores include both overall performance indices and their decrement slope across task block. The EV measures

Table 1

ANTI-Vea core indices. Adapted from https://anti-vea.ugr.es/analysis.html

Domain	Index	Description	Typical results (in-lab / online version) *M* (*SD*) [a]
Attentional networks (ANTI)	Overall RT	Mean correct RT across all ANTI trials.	629 ms (98) / 652 ms (98)
	Overall errors	Percentage of errors across all ANTI trials.	6.10% (4.74) / 5.95% (4.36)
	Alerting RT	RT difference between *No Tone* and *Tone* conditions in trials with no cue.	40 ms (26) / 37 ms (43)
	Alerting errors	Error difference between *No Tone* and *Tone* conditions in trials with no cue.	2.42% (4.79) / 1.46% (4.75)
	Orienting RT	RT difference between *Invalid* and *Valid* conditions.	40 ms (27) / 46 ms (27)
	Orienting errors	Error difference between *Invalid* and *Valid* conditions.	−0.07% (3.76) / 0.44% (3.98)
	Congruency RT	RT difference between *Incongruent* and *Congruent* conditions.	43 ms (27) / 41 ms (33)
	Congruency errors	Error difference between *Incongruent* and *Congruent* conditions.	0.81% (4.70) / 0.36% (3.88)
Executive vigilance (EV)	Hits	Percentage of times the displacement of the central arrow is correctly detected by pressing the spacebar. Synonymous with 1 minus omission errors or misses.	73.24% (17.34) / 78.87% (14.04)
	Hits slope	Linear slope of hits over blocks, which tends to decrease.	−1.89% (3.64) / −1.93% (3.61)
	False alarms	Percentage of times the spacebar is pressed when there is no substantial displacement of the central arrow. Synonymous with commission errors.	6.35% (5.80) / 6.88% (6.02)
	False alarms slope	Linear slope of false alarms over blocks, which tends to decrease.	−0.27% (0.94) / −0.23% (1.23)
Arousal vigilance (AV)	Mean RT	Average time to stop the red down counter.	491 ms (62) / 509 ms (85)
	Mean RT slope	Linear slope of mean RT over blocks, which tends to increase.	4 ms (11) / 5 ms (14)
	SD RT	Response speed variability to stop the red down counter.	90 ms (39) / 83 ms (32)
	SD RT slope	Linear slope of *SD* RT over blocks, which tends to increase.	4 ms (11) / 6 ms (13)
	Lapses	Percentage of times with an excessively large (RT > 600 ms) or no response to the red down counter.	11.35% (14.57) / 13.19% (17.53)
	Lapses slope	Linear slope of lapses over blocks, which tends to increase.	1.47% (3.32) / 1.67% (3.73)

[a] The content in this column represents the weighted average of the results in Cásedas et al. (2022; online version), Coll-Martín et al. (2021; in-lab version), and Luna, Roca et al. (2021; both lab and online versions), with an overall sample of 427 participants for the in-lab version and 522 participants for the online version. The samples of these studies consisted of university students.

are the percentage of hits and FAs; whereas the AV scores are the mean RT, the standard deviation (SD) of RT, and the percentage of lapses. Note that for FAs only a set of ANTI trials (i.e., ANTI trials with more than 2 px of random noise from the target to at least one of its two adjacent flankers; referred to as the FA difficult column in the trial dataset) are considered. This allows for the emergence of a decreasing trend of FAs across blocks due to the avoidance of a floor effect (Luna, Roca, et al., 2021). The analytical method for computing FAs in a subset of ANTI trials, aiming to avoid a floor effect in FA rate, can be reviewed in detail in (Luna, Barttfeld, et al., 2021).

Reliability of the measures

Table 2 summarizes the findings about the internal consistency scores found for the ANTI-Vea core indices. In terms of internal consistency, a recent study conducted by Luna, Roca, et al. (2021) provides consistent evidence that the ANTI-Vea task (administered either in the lab or as an online session) is roughly as reliable as the ANT (MacLeod et al., 2010) and the ANTI-V (Roca et al., 2011) for the measurement of the classic attentional networks. As for EV and AV, while most of the overall scores (i.e., the average performance on the entire task) showed acceptable internal consistency (i.e., split-half correlations corrected by the Spearman-Brown prophecy > .75) in both lab and online settings (Luna, Roca, et al., 2021), the vigilance decrement scores (i.e., the linear slopes of each vigilance outcome across the six blocks of the task) are substantially less reliable (Cásedas et al., 2022; Coll-Martín et al., 2021; Luna et al., 2022). Even so, these measures of decrement are reliable enough to achieve satisfactory statistical power using large samples (Coll-Martín, Carretero-Dios, & Lupiáñez, 2023), which is more feasible thanks to our platform.

Table 2

Internal consistency scores of the ANTI-Vea indices across studies and task versions

Task index	In-lab reliability (r_{SB})		Online reliability (r_{SB})	
	Luna, Roca, et al. (2021)	Coll-Martín et al. (2021)	Luna, Roca, et al. (2021)	Cásedas et al. (2022)
N	314	113	303	219
Attentional networks				
Overall RT	.99	.99	.99	.99
Overall errors	.92	.91	.89	.91
Alerting RT	.22	.47	.36	.45
Alerting errors	.18	.51	.11	.24
Orienting RT	.31	.36	.30	.40
Orienting errors	.60	.26	.28	.22
Congruency RT	.67	.66	.68	.64
Congruency errors	.66	.60	.52	.51
Executive vigilance				
Hits	.94	.94	.92	.91
Hits slope		.27		.58
False alarms	.85	.85	.79	.78
False alarms slope		.40		.21
Arousal vigilance				
Mean RT	.98	.97	.99	.96
Mean RT slope		.75		.65
***SD* RT**	.84	.88	.76	.71
***SD* RT slope**		.54		.65
Lapses	.96	.96	.98	.96
Lapses slope		.78		.81

Note. r_{SB} = Spearman-Brown split-half reliability coefficient.

Previous studies have also shown that the ANTI-Vea is suitable to be used in repeated sessions, thus supporting the stability of the task' scores. In Sanchis et al. (2020), participants completed the ANTI-Vea in the lab in six repeated sessions. Although some EV and AV scores were modulated by experimental manipulations (i.e, caffeine intake and exercise intensity), most of the task' scores were not modulated in the experimental sessions. To specifically assess the stability of the online ANTI-Vea, we have conducted a pre-registered study in which 20 participants completed the online task across ten repeated sessions (https://osf.io/vh2g9/; Unpublished data, currently in preparation). Preliminary analyses showed that main effects of phasic alertness and executive control were not modulated across sessions. Most importantly, the drop in hits for EV and the increase in mean RT for AV were also not modulated across sessions. Interestingly, as observed in Ishigami and Klein (2010) for the ANT and ANTI tasks, split-half reliability scores of the online ANTI-Vea increase as a function of the number of sessions.

How? Online version

Having explained what the ANTI-Vea is, what it measures, and having demonstrated the reliability of its measures, we will now explain the different characteristics of the platform our team has developed, in which contexts the ANTI-Vea can be applied, and, above all, how to collect, analyze and interpret the data. Note that the current versions of the task on the platform are only available to be administered via computer.

The ANTI-Vea-UGR platform (https://anti-vea.ugr.es) is a research resource offered freely to researchers interested in investigating attention. Different programming languages have been used in its design: JavaScript ES5, HTML5, CSS3, and Angular JS. This allows researchers to freely collect data in the laboratory or online with the available task versions. Additionally, they can download the scripts of these attentional tasks in

different programming languages to adapt or modify the existing versions. Although not typically the case, it should be noted that researchers can choose to administer the online version of the task in a lab setting or to send participants the offline (i.e., downloaded) version of the task for them to run it outside the lab.

It is possible to run the complete ANT-Vea task with the ANTI, EV, and AV trials, or to run the tasks with all stimuli but with participants only having to respond to some specific trial types (ANTI, EV, and AV as single tasks, as well as EV-AV as a dual task). Thus, it is possible to run a version in which participants have to respond just to ANTI trials, thus providing only the main measures of the three attentional networks. Similarly, it is possible to run versions of the task in which, although all trial types are presented, participants have to respond only to either EV trials (SART) or to AV trials (PVT), therefore only providing measures of EV and AV, respectively. In addition, the same task versions are provided, but with only the corresponding trials being presented (i.e., presenting only one specific type of trial; ANTI-Only, PVT-Only, SART-Only-Go, SART-Only-NoGo). Two versions are provided for the SART as a function of whether participants are to respond to all trials except for the displaced arrow trials (SART-Only-NoGo) or to only the displaced arrow trials (SART-Only-Go). Note that the SART and PVT tasks as provided in the ANTI-Vea-UGR platform are adapted versions from the original ones running with the specific parameters of the ANTI-Vea task.

Furthermore, it is possible to run an ANTI-Vea version with 8 additional trials per block in which a salient image of a cartoon character is added to measure irrelevant distraction (ANTI-Vea-D). The addition of the salient image does not seem to affect the measurement of the other attentional indices of the ANTI-Vea (Coll-Martín, Carretero-Dios, & Lupiáñez, 2021, 2023).

Finally, the tasks can be run with the standard parameters (presented in **Table 3**) or with some variations of these parameters (e.g., without practice, with more or fewer blocks of trials, with varying degrees of difficulty, and with longer or shorter stimulus duration).

Although experimental conditions (i.e., environmental noise, luminosity, the device on which the task is run) cannot be controlled as much as in the laboratory (Luna, Roca, et al., 2021), this platform is effectively addressing the growing need for online method administration and self-reporting for the collection of large data samples (Germine et al., 2012). This alternative has great advantages, being easy to use in applied contexts. In terms of time and cost-efficiency, the online version of the task is far less expensive (e.g., no need for a laboratory infrastructure and no need for a person to explain the task individually to each participant), and allows data to be collected from participants from anywhere in the world. It should be emphasized that the online version of the ANTI-Vea has been proven to be as reliable as the standard one in assessing the main effects, the interaction, and the independence of the classic attentional components, along with the overall performance and decrement in EV and AV (Cásedas et al., 2022; Coll-Martín et al., 2021; Coll-Martín, Sonuga-Barke, et al., 2023; Luna, Roca, et al., 2021).

In the studies conducted in our lab/research group, the procedure is conducted under the same conditions for all participants: individually in an experimental room, using headphones, using the same device, and under the same conditions of luminosity, distance from the screen, etc. When participants perform the task online, they can do it from home or from any suitable place of their choice, as long as there is a good internet connection. In order to reduce any distractions along the process, the online version of the ANTI-Vea includes additional instructions at the beginning. Participants are warned that the task will be displayed in full screen and that it is important to complete it without interruptions or pauses, and that any other entertainment devices (TV or radio) have to be turned off. The instructions suggest setting the device volume to 75% and to turn off cell

phones or set them to silent mode. The experimenter may also monitor the session by video call and instruct the participants to ensure the correct understanding and performance of the task. Group sessions can also be conducted using the ANTI-Vea platform, as the online server supports more than one session at a time.

Online version. Multiple versions in different languages

The standard ANTI-Vea and its versions on the platform can be run in six different languages, namely Spanish, English, German, French, Italian, and Polish. This is a remarkable feature, as it allows the user to study the attentional functioning within and across different countries and cultures. We are open to incorporating additional languages to expand the free access of attention assessment into diverse populations.

Features and options

On the website, you can find specific sections with different features of the task. The Home section (**Figure 2A**) presents a brief description of the task, as well as statistics on website visits, years in use, number of participants, published papers related to the task, available languages of the task, and countries using the tool worldwide. The webpage also offers more detailed information about the task and the different ANTI-Vea versions. There is also a How to use it section and other useful menu items such as ANTI-Vea Method, Analysis, For Researchers, Publications, Blog, and Contact.

In the For Researchers section, we provide a system for each user to create a customized and unique link for each participant to access the task. **Table 3** shows all the adjustable setting parameters that can be customized to make the task fit your

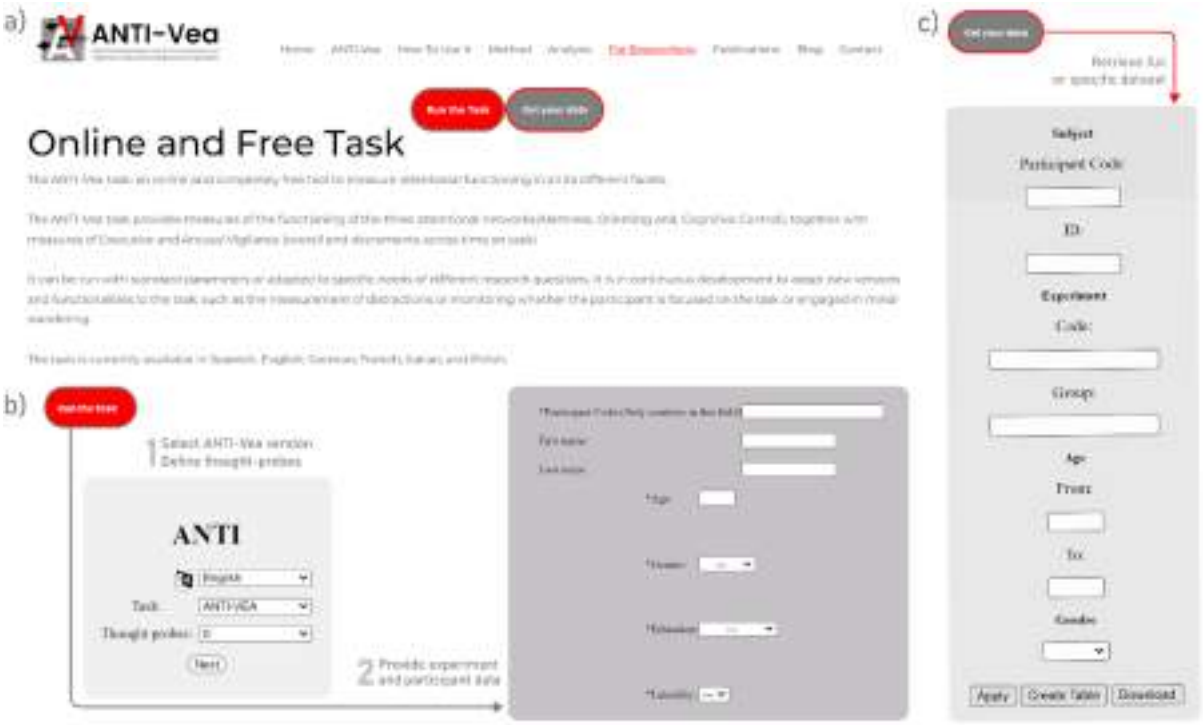


Figure 2. Panel A: Home page of the ANTI-Vea-UGR platform. https://anti-vea.ugr.es/. **Panel B**: Website for data collection for the online version of the ANTI-Vea and other versions and subtasks. Accessed via the "Run the task" button on the ANTI-Vea-UGR platform. **Panel C**: Website for downloading experimental data from the ANTI-Vea Task. Accessed via the "Get your data" button on the ANTI-Vea-UGR platform website.

experimental procedure. Note that this is an advanced option for the data collection process, which will be explained in the next section. This option is useful when the experimenter wants to avoid giving participants control over the selection of task settings. For example, the experimenter may want to ensure that all participants correctly write their code (unique for each participant) and the name of the experiment (the same for all participants in a given study), and perform the practice trials plus four experimental blocks.

Data collection and data protection. Specific know-how information about use of the task in research

On the “How to use it” section of the website, you can find all the know-how knowledge that is necessary to collect data with the task, how to analyze it, and how to interpret it. To collect data with the online ANTI-Vea or any of its versions—regardless of whether you use the online website either inside or outside the lab—, you first need to

Table 3

Setting parameters and all the possible values to create your own link of the task to share with your participants

Setting parameter (parameter=default value)	Description and setting values
lang=en	Language of instructions: “de” for German, “en” for English, “es” for Spanish, “fr” for French, “it” for Italian and “pl” for Polish.
type=ANTI_VEA	Specific task to be performed: ANTI_VEA, ANTI, SART, PVT, SART-PVT, ANTI-Only, SART-Only-Go, SART-Only-NoGo, PVT-Only, ANTI-Vea-D.
pc=1234	Participant code; only numbers allowed here. Any combination of digits is fine. If this parameter is not specified, the task does not start.
exp=Power_ANTI-Vea	The name of your experiment.
gr=Exp	The name of the experimental group, in case there is one.
no=2	Noise; this parameter refers to the random variability of the spatial position of the arrows (1–6); the default value is 2, keep it if you are not interested in this manipulation.
dif=2	Difficulty; this parameter manipulates the perceptual salience of the target and therefore affects EV. It refers to the spatial distance of the central arrow in relation to the adjacent arrows; 1 (most difficult) to 5 (less difficult) values are allowed; the default value is 2, keep it if you are not interested in this manipulation.
st=200	Target display duration; integers from 0 to 1700 ms are accepted values, 200 ms being the value in the standard version of the task.
dP=false	This value should be set to “true” if you want participants to do the whole practice blocks before the experimental blocks, and to “false” if you want them to go straight to the experimental blocks, with just a reminder of the instructions. This feature is useful when collecting data from several sessions in within-subjects designs.
B=6	Number of experimental blocks (1–8); the value can be set to 0 if you want the participants to only run the practice, with no experimental block; 6 is the number of blocks by default.
probes=0	This parameter refers to the number of thought probes (TP) used to measure mind wandering. Depending on the value given to this variable TPs are presented 4, 8 or 12 times per block. By default, the standard version of the task does not include any thought probes. Leave this parameter at 0 to run the standard version of the task, without thought probes.
Link example: https://anti-vea.ugr.es/Sitio_web/ANTI-Vea1/Anti.html?lang=en&type=ANTI_VEA&pc=1234&exp=Power_ANTI-Vea&gr=Exp&no=2&dif=2&st=200&dP=true&B=6&probes=0	

click on the red button “Run the task”. This will bring you to the online website for data collection (**Figure 2B**). There you (or the participants) will have to select the instruction language, the version of the task to be run (default: standard ANTI-Vea), and the number of thought probes to present during the task (default: no thought probes). After clicking on “Next”, the participant’s details have to be entered: Participant Code (only numbers are allowed in this field), First and Last Name (both are optional and will not be visible when you download the data), Age, Gender (“Male”/”Female”), Education (“No education”/”Primary”/”Secondary”/”High School”/”Universitary”) and Laterality (“Left handed”/”Right handed”/”Ambidextrous”). These fields (marked with an asterisk, “*”) must be filled in. On the next page, you have to introduce the Experiment and Group codes. Although these fields are optional, it is very important that you provide a code that can be used later to download the data for your specific experiment. With the “Settings” button you can change the parameters of the task procedure, namely Noise, Difficulty, Stimuli Duration, and Number of Blocks, all with the same values as those shown for the link options (see **Table 3**; but Stimuli Duration is presented here with limited discrete options). In this Settings window you can also unmark the “Do practice blocks” to run the task without practice (only the instructions are presented to remind the participants of the response keys). This is useful when conducting a study in which you manipulate any within-participant variable (e.g., exercise level, time of day, caffeine intake, etc.). In this case, you can ask participants to first perform the whole task in a first familiarization session, and then do it as many times as necessary, according to your within-participant experimental conditions, but without the practice blocks (for an example of this procedure, see Sanchis et al., 2020).

As for the protection of the data, it should be reminded that the provision of identifiable information (i.e., name and surname) is optional and in any case will not be available for download. Furthermore, our platform's server is managed by the University

of Granada, a public institution that adheres to data protection policies and maintains strict ethical guidelines in line with standard academic practices. Researchers can request the removal of their participant data or study information from our database at any time, and participants have the right to request deletion or access to their data. In order to ensure participant's understanding of how their data will be collected and treated, their rights during and after participation, the study's objectives, and any other relevant aspects, researchers must provide them with detailed information in this regard and an informed consent that ensures their understanding and agreement must be signed prior to participation.

Exporting data. Specific know-how information and tools about use and management of the task data in research

Once the data has been collected, if you click on the gray “Get your data” button (located right next to the red “RUN the task” button mentioned in the previous step), you will be able to download your raw data file in CSV format. Here you need to enter the specific details you have used during the collection (typically, the Experiment Code) and click on the “Download” button (**Figure 2C**).

In the raw data file, each row contains the information corresponding to each single trial of the task. The first columns show the participant’s details entered at the beginning (i.e., Participant Code, Age, Gendre, Education, Laterality, Experiment and Experiment Group). Some extra details are provided, such as the Subject ID—identifier automatically generated by the system for each participant,[5] the Session Number (automatically generated by the system based on the Subject ID), and the Session Date (yyyy-mm-dd hh:mm:ss). Following them, you will find the Noise and Difficulty task settings. The

[5] In order for the system to assign the same Subject ID to two or more different sessions, the codes for Participant, Name, Experiment, and Experiment Group must match (in older versions, the access cookies also need to match). Therefore, in studies where participants perform multiple sessions of the task, it is recommended that the experimenter distinguish their participants by using the Participant Code instead of the Subject ID.

following consecutive columns are the specific ones to be used during the analysis: Trial time (the time elapses from the start of the task until the trial begins, in milliseconds), Block (the block number, 0 for practice and 1–n-block for the experimental blocks), Trial number (1 to 16, 32, 40, and 48 for each of the four practice block, respectively; and 1 to 80 for experimental blocks in the ANTI-Vea standard version), Trial type (ANTI, EV or AV in the ANTI-Vea standard version), Reaction time (in milliseconds), Correct answer (C, M, Space or Any), Answer (the keyboard button the participant has pressed), Accuracy (0 or 1, for incorrect and correct trials, respectively), FA Total (if the participant has comitted a FA or not, computed as 1 or 0, respectively), FA difficul (same as FA Total but only computed when there is more than 2 px from the target to at least one of its two adjacent flankers). The next columns describe the characteristics of the stimuli relevant in ANTI or EV trials (i.e., they are not interpretable in AV trials): Target (the direction the target arrow is pointing at; Right or Left), Flankers (the direction the distractors are pointing at; Right or Left); Congruency (Congruent or Incongruous), Cue position (Up or Down), Arrows position (Up or Down), Validity (Valid, Invalid, or No_cue), and Tone (Yes or No). Finally, after some columns with the coordinates of the arrows and others describing characteristics of some subtasks, there is the Task Version column (ANTI_VEA, ANTI, SART, PVT, SART-PVT, ANTI-Only, SART-Only-Go, SART-Only-NoGo, PVT-Only, or ANTI-VEA-D).

Analyzing data. Scripts and tools for analysis of data

In the Analysis section, you will find instructions and tools to analyze your data. Starting from the downloaded raw dataset from the “Get your data” section, the analysis procedure typically begins with a preprocessing phase. Note, however, that what follows is a description of the standard procedure, but researchers may choose to follow alternative analytic strategies depending on their specific research aims. Here, practice

trials are removed and participants with incomplete experimental blocks, task minimizations (i.e., unintentional exits from full-screen task display mode leading to incorrectly registered trials), and poor performance are identified. Note that the raw data allow exclusion thresholds to be chosen based on the characteristics of each particular study (type of participants, design, resource constraints, etc.). In adult community samples, we recommend excluding participants with incomplete blocks or with more than 25% errors in ANTI trials, according to Luna, Barttfeld, et al. (2021). Once the data has been processed, the main analysis consists of obtaining the score of the different indices of the task for each participant.

To support and facilitate the ANTI-Vea analysis process in obtaining the core indices described in **Table 1** we have developed a code in R that is implemented in a Shiny app embedded in the Analysis section of the website (**Figure 3**). This app easily allows the transformation of a raw dataset of the ANTI-Vea into two clean and processed datasets: Data Participant and Data Trial, both in CSV format. In Data Participant each row contains the information of a task session, with the columns including general information about the session (date of the session, noise, difficulty, trials and blocks completed, validity of the performance, etc.) as well as the scores of the ANTI-Vea core indices in that session. Data Trial has the same structure as the raw dataset (i.e., trial-level rows) with additional columns related to the session. To do so, the user does not need any programming knowledge, but only click on the desired options for the following parameters: Task Version, Participant (column used to identify each participant), Administered Blocks per session, Minimal Blocks Completed (sessions with fewer completed blocks are removed), Screen (remove [Full] or retain [Any] sessions in which the screen was minimized by the participant), Validity Performance (remove [Valid] or retain [Any] sessions due to poor performance), Extra Sessions of the Same Participant, and Columns shown in the Data Participants file related to task indices. The website includes sample CSV files for Data

Participant and Data Trial, as well as their corresponding codebooks to ensure they are correctly interpreted.

Finally, the Shiny app includes the option to download a technical report (PDF format) of the whole analysis procedure and summary statistics of the task indices (see the website for a sample report).

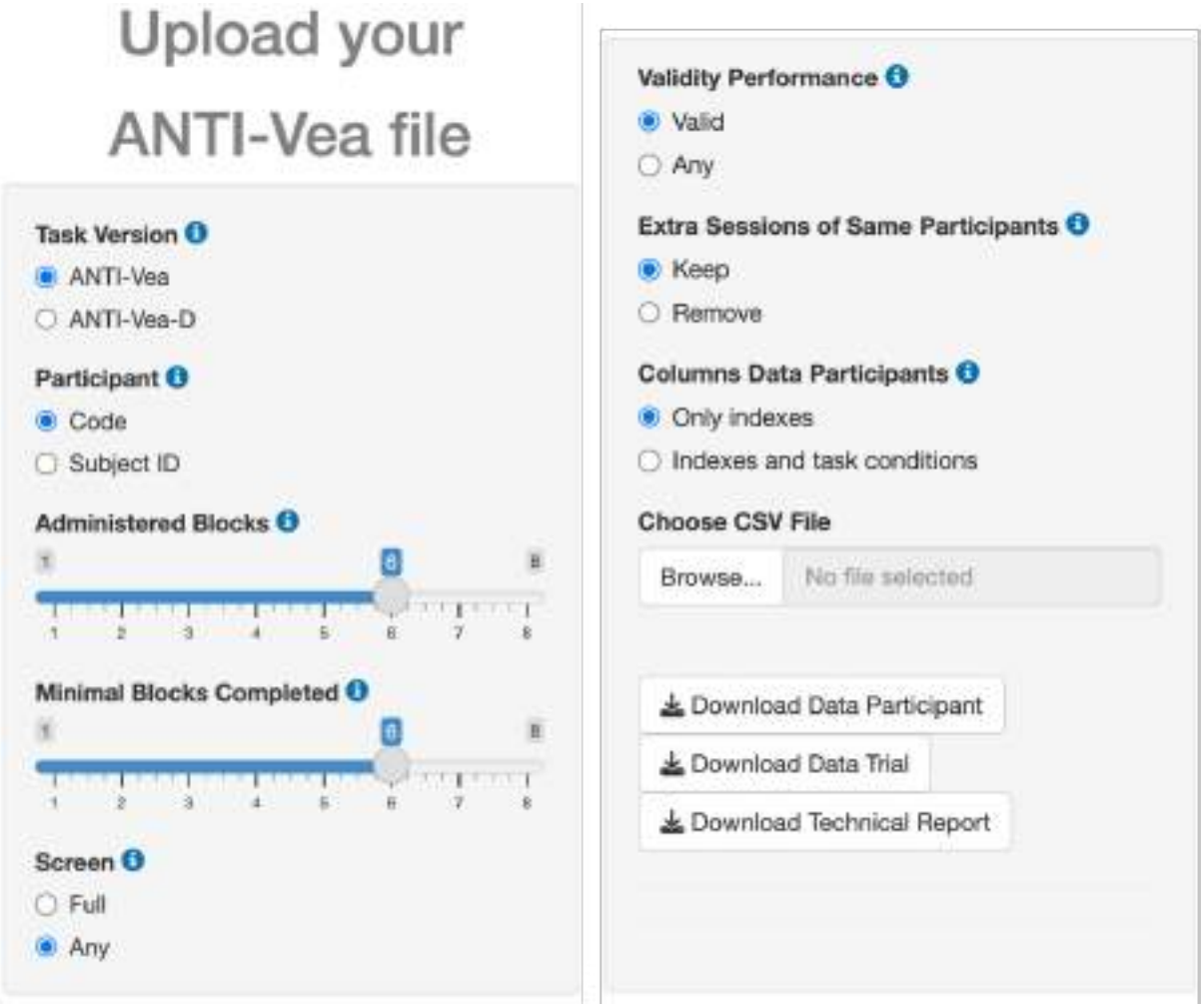


Figure 3. Shiny app to analyze the ANTI-Vea raw data (image adapted from the ANTI-Vea website, Analysis section). For the application to work correctly, it is necessary to set the parameters before uploading the file. Note that the current version of the app does not support ANTI-Vea versions with thought probes.

For those with some programming skills, the R code underlying the Shiny app (default setting) is openly available at the website. This format can be useful for a better understanding of the code and to facilitate modifications in the analysis flow (e.g., different filters and new indices). Indeed, beyond the ANTI-Vea core indices, there are several

outcomes of the task that are worth considering. In this sense, the conditions that are manipulated to obtain the effects of the three attentional networks and the slope of decrement in vigilance can be specifically analyzed for a more detailed analysis (e.g., comparing congruent and incongruent conditions between two groups via a 2 × 2 mixed ANOVA). Having the conditions separated also allows us to check whether the task manipulation worked correctly, although this can also be checked by a one-sample *t*-test on the difference scores or slopes from the ANTI-Vea core indices. Secondly, examples of new indices that have been or may be derived from the core indices are the slope of cognitive control (Luna et al., 2022), mean and variability of RT in EV trials (Sanchis et al., 2020), scores from the Signal Detection Theory (SDT; i.e., sensitivity and response criterion; Luna et al., 2018), sequential effects such as post-error slowing and Gratton effect (Román-Caballero et al., 2021), scores from the psychometric-curve analysis (i.e., scale, shift, and lapse rate; Román-Caballero et al., 2023), between-blocks variability of vigilance scores, and scores from the diffusion decision model (i.e., drift rate, boundary separation, starting point, and nondecision component). We are in the process of implementing these extra scores into the R code. Suggestions for new additions to the code are welcome.

Discussion. Summary of published research with ANTI-Vea

The aim of the present tutorial was to provide a detailed, step-by-step user guide of the ANTI-Vea-UGR online platform (https://anti-vea.ugr.es/index.php), enabling researchers worldwide to collect, download, and analyze data using the ANTI-Vea task (Luna et al., 2018) and its adapted versions.

The ANTI-Vea is the latest version of the attentional networks test for measuring the functioning and interactions of the three attentional networks described by M. I. Posner

and Petersen (1990). It combines different paradigms to assess phasic alertness, orienting, and executive control together. It employs the typical flanker paradigm (Eriksen & Eriksen, 1974) along with the spatial cueing task (M. I. Posner, 1980) and the auditory tone used in the ANTI (Callejas et al., 2004). Moreover, one of the most remarkable contributions of the ANTI-Vea is the theoretical distinction between two components of vigilance: executive vigilance, which refers to the ability to monitor and detect critical signals that rarely occur over a long period of time, and arousal vigilance, understood as the capacity to maintain a fast response to any stimulus in the environment (Luna et al., 2018). Both components had already been described and tested separately with the MCT (N. H. Mackworth, 1948) and the SART (Robertson et al., 1997), for EV; and with the PVT (Dinges & Powell, 1985) for the AV. However, the ANTI-Vea also succeeds in assessing the two vigilance components together in a single session.

When we analyze the vigilance decrement, it manifests as an increase in the average and variability of response time for those trials that evaluate AV and in which the participants have to stop a millisecond backward counter as quickly as possible. In contrast, in EV trials, where the participants have to focus and discriminate the vertical displacement of the central flanker (target), i.e., detect infrequent stimuli, the results show that there is no loss of sensitivity to these infrequent stimuli. What happens rather is that the subject's response bias increases, according to what Thomson et al. (2016) state in their review. The interpretation that has been given to this phenomenon can be debated if we found a floor effect in FAs, an effect that is frequently observed in simple signal detection tasks such as the SART (Luna, Barttfeld, et al., 2021). Nevertheless, to avoid this floor effect in the ANTI-Vea, which is a more complex task, FAs are only computed in those ANTI trials in which a FA response is more likely to be observed.

A number of studies have been carried out since the implementation of this task (Cásedas et al., 2022; Coll-Martín et al., 2021; Feltmate et al., 2020; Hemmerich et al.,

2023; Román-Caballero et al., 2021). Furthermore, the ANTI-Vea itself, or some studies that have used this task, have been featured in different dissemination reports. You can find more in the “Blog” section on the website.

When the participants perform this task online, on their own, there may be some potential difficulties that may cast doubt on the validity of the obtained data. Lighting conditions, distance to the screen, environmental noise, as well as the device features (operating system, screen size, etc.) may vary between participants. In addition, the participants may not understand the instructions and may not perform the task properly. Nevertheless, it is worth mentioning that vigilance has been successfully assessed in some other online studies, in which experimental conditions were not controlled as in typical studies in the lab (Claypoole et al., 2018; Fortenbaugh et al., 2015; Sadeh et al., 2011). Indeed, Luna, Roca, et al. (2021) concluded that the online ANTI-Vea was as effective as the standard ANTI-Vea carried out in the laboratory in assessing the functioning and interactions of the classical attentional components, along with EV and AV decrements. If you would like to monitor the conditions under which participants do the task, even if it takes a bit longer, you can make a video-call to explain previously all the necessary conditions under which they have to perform the task or keep the video-call while the participants carry out the task online from their house or other place meeting the desired experimental conditions (to ensure they do it correctly). Several studies (e.g., Cásedas et al., 2022) have also used the online ANTI-Vea allowing them to reach large samples of participants from remote places and countries. In short, the use of this platform allows the research teams to investigate human attention in a simpler, cheaper, and more accessible way.

Thanks to the versatility offered by our online platform, the task and the different subversions can be applied to explain the variations and the poor functioning of attention in different populations, like attention-deficit/hyperactivity disorder (e.g., Coll-Martín et al.,

2021; Coll-Martín, Sonuga-Barke, et al., 2023) brain-damaged patients (e.g., Narison et al., 2021), to apply it under multiple conditions (e.g., sleep deprivation; McIntire et al., 2017), or states (e.g., fatigued, relaxed, mindful, excited; e.g., Feltmate et al., 2020).

In summary, the online ANTI-Vea task can be run with standard parameters or adapted to the specific needs of different research questions. It is in continuous development to adapt new versions and functionalities to the task, such as the measurement of distractions or monitoring whether the participants are focused on the task or engaged in mind-wandering.

Conclusions

In conclusion, the ANTI-Vea-UGR platform provides a rigorous, accessible, and free assessment of the attentional functioning, encompassing the three attentional networks and two vigilance components. These functions, grounded in influential theoretical frameworks and extensive empirical research, are measured with a reasonable reliability in the ANTI-Vea, the main task of the platform. The resources for online data collection adapted to different languages and analysis through a user-friendly app facilitate task administration by different researchers and in diverse contexts and populations. Finally, the platform's free nature aligns with open science principles, while being supported by a public institution that ensures proper data protection. Therefore, we encourage researchers to take advantage of this valuable resource to advance the study of attention across different areas.

CHAPTER IV:
ATTENTIONAL PROCESSES IN ADHD SYMPTOMS ACROSS DEVELOPMENT

Study 2

Attentional Networks, Vigilance, and Distraction as a Function of Attention-Deficit/Hyperactivity Disorder Symptoms in an Adult Community Sample

This work has been published as:

Coll-Martín, T., Carretero-Dios, H., & Lupiáñez, J. (2021). Attentional networks, vigilance, and distraction as a function of attention-deficit/hyperactivity disorder symptoms in an adult community sample. *British Journal of Psychology*, *112*(4), 1–27. https://doi.org/10.1111/bjop.12513

Abstract

Attentional difficulties are a core axis in attention-deficit/hyperactivity disorder (ADHD). However, establishing a consistent and detailed pattern of these neurocognitive alterations has not been an easy endeavor. Based on a dimensional approach to ADHD, the present study aims at comprehensively characterizing three key attentional domains in ADHD symptomatology: the three attentional networks (alerting, orienting, and executive attention), two components of vigilance (executive and arousal vigilance), and distraction. To do so, we modified a single, fine-grained task (the ANTI-Vea) by adding irrelevant distractors. One hundred and twenty undergraduates completed three self-reports of ADHD symptoms in childhood and adulthood and performed the ANTI-Vea. Despite the low reliability of some ANTI-Vea indices, the task worked successfully. While ADHD symptoms in childhood were related to alerting network and arousal vigilance, symptoms in adulthood were linked to executive vigilance. No association between ADHD symptom severity and executive attention and distraction was found. In general, our hypotheses about the relationships between ADHD symptoms and attentional processes were partially supported. We discuss our findings according to ADHD theories and attention measurement.

Materials, data, and analyses that support the findings of this study are openly available at https://osf.io/k8jdm/

Introduction

Attentional difficulties are one of the core axes in attention-deficit/hyperactivity disorder (ADHD). However, establishing a consistent and detailed pattern of these alterations at the neurocognitive level has not been an easy endeavor, with rather inconsistent and null findings (Huang-Pollock et al., 2005; Huang-Pollock & Nigg, 2003; Wilding, 2005). In the development of translational science, identifying such neurocognitive mechanisms underlying ADHD symptoms is crucial to enhance the approach to the disorder (Castellanos & Tannock, 2002; Luo et al., 2019; Sonuga-Barke & Halperin, 2010). Moreover, recent advances towards a dimensional model of ADHD have led to an interest in studying the neurocognitive correlates of ADHD symptoms in nonclinical community samples (Hilger et al., 2020; Hilger & Fiebach, 2019). Before introducing this dimensional framework underpinning the present study, we will describe the literature on attentional functioning in ADHD, which is mostly built upon case-control designs.

Neurocognitive research on attention in ADHD should be grounded on theoretical frameworks that consider the distinct aspects of attention along with their neurobiological substrates (Booth et al., 2007; Bush, 2010). Different theories have emphasized different aspects of attention, giving rise to a diversity of attentional phenomena that have even led some authors to question the very existence of attention as a consistent phenomenon (Hommel et al., 2019). Alternatively, the three attentional networks model by M. I. Posner and colleagues (Petersen & Posner, 2012; M. I. Posner & Petersen, 1990) tries to solve this problem by considering the attentional system as three independent, albeit interactive, networks, each one implementing a different attentional function. First, the *alerting network* regulates the level of arousal and activation for both momentary readiness to imminent events (phasic alertness) and sustained performance over long time periods

(tonic alertness or vigilance). This network involves noradrenergic innervations from the locus coeruleus towards the frontal and parietal lobes of the right hemisphere. The second subsystem is the *orienting network*, responsible for prioritizing sensory inputs by selecting a modality, spatial location or object. It comprises cortical regions such as parietal cortices and frontal eye fields, and the subcortical structures of pulvinar nuclei and superior colliculi. Finally, the *executive network* is in charge of monitoring performance and prioritizing goal-oriented responses in conflict situations. This third subsystem includes the anterior cingulate and prefrontal regions.

Several tasks have been developed to simultaneously measure these three components of attention, the most common being the Attention Network Test (ANT; Fan et al., 2002; see de Souza et al., 2021, for a review). This computerized task and other variants like the ANTI (Attention Network Test for the interaction; Callejas et al., 2004) presents a sequence of visual stimuli that combines a spatial cueing (M. I. Posner, 1980) and warning signal task with a flanker paradigm (Eriksen & Eriksen, 1974). Subtractions between the tasks conditions resulting from specific manipulations of warning, cueing, and flankers provide the effects of *alerting*, *orienting*, and *congruency* (an index of the executive network), respectively. Different from the ANT, the use of a different cue for measuring alertness and orienting in the ANTI also allows the measure of the interaction between the three attentional networks.

Extensive research has used the ANT/ANTI or some of its variants to analyze the attentional networks in ADHD. A recent meta-analysis including the ANT and the ANT child version (Rueda et al., 2004) compared 491 ADHD children with 402 typical developing controls in nine studies (Arora et al., 2020). They found the functioning of the alerting and executive networks—but not orienting—to be impaired in ADHD. Moreover, Mullane et al. (2011) reported similar group differences using the ANTI. These results support Berger and Posner's (2000) original predictions regarding attentional networks in

ADHD. In the same vein, impaired alerting and executive processes fall in line with energetic (Sergeant, 2000, 2005) and executive (Barkley, 1997) accounts of ADHD (Martella et al., 2020), respectively.

Notwithstanding the numerous studies using the ANT as a tool to characterize the attentional profile of ADHD, some concerns with this literature motivated our work. First, compared to children, the amount of research on ADHD adults and the ANT is somewhat limited (Vázquez-Marrufo et al., 2019). Moreover, this body of research offers mixed evidence about ADHD deficits in alerting and executive networks (Bueno et al., 2015; Hasler et al., 2016; Lampe et al., 2007; Oberlin et al., 2005), with those studies of greater statistical power failing to find differences between ADHD and controls individuals (e.g., (Lundervold et al., 2011). Thus, the functioning of the attentional networks in relation to adult ADHD symptomatology remains unclear. The two remaining issues concern the role of *vigilance* and *distraction* in the ANT/ANTI as well as in the literature of attentional processes in ADHD. The next two sections will address each of them.

Measuring Vigilance in ADHD: A Novel ANT Version

Vigilance, understood as the attentional capacity to maintain performance over time, is one of the most widely studied phenomena in the ADHD literature (Huang-Pollock et al., 2012; Schoechlin & Engel, 2005; Willcutt et al., 2005). The variety of terms and measures linked to vigilance have led some researchers to deem it as a multicomponent concept (Langner & Eickhoff, 2013; Luna et al., 2018; Sturm et al., 1999).

On the one hand, vigilance tasks often consist in detecting an infrequent target among non-target stimuli (e.g., Test of Variables of Attention [TOVA], Greenberg & Waldman, 1993), in line with the Continuous Performance Test (CPT) paradigm, suggesting executive aspects of vigilance (Luna et al., 2018). Substantial research has shown that both ADHD children (Huang-Pollock et al., 2012; 2020) and adults (Advokat et

al., 2007; Barkley & Murphy, 2011; Nikolas et al., 2019; Riccio & Reynolds, 2001; Salomone et al., 2020) exhibit worse performance in numerous CPT indices (i.e., reaction time [RT] mean and variability, hits, false alarms, and *d'*). However, most of these studies only compare overall performance, rather than vigilance decrement over time (i.e., group-by-time interaction), the defining feature of vigilance (Esterman & Rothlein, 2019; Huang-Pollock et al., 2012; L. Tucha et al., 2017). Indeed, research examining such change over time has often failed to demonstrate a greater vigilance decline in ADHD individuals (A. L. Cohen & Shapiro, 2007; Epstein et al., 1998, 2001; Johnson et al., 2001; Solanto et al., 2004; L. Tucha et al., 2009). Only a few studies found that, compared to controls, ADHD participants displayed over time higher variability (Marchetta et al., 2008; Weyandt et al., 2017), more false alarms (L. Tucha et al., 2017), and lower reaction time (Weyandt et al., 2017) or fewer hits (Gmehlin et al., 2016).

Alternatively, vigilance has been operationalized as reactivity to the environment, reflecting tonic arousal levels (Luna et al., 2018; Oken et al., 2006), and measured with tasks demanding fast reactions to stimuli without exerting much control (i.e., without response selection; e.g., the Psychomotor Vigilance Test, Dignes & Powell, 1985). When these tasks are extremely short (≤ 20 trials), no differences between ADHD and controls have been found (L. Tucha et al., 2008, 2009; O. Tucha et al., 2006). Nonetheless, as tasks are longer, some evidence indicates that both children and adults with ADHD show slower RT and higher variability of response (Mary et al., 2016; L. Tucha et al., 2017). Similar to CPT, only a few studies have measured performance over time for this type of vigilance, with ADHD adults exhibiting a greater increase in variability—in terms of standard deviation or lapses, but not in mean RT (Gmehlin et al., 2016; L. Tucha et al., 2017).

Although some efforts have been made to obtain measures of vigilance from the ANT/ANTI in the ADHD literature (Adólfsdóttir et al., 2008; Bueno et al., 2015; Lundervold

et al., 2011), these tasks cannot provide a direct measure of such construct (Roca et al., 2011). A novel version of the ANT has been developed: the *ANT for Interactions and Vigilance—executive and arousal components* (ANTI-Vea; Luna et al., 2018). Grounded on the aforementioned distinction, the ANTI-Vea is suitable to measure the two independent aspects of vigilance besides the three attentional networks and their interactions. To assess executive vigilance (EV), the flanker task is embedded in a CPT structure where participants have to detect a rare target. For its part, arousal vigilance (AV) is measured with a salient stimulus (i.e., a red down counter) that participant must stop as fast as possible. Worthy of note, the length of the task (~ 33 min) enables the analysis of the decrement of both types of vigilance across the six blocks with sufficient precision, and adequate reliability for using the task in experimental designs (Luna, Roca, et al., 2021)

Research on the ANTI-Vea has focused on providing empirical dissociation of and task sensitivity to both vigilance components. In this vein, EV decrement—but not AV—is mitigated by high-definition transcranial direct current stimulation over the right frontal and parietal cortices (Luna, Román-Caballero, et al., 2020) or acute moderate exercise (Sanchis, et al., 2020), and modulated by the cognitive task load (Luna, 2019). Conversely, AV decrement—but not EV—is reduced by acute caffeine intake (Sanchis et al., 2020) and increased with fatigue across 8 hr of testing (Feltmate et al., 2020). Furthermore, the ANTI-Vea has been used to study individual differences related to musical (Román-Caballero et al., 2021) or sport (Huertas et al., 2019) practice as well as mindfulness and mind-wandering dispositions (Cásedas et al., 2022). No previous studies have employed this task in the field of ADHD.

Measuring Distraction in ADHD: A Novel Paradigm

Although distraction is central to ADHD symptomatology, evidence of increased distractor interference in ADHD is rather inconsistent (Albrecht et al., 2008; Brodeur &

Pond, 2001; Chan et al., 2009; Huang-Pollock et al., 2005; Lundervold et al., 2011; Mason et al., 2004; Wilding, 2005). Forster (2013) pointed out that this literature failed in the attempt to employ a paradigm with distractors that were entirely irrelevant to the task. For instance, in the response-competition paradigm (e.g., flanker tasks), although distractors appear in an irrelevant location where the target is never presented, their identity is highly relevant to the task, as it is associated with one of the target responses (i.e., congruent vs. incongruent). This does not reflect the type of distraction that interferes with people—mostly those with ADHD—in daily life, where the distractor (e.g., a mobile notification) is entirely unrelated to the task being performed (e.g., reading a paper).

Therefore, to measure task-irrelevant distraction, distractors must be presented in an irrelevant location, unrelated to any task responses, visually dissimilar from the search stimuli, and irrelevant to any attentional setting for the current task (Forster, 2013). In line with this, Forster and Lavie (2008) designed the *irrelevant-distractor paradigm* to measure the interference associated with the peripheral presentation of a colorful salient task-irrelevant distractor, typically a well-known character (e.g., Pikachu). Using this paradigm, ADHD adults exhibited higher irrelevant distraction than controls (Forster et al., 2014). Crucially, Forster and Lavie (2016) found that while interference from irrelevant distractors correlated positively with ADHD symptoms in nonclinical adults, interference from response-competition distractors did not.

Since the ANTI-Vea measures interference by a response-competition paradigm (i.e., flanker task), it may be possible that integrating the irrelevant-distractor paradigm could enhance the task sensitivity to ADHD symptoms.

A Dimensional Model of ADHD

Classical disease models and diagnostic systems have conceptualized mental disorders as discrete categories qualitatively different from normality. Nevertheless,

converging evidence at behavioural (Haslam et al., 2006), neurocognitive (Frazier et al., 2007), and genetic (Gjone et al., 1996) levels support a *dimensional* rather than a categorical structure of ADHD. A dimensional model posits continuity in symptoms and underlying causes, so that ADHD would be viewed as an extreme expression of normal variation in the population (Coghill & Sonuga-Barke, 2012; Sonuga-Barke, 2013). This approach opens up new opportunities to ADHD-related research.

On the one hand, neurocognitive ADHD theories could serve to explain symptom-level variation in nonclinical or community samples (Hilger et al., 2019, 2020). Conversely, research on neurocognitive correlates of ADHD symptom severity in community samples might shed light on processes likely to be altered in ADHD (Coghill & Sonuga-Barke, 2012). For example, impaired vigilance (Craig & Klein, 2019) and higher irrelevant distraction (Forster & Lavie, 2016) positively correlated with ADHD symptoms in nonclinical samples (but see Craig & Klein, 2019, and Zamani Sani et al., 2020, for null findings on attentional networks). However, unless a substantial number of individuals with ADHD are included in community samples, these correlational designs might only offer preliminary or indirect insights about the disorder, which need to be confirmed in clinically referred samples. Worthy of mention, even subclinical variations in ADHD symptoms have been associated with negative family impact, psychosocial problems, and poorer satisfaction with life (Cussen et al., 2012; Gudjonsson et al., 2009).

The Present Study

The aim of our study was to investigate the main attentional processes related to ADHD symptoms through a single, fine-grained task. For that purpose, we integrated the irrelevant-distractor paradigm into the ANTI-Vea. This allows simultaneous measures of the attentional networks, vigilance, and distraction, three key domains in the field of attention and ADHD. To characterize ADHD symptoms, we employed a community sample of undergraduates, and both childhood and current symptoms were evaluated.

Grounded on the aforementioned literature, we expected higher ADHD symptoms to predict: (a) poorer functioning in alerting and executive networks (i.e., higher effects), but not in orienting; (b) impoverished EV and AV—crucially in performance over time (i.e., vigilance decrement); and (c) a higher irrelevant-distraction effect.

Methods

Participants

Following the reference work by Forster and Lavie (2016), we decided to collect data from 120 participants. This sample size allows the detection of a small to medium effect size (r = .22; smaller than r = .32, observed by Forster and Lavie, 2016) in one-tailed, zero-order correlations with 1 – β = .80 and α = .05, as computed with G*Power 3.1. Therefore, a sample of 120 undergraduates from a Spanish university participated in the study. They received extra credit course as a compensation for their voluntary participation. All participants (97 women, 23 men; age, M = 20.21, SD = 1.91, range 18–28) were Spanish-speaking and had a normal or corrected-to-normal vision. Two participants reported a prior diagnosis of ADHD. All participants completed an informed consent form. The study was conducted in accordance with the guidelines laid down by our institutional ethics committee, in compliance with the ethical standards of the 1964 Declaration of Helsinki and was part of a larger research project approved by our institutional ethics committee.

Instruments

Barkley Adult ADHD Rating Scale-IV: Childhood and Current Symptoms

The self-reports of the Barkley Adult ADHD Rating Scale-IV (BAARS-IV; Barkley, 2011) include two scales to assess ADHD symptoms: retrospectively in childhood (cBAARS-IV) and concurrently in adulthood (aBAARS-IV). Each scale is composed of 18

items, nine of inattention (e.g., “forgetful in daily activities) and nine of hyperactivity-impulsivity (e.g., “fidget with hands or feet or squirm in seat”), in a Likert scale ranged from 1 (*never or rarely*) to 4 (*very often*). Since the items are based on the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; DSM-IV; American Psychiatric Association [APA], 1994), we used the Spanish version of the manual for the translation (APA, 1994/1995). In our sample, reliability was α = .89 and α = .86 for cBAARS-IV and aBAARS-IV, respectively, close to the α = .95 and α = .92 of the original BAARS-IV (Barkley, 2011). Barkley proposed the 95th percentile as a cutoff to identify individuals at high risk of ADHD.

Adult ADHD Self-Report Screening Scale for DSM-5

The Adult ADHD Self-Report Screening Scale for DSM-5 (ASRS-5; Ustun et al., 2017) specifically assesses the adult presentation of ADHD based on DSM-5 conceptualization (APA, 2013). It includes six items (e.g., “how often do you put things off until the last minute”) in a 5-point Likert scale (0 = *never* to 4 = *very often*). Items 1 to 4 had been adapted into Spanish from a previous versions of the ASRS (Sanchez-Garcia et al., 2015). For items 5 and 6, we used the forward translation of the ASRS-5 from a Spanish journal specialized in health sciences (Redacción Médica, n.d.). Then, both items were back-translated into English, where no discrepancies where found. Reliability of ASRS-5 in our sample was α = .64, which is within the range of the original study (Ustun et al., 2017), in which a threshold of 14 points was established as preferred for screening purposes.

ANTI-Vea With Irrelevant Distractors

The original ANTI-Vea (Luna et al., 2018; see online version on https://www.ugr.es/~neurocog/ANTI/), which evaluates the three attentional networks (ANTI trials) and two types of vigilance (EV and AV trials), was modified in order to add

the irrelevant distractor paradigm on the task (ID trials). Everything was used as in the original task, except that 8 ID trials were added to each of the 6 bocks of trials. These trials were built as ANTI trials (see below), but with the replacement of nontarget arrows by lines, and the inclusion of a completely irrelevant distractor.

Procedure

The study was conducted between November 2019 and March 2020—before COVID-19 preventive measures were implemented in our region. First, participants filled out an online survey—via LimeSurvey (https://www.limesurvey.org)—composed of questionnaires about attention and distraction dispositions.[6] The survey began with the cBAARS-IV, the aBAARS-IV, and the ASRS-5, in that order; and ended with a question about previous diagnosis of ADHD. After completing the survey, participants were invited in our laboratory to conduct the cognitive task.

Upon arrival at the laboratory, participants were individually brought into a soundproof room adequately illuminated. Participants were sitting at about 60 cm from a 15 inches computer screen with an aspect ratio of 16:9. Participants were provided with headphones at 60% sound level of the computer and were asked to turn-off or silence their mobile phone. Then, the experimenter presented the ANTI-Vea, designed and run in E-Prime (Version 2.0; Psychology Software Tools, Inc., 2012). The stimuli sequence and correct responses for each type of trial are depicted in **Figure 1**.

All the trials lasted 4100 ms and had a fixation point constantly present at the center of the screen. The ANTI-Vea comprised four different types of trials: the three from the original task (ANTI, EV, and AV) and one added to measure irrelevant distraction (ID). Trials were pseudorandomly presented within their experimental block. In ANTI (~54%; 48 trials per block) and EV trials (~18%; 16 trials per block) an auditory warning signal

[6] The full set of questionnaires, which is part of a larger project, is available at a public repository (https://osf.io/k8jdm/).

sounded in half of the trials (*tone condition*), whereas in the other half no warning signal was presented (*no tone condition*). Next, an asterisk (i.e., visual spatial cue) appeared in two third of the trials, equally presented in the same (*valid condition*) or the opposite

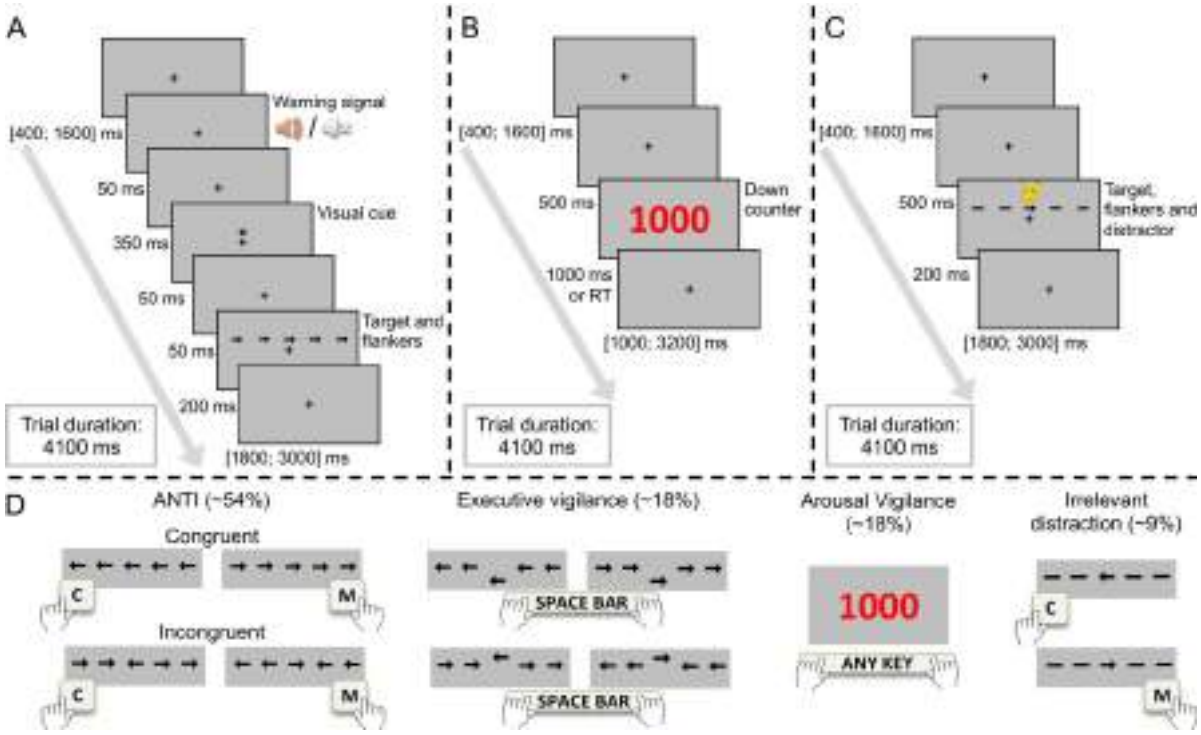


Figure 1. Attention Network Test for Interaction and Vigilance—Executive and Arousal Components (ANTI-Vea) Procedure in our Study. Panel A: Temporal sequence in Attention Network Test for Interaction (ANTI) and Executive Vigilance (EV) trials. Target and flankers could appear above (see example) or below the fixation point. Visual cue could appear in the same location as the target (valid cue; see example), in the opposite location (invalid cue), or could not appear (no cue). **Panel B**: Temporal sequence in Arousal Vigilance (AV) trials. **Panel C**: Temporal sequence in Irrelevant distraction (ID) trials. Target and flankers could appear above (see example) or below the fixation point. Irrelevant distractor could appear at the top (see example) or at the bottom of the screen; or it could not appear. Distractor could be Pikachu (see example), SpongeBob, or Mickey Mouse. **Panel D**: Correct responses for each type of trial. The five arrows are randomly displaced ± 2 px to generate noise in ANTI and ID trials, and the target is displaced by 8 px in EV trials.

(*invalid condition*) location as the upcoming target. A central arrow (i.e., target) with four flankers appeared 100 ms later either above or below the fixation point. In ANTI trials, participants had to discriminate the direction of the target (by pressing either “c” for leftward direction, or “m” for rightward direction) while ignoring the direction of the flanking arrows, which could equally point to the same (*congruent condition*) or the opposite

(*incongruent condition*) as the target. In contrast, on EV trials the target appeared vertically displaced for participants to detect the displacement by pressing the space bar. In contrast, AV trials (~18%; 16 trials per block) only displayed a red millisecond down counter at a variable time interval (900–2100 ms) for participants to stop the it by pressing any key as fast as possible. Finally, ID trials (~9%; 8 trials per block) had the same structure and correct response as ANTI trials without tone or cue, except: (a) nontarget arrows were replaced by lines to reduce perceptual load, and (b) in half of the trials an irrelevant distractor (SpongeBob, Pikachu, or Mickey Mouse; ~200 px width × ~200 px height) appeared either at the top or at the bottom of the screen (above ~150 px- or below ~290 px- the central arrow) for the same time as the target (*distractor present condition*), whereas no distractor was presented in the other half (*distractor absent condition*).

The ANTI-Vea task started with several phases of progressive practice, as in Luna et al. (2018), with the addition of 8 ID trials in a last practice block of 48 randomized trials (24 ANTI, 8 EV, 8 AV, 8 ID) without visual feedback. Before this practice block, the three type of distractors were shown to participants, who were told to “ignore them for being irrelevant to the task goal”. After this block, participants were given the possibility to search for and ask any questions to the experimenter, who had left the room at the beginning of the practice phase. Then, participants started the six seamless experimental blocks (48 ANTI, 16 EV, 16 AV, 8 ID trials per block). The whole experimental session—instructions and task—lasted ~50 min.

Data Analysis

Behavioural data were treated based on Luna et al. (2018) through an R script. Because of a computer or experimenter error, ANTI-Vea data from three participants were corrupted and they could not be analyzed. Participants with more than 25% errors in ANTI trials (n = 4, among them, one of the two participants with ADHD) were excluded from all task analyses, and those remaining participants with more than 25% errors in the

distractor present condition (n = 11) were excluded from all ID trials analyses.[7] For ANTI and ID RT analyses, trials with incorrect responses (ANTI = 5.75%; ID = 5.68%) and RTs smaller than 200 ms (ANTI = 1.24%; ID = 1.96%) or higher than 1500 ms (ANTI = 0.45%; ID = 0.78%) were excluded.

We extracted several measures from the ANTI-Vea. For *mean RT* and *percentage errors* in ANTI trials, we calculated the *overall mean score* and difference scores for *alerting* (no tone – tone conditions[8]), *orienting* (invalid – valid conditions), and *congruency* (incongruent – congruent conditions). Following Luna, Barttfeld, et al. (2021), EV outcomes included *hits* (percentage of correct responses in EV trials), *false alarms* (percentage of space bar responses in ANTI trials with more than 2 px from the target to at least one of its two adjacent flankers), and the signal detection theory metrics of *A′* (sensitivity) and *B″* (response bias). AV outcomes compressed the *mean RT*, the *standard deviation RT*, and the *percentage of lapses* (RTs > 600 ms). Each EV and AV outcome included both the *overall performance* and the *slope* of the regression line—representing performance over the six experimental blocks. Finally, ID trials provided interference from irrelevant distractor. As per Forster and Lavie (2016), we computed the *percentage increase in mean RT* due to distraction by dividing the difference score (distraction present – distractor absent conditions) by RT in the distractor absent conditions. Distraction interference in *percentage errors* only employed raw difference scores.

We analyzed the quality of the ANTI-Vea measures. First, we checked the task functioning. To this end, we conducted Student's t-tests for indices based on difference scores. For indices based on performance over experimental blocks (i.e., EV and AV slopes), we conducted six-level one way repeated-measures analyses of variance

[7] This filter for (ID) trials was added in response to the first data analysis, due to the extremely high percentage errors of these participants in the distractor present condition (Mdn = .94). Most of them probably understood that "ignore the distractors" meant "do not response when the distractor appears".
[8] Although the measure exclusively considering the no-cue conditions is a purer measure of alertness, the measure considering all conditions is more powerful and reliable (de Souza et al., 2021).

(ANOVAs) with planned comparisons to test the polynomial linear component. Where appropriate, Huynh-Feldt or Greenhouse-Geisser corrections were applied. Second, we estimated the reliability of each ANTI-Vea outcome. To do so, we used a permutation-based split-half correlation approach with 10,000 random splits and then applied the Spearman-Brown correction (for a rationale, see Parsons et al., 2019). These reliability estimations were computed by adapting an R script that had previously been used with the original ANTI-Vea (Luna, Roca, et al., 2021).

Finally, we used JASP (Version 0.13; JASP Team, 2020) to test the correlations between the three questionnaires of ADHD symptoms (i.e., cBAARS-IV, aBAARS-IV, and ASRS-5) and the 24 ANTI-Vea outcomes (8 ANTI, 8 EV, 6 AV, 2 ID). Normality was violated for the vast majority of pairwise comparisons, as assessed by Shapiro-Wilk tests. Therefore, we used the Kendall's τ rank correlation coefficient, interpreted as per Gilpin (1993): .07 = small, .21 = medium, .35 = large. We conducted one- or two-sided contrasts according to whether they were based on directional or nondirectional hypotheses. Statistical significance was set at $\alpha = .05$.

Results

ADHD Self-Reports

Figure 2 shows the distribution of ADHD symptoms compared to an estimated normative sample (for a detailed procedure and statistical report, see **Supplemental Text 1**). Taking together, although ADHD symptom distributions in our sample might slightly differ from the population, this does not seem to undermine its spread and variability throughout each scale, as compared to an estimated normative sample.

Unsurprisingly, the cBAARS-IV ($M = 29.6$, $SD = 8.46$), the aBAARS-IV ($M = 28.4$, $SD = 7.32$), and the ASRS-5 ($M = 8.04$, $SD = 3.54$) showed significant positive correlations

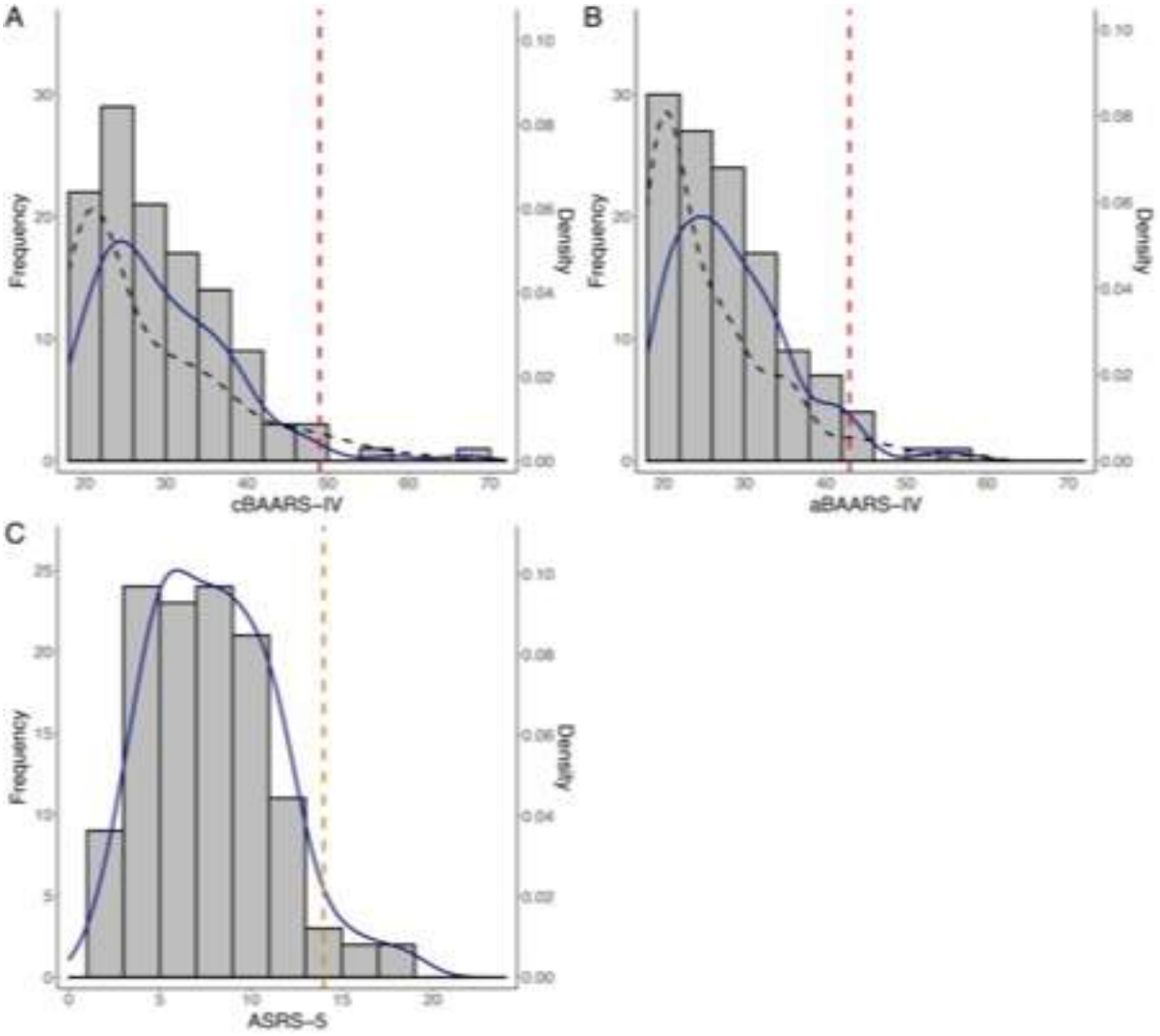


Figure 2. Distribution of Total ADHD Symptom Scores for Each of the Three Scales Compared to an Estimated Normative Sample. *N* = 120. ADHD = Attention-deficit/hyperactivity disorder. Histogram and blue solid line represent the frequency and density curve of ADHD total scores in the study sample. Dashed black lines represent the density curve of ADHD total scores in an estimated normative sample. This normative, equally-sized sample was obtained by extracting 120 quantiles form a large bootstrapped sample (N = 10,000) that fits the percentile values available in Barkley (2011). Vertical dashed red lines represent the normative 95th percentile, a cutoff to identify individuals at high risk of ADHD. The vertical dashed orange line represents a threshold for ADHD screening purposes. Panel A: cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms. Panel B: aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms. Panel C: ASRS-5 = Adult ADHD Self-Report Screening Scale for DSM-5.

among them, with effect sizes from medium to large. Concretely, for the cBAARS-IV with the aBAARS, $r(118) = .51$, $p < .001$, for the cBAARS-IV with the ASRS-5, $r(118) = .35$, $p < .001$, and for the aBAARS-IV with the ASRS-5, $r(118) = .70$, $p < .001$. Interestingly, the correlation between the two measures of symptoms in adulthood was higher than those between these measures and the one of symptoms in childhood.

ANTI-Vea

Table 1 shows descriptive statistics, reliability, and correlations with ADHD symptoms for each of the ANTI-Vea indices. Correlations among ANTI-Vea indices are presented in **Supplemental Table 2**.

ANTI Outcomes

As reported by Luna et al., (2018), ANTI trials revealed effects of alerting, orienting and congruency for RTs and, except orienting ($p < .077$), for percentage errors. Specifically, RTs were faster in the tone than in the no tone trials, $t(112) = –9.18$, $p < .001$, $d = –0.84$, in valid than invalid trials, $t(112) = –14.45$, $p < .001$, $d = –1.36$, and in congruent than incongruent trials, $t(112) = –14.80$, $p < .001$, $d = –1.39$. Percentage errors were higher in no tone than in tone trials, $t(112) = 6.54$, $p < .001$, $d = 0.62$, and in incongruent than congruent trials, $t(112) = 3.69$, $p < .001$, $d = 0.35$. Reliability of ANTI outcomes ranged from $r_{SB} = .26$ to $r_{SB} = .99$, with the usual higher values for overall than for difference scores (see **Table 1**).

In line with our hypotheses, we observed significant positive correlations between the cBAARS-IV and the magnitude of the alerting effect (i.e., the difference between no tone and tone trials) in both RTs, $\tau(111) = .13$, $p = .021$, and percentage errors, $\tau(111) = .15$, $p = .013$. Such correlations were not significant for the aBAARS-IV (both $p > .063$) and the ASRS-5 (both $p > .248$). Contrary to our predictions, none of the three ADHD symptom self-reports significantly correlated with the overall scores of RT (all $p > .193$) or percentage errors (all $p > .186$) nor with the congruency effect, either measured with RTs (all $p > .205$) or percentage errors (all $p > .314$). Finally, as expected, orienting indices of RT (all but one $p > .804$) and percentage errors (all $p > .085$) did not correlate with any ADHD symptom self-report.

Table 1

Descriptive Statistics, Reliability, and Kendall's Rank Correlations With ADHD Symptoms in Childhood and Adulthood for all ANTI-Vea Outcomes

				Kendall's τ correlation coefficient		
ANTI-Vea index	***M***	***SD***	r_{SB}	**cBAARS-IV**	**aBAARS-IV**	**ASRS-5**
ANTI outcomes						
RT overall	600	95	.99	.06	–.08	–.03
% errors overall	5.75	4.34	.91	.06	–.08	–.06
RT alerting	20	23	.47	.13*	.10	.03
% errors alerting	2.33	3.79	.51	.15*	.09	.05
RT orienting	35	26	.36	.01	.02	.11
% errors orienting	0.65	3.90	.26	.04	–.01	–.05
RT congruency	40	28	.66	.05	.03	.03
% errors congruency	1.46	4.21	.60	.03	.01	–.06
EV outcomes						
% hits	68.62	17.29	.94	.001	–.05	.01
% false alarms	5.16	5.09	.85	–.001	–.11	–.02
***A'* (sensitivity)**	.90	.04	.88	–.01	–.02	.02
***B"* (response bias)**	.59	.35	.86	–.02	.11	.02
% hits slope	–1.74	3.00	.27	–.02	–.04	–.11*
% false alarms slope	–0.42	1.51	.40	.05	.09	.11*
***A'* (sensitivity) slope**	–.003	.01	.40	–.03	–.09	–.14*
***B"* (response bias) slope**	.04	.10	.26	–.01	–.11	–.08
AV outcomes						
RT mean	504	58	.97	.11*	.04	.10†
RT standard deviation	97	49	.88	.11*	.06	.01
% lapses	12.98	14.35	.96	.11*	.07	.08
RT mean slope	5.36	12.47	.75	.17**	.06	–.01
RT *SD* slope	6.10	12.62	.54	.08	.02	–.05
% lapses slope	1.99	3.74	.78	.18**	.06	–.01
ID outcomes [a]						
% interference in RT	5.37	7.35	.21	.02	.02	.03
% errors interference	0.61	6.49	.03	–.01	.02	.05

Note. r_{SB} = Spearman-Brown split-half reliability Note. *n* = 113. ADHD = Attention-deficit/hyperactivity disorder. r_{SB} = Spearman-Brown reliability coefficient. cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms. aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms. ASRS-5 = Adult ADHD Self-Report Screening Scale for DSM-5. ANTI = Attention Network Test for Interaction. EV = Executive vigilance. AV= Arousal Vigilance. ID = Irrelevant distraction. RT = Reaction time. According to our hypotheses, correlation tests are one-tailed for positive correlations in all indices, except: (a) orienting (RT and errors; two-tailed); (b) hits and *A*' (both overall and slope; one-tailed for negative correlations); and (c) *B*" (only overall; two-tailed), as it is the only index not directionally associated with performance in vigilance.

[a] *n* = 102.

*p < .05, one-tailed. **p < .01, one-tailed. No other p < .05 appeared with exploratory two-tailed tests.

EV Outcomes

The four EV indices of overall performance (i.e., hits, false alarms, *A′*, and *B″*) yielded high reliability scores, from r_{SB} = .85 to r_{SB} = .94 (see **Table 1**). However, none of these indices showed significant correlations with any of the three ADHD symptom self-reports (all but one $p > .077$).

Consistently with Luna et al., (2018), we found a main effect of experimental block for hits, $F(5, 560) = 8.85$, $p < .001$, $\eta^2 = .07$, false alarms, $F(4.51, 505.16) = 2.56$, $p < .032$, $\eta^2 = .02$, and *B″*, $F(4.79, 536.58) = 4.13$, $p < .001$, $\eta^2 = .04$. Planned comparisons revealed a linear component indicating that, over the six blocks, there were a decrement in the percentage of hits, $t(560) = –6.27$, $p < .001$, and false alarms, $t(112) = –2.94$, $p = .004$, as well as an increase in *B″*, $t(112) = 4.01$, $p < .001$. Different from Luna et al., we also observed the block effect on *A′*, $F(4.28, 478.82) = 2.91$, $p < .019$, $\eta^2 = .03$, yielding a linear decrease over the blocks, $t(112) = –3.13$, $p = .002$. These indices of slope exhibited a low reliability, ranging from r_{SB} = .26 to r_{SB} = .40.

Concerning our hypotheses, only the ASRS-5 correlated with three indices of EV slopes. Specifically, higher ASRS-5 scores predicted a greater decrement in percentage of hits, $\tau(111) = –.11$, $p = .044$, and *A′* (sensitivity), $\tau(111) = –.14$, $p = .017$, as well as a more attenuated decrement in percentage of false alarms, $\tau(111) = –.11$, $p = .044$. The remaining correlations were not significant (all $p > .085$).

AV Outcomes

Similar to EV, we found high reliability for the three AV indices of overall performance, oscillating between r_{SB} = .88 and r_{SB} = .97 (see **Table 1**). As predicted, the cBAARS-IV exhibited significant positive correlations with the three indices, namely, mean RT, $\tau(111) = .11$, $p = .043$, standard deviation of the RT, $\tau(111) = .11$, $p = .044$, and

percentage of lapses, τ(111) = .11, p = .041. Neither the aBAARS-IV nor the ASRS-5 significantly correlated with any AV index (all p > .061).

In line with Luna et al., (2018), there was a main effect of experimental block for mean RT, F(3.94, 441.51) = 8.47, p < .001, η^2 = .07, standard deviation of the RT, F(4.18, 468.41) = 7.46, p < .001, η^2 = .06, and percentage of lapses, F(3.46, 387.16) = 14.38, p < .001, η^2 = .11. All these variables increased linearly across the blocks, namely, mean RT, t(112) = 4.56, p < .001, standard deviation of the RT, t(112) = 5.13, p = .001, and percentage of lapses, t(112) = 5.68, p < .001. Reliability for the three indices of slope ranged from r_{SB} = .54 to r_{SB} = .78.

Like for AV overall performance, only the cBAARS-IV exhibited significant correlations with indices of AV slopes, concretely, with the slope of mean RT, τ(111) = .17, p = .004, and the slope of percentage of lapses, τ(111) = .18, p = .002; but not with the slope of standard deviation of the RT (p = .099). No significant correlations were found between the two other self-reports (i.e., the aBAARS-IV and the ASRS-5) and the three measures of AV slope (all p > .169).

ID Outcomes

In the same vein as Forster and Lavie (2016), participants were slower in the presence (M = 640, SD = 103) versus in the absence (M = 608, SD = 103) of the irrelevant distractor, t(101) = 7.14, p < .001, d = 0.71. Nevertheless, both conditions did not significantly differ in the percentage of errors, t(101) = 0.95, p = .342, d = 0.09. Reliability for indices of percentage increase in mean RT (r_{SB} = .21) and percentage errors (r_{SB} = .03) were found to be low. Contrary to our predictions, none of the three self-reports correlated with either percentage increase in mean RT (all p > .310) or percentage errors (all p > .240).

Discussion

This study aimed at analyzing the main attentional processes related to ADHD symptoms, namely, attentional networks, executive and arousal vigilance, and distraction. To do so, we modified a single, fine-grained task (i.e., the ANTI-Vea) to add a distraction component (Forster & Lavie, 2016). Based on a dimensional model of ADHD, we employed a community sample of undergraduates and measured retrospective and current subjective ADHD symptoms. Although the ANTI-Vea worked successfully, the reliability was reduced for many indices. A significant relation was observed between ADHD symptoms and a higher alerting effect, but not orienting or congruency effects. ADHD symptom ratings also related to a poorer performance over time in EV and to alterations in different AV measures. No association was found between ADHD symptoms and irrelevant distraction. Worthy of note, our pattern of results was not consistent across the three ADHD symptom self-reports or the specific task indices. Therefore, our hypotheses were supported only partially. These findings have implications for the neurocognitive mechanisms of ADHD symptoms and for the role of the ANTI-Vea in this literature.

Attentional Networks

In line with our hypothesis, the finding of a higher alerting effect associated with ADHD symptoms is consistent with Berger and Posner's (2000) predictions. It also fits the state regulation deficit account of ADHD (Sergeant, 2000, 2005; Sonuga-Barke, Wiersema, et al., 2010). From this view, a task context such as the ANTI-Vea, which has been shown to be suitable to measure vigilance decrement, would tend to induce underactivation. This state would be especially detrimental for the tonic arousal or activation in individuals with higher ADHD symptoms. As a consequence, environmental stimulation, such as warning signals, would compensate for that underactivated state,

thereby bringing performance to normal levels. Although impaired alerting network is well-established in ADHD children (Arora et al., 2020), this phenomenon has been less frequently reported in adults with ADHD (e.g., Oberlin et al., 2005). Our findings are inconsistent with Zamani Sani et al.'s (2020) report of no association between alerting network with ADHD symptoms in nonclinical adults, despite they had higher statistical power than us. Differences in the task length or difficulty, in the type of warning signal (auditory vs. visual), or in the measure of ADHD symptoms (childhood vs. adulthood) could help explain these contradictory findings.

The lack of an association between ADHD symptoms and the orienting effect in our data is theoretically and empirically consistent with previous literature (Arora et al., 2020; Berger & Posner, 2000; Lundervold et al., 2011; Zamani Sani et al., 2020). Of note, most research uses the original ANT, which provides a global index of orienting network. However, tasks such as the ANTI or the ANTI-Vea specifically assesses exogenous orienting, which is related to automatic processes (Ishigami et al., 2016). The scarce research on exogenous orienting in ADHD has failed to find alterations in children (Casagrande et al., 2012; Mullane et al., 2011), which is consonant with our results with symptoms in nonclinical adults.

Contrary to our hypothesis, we could not find an association between ADHD symptoms and the congruency effect. Indeed, executive attention has been hypothesized to be deficient in ADHD (Berger & Posner, 2000), and evidence using the ANT in children (Arora et al., 2020) and adults (Lampe et al., 2007; Oberlin et al., 2012; but see Lundervold et al., 2011) has supported this notion. However, both Zamani Sani et al.'s (2020) and us failed to extend those findings to nonclinical samples. From a dimensional view of ADHD, it could be argued that the association between executive attention and ADHD symptoms is not sufficiently meaningful in nonclinical adults. In parallel, we believe that the difference between tasks is highly relevant in this regard. In the ANT/ANTI, the flanker task is

performed as a single task whose only goal is to respond to the target direction. By contrast, the ANTI-Vea incorporates a second goal into the mindset, which is simultaneous to the first one—namely, to respond to the vertical displacement of the target. This increase in working memory load has been found to reduce the flanker interference, leading to a lower congruency effect (Luna, Telga, et al., 2020). Indeed, the congruency effect we obtained for RT and percentage errors were less than half of the usually reported in the ANT in nonclinical adults (MacLeod et al., 2010). This substantially lower congruency effect probably makes the index less sensitive to modulation from individual differences, such as ADHD symptoms, which is a concern about the ANTI-Vea to bear in mind. Alternatively, this result could be interpreted in the sense that adults with higher ADHD symptom scores, when appropriately challenged by task demands, as in the ANTI-Vea task, can overcome any putative executive deficit they might have.

Executive and Arousal Vigilance

Partial support for our hypothesis of a poorer EV associated with ADHD symptoms was limited to indices of performance over time (i.e., vigilance decrement). This is consonant with Craig and Klein's (2019) finding in nonclinical adults. However, this is rather the opposite pattern as Huang-Pollock et al.'s (2012) meta-analysis with ADHD children, who found larger deficits in overall performance than in performance over time. Performance over time is considered the appropriate form to measure vigilance (Huang-Pollock et al., 2012; L. Tucha et al., 2017), although numerous tasks used in ADHD research have failed to measure it (e.g., Johnson et al., 2001; Marchetta et al., 2008; L. Tucha et al., 2017). However, the ANTI-Vea task has been specifically developed to induce such vigilance decrement. Further research comparing clinical ADHD with nonclinical controls in the ANTI-Vea is likely to find larger and more consistent differences in vigilance decrement than previously reported.

Different from other EV tasks, vigilance decrement in the ANTI-Vea mainly manifests as a change to a more conservative response criterion, rather than a loss of sensitivity[9]. However, our data showed ADHD symptoms to be associated with a decrement of sensitivity over the task, but not with a more conservative response style—indeed, we observed the opposite trend. This pattern, consistent with clinical research (Huang-Pollock et al., 2012, 2020), suggest that EV impairments in ADHD symptoms are more a matter of sensitivity than a response bias (Thomson et al., 2016). However, the relatively low rate of false alarms in this literature, prevents us from ruling out a floor effect that might be overestimating the role of sensitivity at the expense of underestimating the role of response criterion. Indeed, Luna, Roca, et al. (2021) found a drop in sensitivity only among those participants with a percentage of false alarms close to the floor (≤ 5%) in the first block, but not for the rest of participants. A similar pattern was observed in our data.

Furthermore, we found ADHD symptoms—only retrospectively reported in childhood—to be associated with a diminished AV, in both mean RT and response variability (i.e., standard deviation and percentage of lapses). These results support our hypothesis and are consonant with the scarce clinical research comparing adults with ADHD in overall and over time AV measures of response variability (Gmehlin et al., 2016; L. Tucha et al., 2017). However, different from clinical studies, we also found that a greater increment of mean RT was positively associated with ADHD symptoms. As in the case of EV, the fact that the ANTI-Vea is the only task of this literature that generates decrement in AV might account for such discrepancies. Moreover, higher response variability associated with ADHD is ubiquitous to multiple types of tasks (Epstein et al., 2011; Kofler et al., 2013). Our data extended this phenomenon to symptoms in nonclinical adults in an AV task that is embedded in a complex structure (i.e., the ANTI-Vea).

[9] Although a loss of sensitivity over the task has been reported in our data as well as in studies with high statistical power (Feltmate et al., 2020; Luna, Roca, et al, 2021), this effect size seems to be lower than the effect on the response criterion.

The relationship between EV and AV is also relevant to the field of ADHD. Grounded on van Zomeren and Brouwer's (1994) attentional model, Gmehlin et al. (2016) argued that sustained alertness (strongly related to AV) is a precondition for more complex attentional functions over time—including processes that could be considered as components of EV. According to this view, Gmehlin et al. found that, when controlling for the slope of AV (i.e., change in percentage of lapses across blocks), differences between ADHD and control groups in EV disappeared. By contrast, there is evidence supporting that EV and AV, albeit probably related, constitute independent components of vigilance (e.g., Luna et al., 2019; Luna, Román-Caballero, et al., 2020; Sanchis et al., 2020). In our data, an equivalence test (Lakens, 2017) showed that the correlation of $r(111) = -.06$ between the slopes of hits (EV) and lapses (AV) fell below the upper bound of $r = .1$ ($p = .044$). This suggests that EV and AV do not depend on each other in a meaningful way. Furthermore, the partial correlation between ADHD symptoms (ASRS-5) and the slope of the percentage of hits, controlling for the percentage of lapses, remained significant, $\tau(110) = -.11$, $p = .037$. This result, inconsistent with Gmehlin et al., does not support the idea of AV as a prerequisite for EV and could be in line with the notion of ADHD as a heterogeneous condition (Fair et al., 2012).

Irrelevant Distraction

Although we found an acceptable effect of ID on the RT, the lack of correlation with ADHD symptoms does not support our hypothesis, and it is contrary to Forster and Lavie's (2016) findings. In fact, our results are in line with Meier's (2021) failed attempt to replicate Forster and Lavie's results using exactly the same task and a similar sample composition (i.e., university students). Against the case of a Forster and Lavie's false positive, it should be noted that they also found a positive correlation in a second experiment with a different task as well as in a case-control study comparing ADHD with controls (Forster et al., 2014). Therefore, the possibility of a true effect is still likely. Regarding the event of a false

negative in Meier's and our study, assuming the effect found by Forster and Lavie (r = .32), a very high statistical power was achieved by Meier (.99) and us (.95). Moreover, Meier found Bayesian evidence favoring the null hypothesis. Of note, the reliability of the ID index reported by Meier and us was rather low (r_{SB} = .26 and .21, respectively). This importantly reduces the size of the observed correlation with ADHD symptoms, leading to the need for a larger sample size and higher reliability scores to reach the desired power (Parsons et al., 2019). Further studies are warranted not only to consistently determine the existence of a positive correlation between the ID effect and ADHD symptoms, but also to test whether this correlation is stronger than those using task-relevant distractors (e.g., flanker task).

Measuring ADHD Symptoms in Childhood and Adulthood

To gain a better knowledge of ADHD symptomatology, we used three different but complementary measures: one for symptoms in childhood (cBAARS-IV) and the other two for symptoms in adulthood (aBAARS-IV and ASRS-5). Characterizing developmental trajectories in ADHD is important to obtain more homogeneous subgroups and phenotypes, also at the neurocognitive level (Luo et al., 2019; Sonuga-Barke & Halperin, 2010). In a longitudinal study, Moffit et al. (2015) found that ADHD in childhood had very little overlap with the adult-onset form of ADHD. Moreover, at age 38, only participants with ADHD in childhood showed neuropsychological deficits, including overall performance in EV. Although EV was the only domain where we found poorer performance to be associated with ADHD symptoms in adulthood but not in childhood, our altered EV indices were of performance over time. In fact, our general picture of results differentiated ADHD symptoms in childhood versus in adulthood. While the former predicted alterations in arousal (i.e., alerting network and AV), the latter were negatively associated with executive outcomes (i.e., EV decrement). This dissociation is, to some extent, consonant with Halperin and Schulz's (2006) neurodevelopmental model of ADHD.

This model postulates that, while the early onset of the disorder is associated with subcortical structures involving arousal, the persistence of the ADHD in the adulthood is related to prefrontal regions which underlie executive processes. The fact that this model could explain developmental differences in the neuropsychological correlates of nonclinical symptoms coheres with the dimensional nature of ADHD.

Within ADHD symptoms in adults, it is noteworthy that, while the ASRS-5 yielded some significant correlations with ANTI-Vea measures, the aBAARS-IV did not. Besides the possible statistical errors that will be mentioned in the next section, a tentative account is related to the different form of both self-reports to measure adult ADHD symptoms. The aBAARS-IV uses the 18 DSM-IV criteria (without examples) as items. The content of these items is generic for children and adults. By contrast, the ASRS-5 is not only based on DSM-5 criteria, which better reflect the adult presentation, but also include items specifically designed to detect ADHD in adults (Ustun et al., 2017). Therefore, instead of a lack of relationship between adult ADHD symptoms and neuropsychological deficits, it might be that highly sensitive self-reports are needed to accurately capture the adult presentation of ADHD symptomatology, along with its underlying alterations.

Limitations

We have identified four main caveats in our study. The first one regards the generalization of our findings. Our community sample consisted of undergraduates, with a majority of women. Not only are both sociodemographic characteristics unrepresentative of the general population, but they also are negatively correlated with ADHD symptom severity (Arnett et al., 2015; Birchwood & Daley, 2012). Despite this sampling bias, our statistical analyses do not suggest that the distribution of ADHD symptoms in our sample is meaningfully more homogeneous—and less sensitive to correlate with behavioural tasks—than in a representative community sample. Moreover, only two out of our 120 participants (i.e., 1.6%) had the diagnosis of ADHD. While this proportion is lower than the

estimated worldwide prevalence of the disorder in adults (3.6%), it is close to the Spanish prevalence (1.2%; Fayyad et al., 2017)). Of note, a study conducted at a Spanish primary care center found an extremely low prevalence (0.04%) of registered ADHD diagnoses in adults (Aragonès et al., 2010). In any case, our unsubstantial number of potential participants with ADHD prevents our results from having direct implications for clinical ADHD research and practice. Therefore, replications of our findings with more representative samples including a substantial amount of ADHD individuals are warranted.

The second concern has to do with the construct validity of ADHD symptoms in our study. We failed to assess relevant symptoms such as depression or anxiety and did not ask for other psychiatric disorders. Thus, it is not clear to what degree the ratings obtained from our sample validly reflect an ADHD symptom status rather than a general psychological distress severity. In fact, symptoms of depression and anxiety have been linked to ADHD symptoms (Combs et al., 2015), and ADHD diagnosis requires that its symptoms be not better explained by another disorder such as mood or anxiety disorders (APA, 2013). Ultimately, we cannot rule out that the relation found between ADHD symptoms and attentional functioning in our study might be a byproduct of a third construct (e.g., depression, stress, other disorders, intelligence, sociodemografic factors). Future research should properly assess and control for these potential confounders as well as incorporate measures of ADHD symptoms beyond self-reported questionnaires (i.e., other-reports, clinical interviews).

Third, the general picture of correlations between attentional processes and ADHD symptom self-reports shows that, at best, our hypotheses were supported only partially. That is, no attentional domain exhibited significant correlations with ADHD symptoms across the three self-reports. Also, for those observed significant correlations the effect sizes were at most small to medium. Besides the sampling bias discussed above, a more

plausible reason is related to the psychometric properties of the ANTI-Vea indices. Although our task reliability scores are similar to the ones reported in Luna, Roca, et al. (2021), the reliability found for difference scores and slopes tended to be fairly low. This limitation, which is also inherent to most cognitive tasks (Dang et al., 2020; Hedge et al., 2018), could dramatically attenuate the observed correlations coefficients. Futures studies should either attempt to improve the reliability of their tasks or use valid methods to correct for low reliability to estimate the true correlation between ADHD symptoms and attentional processes.

The fourth limitation concerns the control of the type I error rate in our results. Since our study did not reach a very high statistical power, strict corrections for multiple comparisons were likely to dramatically increase the rate of false negatives. Following McDonald's (2014) suggestion, we conducted an exploratory secondary analysis where we applied the Benjamini-Hochberg procedure (Benjamini & Hochberg, 1995) to our correlation matrix in order to control for a false discovery rate of 20%. Groups for multiple comparisons were set according to our hypotheses. The significant findings of this corrected pattern of correlations are roughly similar to such comparisons before the correction (see **Supplemental Table 3**). In any case, to attain a more proper control of both types of statistical errors, our study needs to be replicated with a larger sample.

Conclusion

To conclude, our modified version of the ANTI-Vea was useful for measuring the functioning of the attentional networks, executive and arousal vigilance, and irrelevant distraction. This fine-grained distinction between attentional processes is relevant to gain a depth understanding of the mechanisms underlying ADHD symptomatology. In a sample of undergraduates, we found that subjective ADHD symptoms in childhood were related to alerting and arousal processes, while symptoms in adulthood were rather associated with the executive component of vigilance. Different from other neuropsychological tasks,

the ANTI-Vea could successfully induce vigilance decrement. However, compared to other tasks (e.g., ANT), our index of the executive attentional network (i.e., congruency effect) was fairly reduced by task demands. Moreover, some of the task indices (especially those involving difference scores) exhibited poor reliability. Although replications with larger and clinical samples are necessary, this thorough approach to the attentional processes underlying ADHD symptoms might shed light on the search for more homogeneous subgroups of the disorder.

Supplemental Materials

SUPPLEMENTAL TEXT 1: ADHD Symptom Distribution in the Present Study Compared to an Estimated Normative Distribution

Procedure

We calculated the total scores of ADHD symptoms for each of the three self-reports (i.e., the cBAARS-IV, the aBAARS-IV, and the ASRS-5). Percentage of individuals above the preliminary clinical cutoff were analyzed. Furthermore, for the cBAARS and the aBAARS, the distribution of ADHD symptoms was compared with an estimation of Barkley's (2011) normative distribution, which was obtained from a large representative sample of adults from the United States. This normative sample was summarized in a table that includes the values corresponding to 22 and 20 percentiles for the cBAARS and the aBAARS, respectively. Based on such percentiles, we used bootstrapping to generate a sample of 10,000 simulated scores for each questionnaire. The bootstrap procedure was conducted using an R script (for a rationale, see Ernst & Hutson, 2003). Percentiles of the simulated sample fitted Barkley's percentiles for each ADHD symptom scale, and they may be compared with our study percentiles in **Supplemental Table 1.** Finally, the simulated normative sample allowed the extraction of 120 theoretical quantiles, thereby providing a sample equally sized as our study sample. This normative equally-sized sample is crucial to conduct the following distribution analyses.

Results

Figure 2 (in the main text) shows the distribution of ADHD symptoms compared to the estimated normative sample (see Supplemental Table 1 and Supplemental Figure 1 for more details). Two-sample Kolmogorov-Smirnov test indicated that ADHD symptom distributions in the cBAARS-IV and the aBAARS-IV were significatively different from the estimated normative sample (both *p*s < .001). Indeed, a visual comparison shows that the

normative distributions are more left-skewed than their respective sample distributions. Compared to the 50th percentile in the normative sample, Wilcoxon Signed-Ranks Test indicated that the medians of the cBAARS-IV and the aBAARS-IV were significatively higher (both *p*s < .001). Levene's test for homogeneity of variances showed no significant differences between our sample and normative sample for the cBAARS-IV and the aBAARS-IV (*p*s = .083 and .600, respectively). Binomial tests indicated that the percentage of participants above the 95th percentile cutoff in the cBAARS (2.5%) and the aBAARS-IV (5%) was not significantly different from the expected 5% (both *p*s > .292). Neither were the percentage of participants above the cutoff of 14 points in the ASRS-5 (5.83%) significantly different from the expected 11.2% (*p* = .060[10]). Indeed, the level of ADHD symptoms in our participants with high scores was close or similar to subjective symptom severity in ADHD patients.

[10] Although the difference of proportions is marginally significant, note that the criterion 11.2% comes from a sample of adults with 8.2% of ADHD cases (Ustun et al., 2017), which is a higher percentage than in the general population.

SUPPLEMENTAL TEXT 2: Irrelevant Distractor Position and Attention-Deficit/Hyperactivity Disorder Symptoms: An Exploratory Analysis

In order to examine the role of the distractor position in relation to the target, we conducted a further exploratory analysis. Distractor present trials were divided according to whether the distractor appeared on the same (as in **Figure 1**) or on the opposite side as the target (e.g., below fixation when the target appeared above). Compared to the condition without distractor, RTs were slower for both the distractor present same side condition, $M_{\Delta RT}$ = 36 ms, $SD_{\Delta RT}$ = 46 ms, $t(101) = 7.91$, $p < .001$, $d = 0.78$, and the distractor present opposite side condition, $M_{\Delta RT}$ = 27 ms, $SD_{\Delta RT}$ = 58 ms, $t(101) = 4.82$, $p < .001$, $d = 0.48$. There were no differences between the two distractor present conditions, $t(101) = 1.56$, $p = .123$, $d = 0.15$. While the effect of distraction (i.e., percentage increase in mean RT) from the opposite side did not correlate with any ADHD symptom self-report (all *p*s > .494), distraction-same effect showed a significant positive correlation with the ASRS-5, $\tau(100) = .12$, $p = .037$, but not with the cBAARS-IV or the aBAARS-IV (both *p*s > .172). Nonetheless, the reliability estimate was low for distractor-same effect (r_{SB} = .26) and virtually zero for distractor-opposite effect (r_{SB} = –.08).

As a conclusion, the distractor position might play an important role to replicate the correlation between ADHD symptoms and the irrelevant distractor effect (Forster & Lavie, 2016) in the ANTI-Vea. The low reliability of the indices is a limitation to bear in mind.

SUPPLEMENTAL TABLE 1: Percentiles Values of the cBAARS-IV and the aBAARS-IV in Original and Estimated Normative Samples Compared to the Sample of the Present Study

	cBAARS-IV total scores		aBAARS-IV total scores	
Percentile	**Normative sample**	**Study sample**	**Normative sample**	**Study sample**
99	60 (61)	56.29	54 (55)	51.48
98	55	48.24	49	44.24
97	52 (53)	47.00	46	43.00
96	51	45.48	44	43.00
95	49	45.00	43	42.05
94	47 (48)	42.86	42 (41)	41
93	46	41.67	39	40.67
92	45	40.48	38	40.00
91	44	39.29	37	40.00
90	43	39.00	36	39.10
89	42	39.00	—	—
88	41 (40)	39.00	—	—
87	39	39.00	—	—
85	38	38.00	35	35.00
84	37	37.00	—	—
83	—	—	34	35.00
82	36	37.00	33	34.58
81	—	—	32	34.00
79	—	—	31	33.01
77	35	35.00	30	33.00
76	34	35.00	—	33.00
75	33	35.00	29	32.25
51	24 (23)	28.00	23 (22)	27.00
50	23	28.00	22	27.00
1	18	18.00	18	19.00

Note. cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms. aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms. Percentiles in the normative sample were extracted from Barkley (2011), which is based on a large representative sample of adults from the United States. These percentile values were used to generate a simulated normative sample through bootstrapping (N = 10,000). Direct scores of the simulated sample are in parentheses when they differ from the original normative sample.

SUPPLEMENTAL TABLE 2: Kendall's Rank Correlations Among ANTI-Vea Outcomes

ANTI-Vea Index	1	2	3	4	5	6	7	8	9	10	11	12
Attention Network Test for Interaction (ANTI) outcomes												
1. RT overall	—											
2. RT alerting	.05	—										
3. RT orienting	−.08	–.07	—									
4. RT congruency	–.15*	–.05	.08	—								
5. % errors overall	.21**	.15*	–.09	.04	—							
6. % errors alerting	.07	.15*	.08	.22***	.21***	—						
7. % errors orienting	–.06	.07	.26***	.15*	.07	.25***	—					
8. % errors congruency	–.09	.11	–.02	.22***	.20***	.14*	.12	—				
Executive vigilance (EV) outcomes												
9. % hits	.25***	–.01	–.15*	–.22***	–.02	–.18**	–.29***	–.15*	—			
10. % hits slope	.04	.04	–.05	–.06	-001	–.03	–.005	–.09	.16*	—		
11. % false alarms	.24***	.03	–.25***	–.14*	.45***	–.08	–.15*	–.07	.32***	.05	—	
12. % false alarms slope	.01	.01	–.07	.002	–.14*	.05	–.05	.05	–.004	.01	–.14*	—
13. *A′* (sensitivity)	.13*	–.04	–.05	–.18**	–.21**	–.18**	–.22***	–.13*	.76***	.15*	.08	.05
14. *A′* (sensitivity) slope	–.01	.04	.02	–.03	.10	–.01	.06	–.08	.09	-58***	.13*	–.40***
15. *B″* (response bias)	.26***	–.003	.24***	.18**	–.33***	.12	.20**	.09	–.51	–.08	–.81***	.12
16. *B″* (response bias) slope	.02	–.03	.05	–.01	.10	–.11	.01	.01	.10	–.10	.16*	–.68***
Arousal vigilance (AV) outcomes												
17. RT mean	.33***	.04	–.03	.01	.13*	.13*	.02	.03	.04	–.06	.07	.03
18. RT mean slope	.08	.07	.01	.15*	.18**	.25***	.12	.11	–.08	.06	.05	–.08
19. Standard deviation	.28***	.08	–.03	.11	.26***	.33***	.10	.15*	–.05	–.13*	.06	–.02
20. *SD* slope	.04	–.04	.04	.05	.11	.10	.10	.06	–.08	–.01	–.01	–.06
21. % lapses	.33***	.07	–.03	.08	.18**	.25***	.05	.11	–.01	–.10	.03	.03
22. % lapses slope	.14*	.02	.04	.11	.14*	.20**	.10	.14*	–.06	–.02	.02	.01
Irrelevant distraction (ID) outcomes [a]												
23. % interference in RT	.07	–.11	.01	.01	–.12	.07	–.11	–.10	–.01	–.004	–.06	.17**
24. % errors interference	–.11	–.10	.05	.01	–.10	–.07	.09	.04	–.12	–.11	–.15*	.10

ANTI-Vea Index	13	14	15	16	17	18	19	20	21	22	23	24
Attention Network Test for Interaction (ANTI) outcomes												
1. RT overall												
2. RT alerting												
3. RT orienting												
4. RT congruency												
5. % errors overall												
6. % errors alerting												
7. % errors orienting												
8. % errors congruency												
Executive vigilance (EV) outcomes												
9. % hits												
10. % hits slope												
11. % false alarms												
12. % false alarms slope												
13. *A′* (sensitivity)	—											
14. *A′* (sensitivity) slope	.06	—										
15. *B″* (response bias)	–.26***	–.12*	—									
16. *B″* (response bias) slope	.05	.23***	–.17**	—								
Arousal vigilance (AV) outcomes												
17. RT mean	.03	–.06	–.08	.04	—							
18. RT mean slope	–.10	.09	–.01	.05	.22***	—						
19. Standard deviation	–.08	–.05	–.04	.04	.39***	.27***	—					
20. *SD* slope	–.07	.02	.03	.05	.07	.36***	.22***	—				
21. % lapses	–.02	–.07	–.03	.04	.71***	.31***	.60***	.16*	—			
22. % lapses slope	–.06	–.01	–.001	–.01	.24***	.55***	.28***	.51***	.35***	—		
Irrelevant distraction (ID) outcomes [a]												
23. % interference in RT	–.01	.11	.07	–.14*	–.06	–.09	–.01	–.08	–.03	–.004	—	
24. % errors interference	–.05	–.14*	.16*	–.10	–.03	.0003	–.03	.11	–.01	.02	–.09	—

Note. n = 113. ANTI-Vea = Attention Network Test for Interactions and Vigilance—executive and arousal components. RT = Reaction time.

[a] n = 102.

*p < .05. **p < .01. ***p < .001.

SUPPLEMENTAL TABLE 3: Kendall's Rank Correlations Between ANTI-Vea Outcomes and ADHD Symptoms With Corrections for Multiple Comparisons

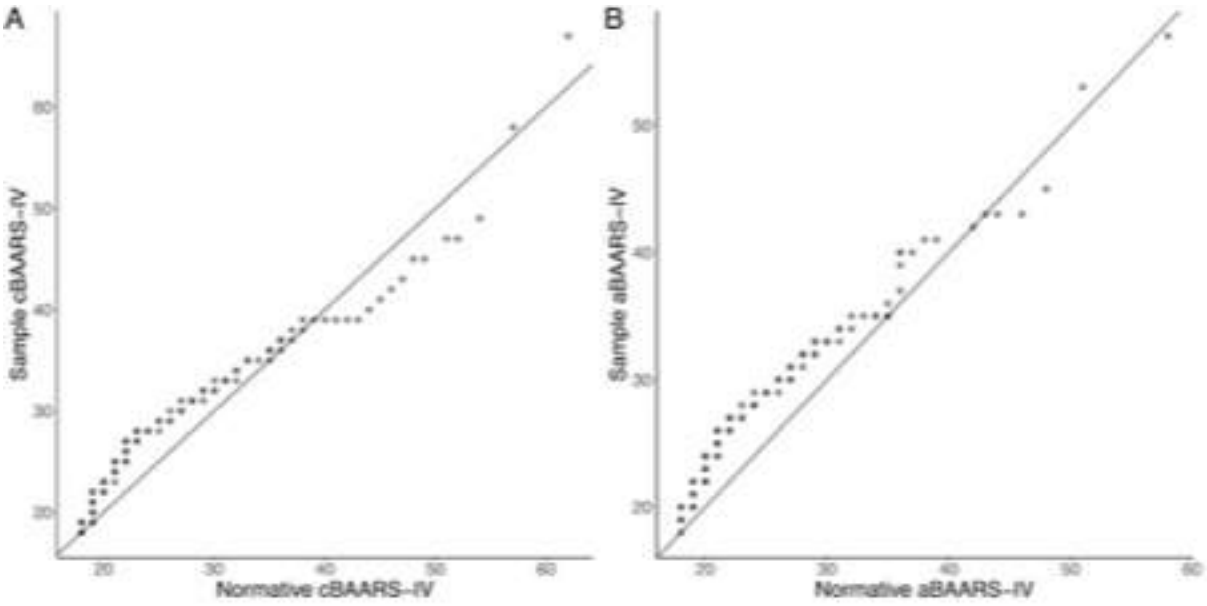
		ADHD symptoms (self-reports)		
ANTI-Vea index	**H_1 direction**	**cBAARS-IV**	**aBAARS-IV**	**ASRS-5**
ANTI outcomes				
RT overall	+	.06	–.08	–.03
% errors overall	+	.06	–.08	–.06
RT alerting	+	**.13***	**.10**	.03
% errors alerting	+	**.15***	**.09**	.05
RT orienting	≠	.01	.02	.11
% errors orienting	≠	.04	–.01	–.05
RT congruency	+	.05	.03	.03
% errors congruency	+	.03	.01	–.06
EV outcomes				
% hits	–	.001	–.05	.01
% false alarms	+	–.001	–.11	–.02
***A′* (sensitivity)**	–	–.01	–.02	.02
***B″* (response bias)**	≠	–.02	.11	.02
% hits slope	–	–.02	–.04	**–.11***
% false alarms slope	+	.05	.09	**.11***
***A′* (sensitivity) slope**	–	–.03	–.09†	**–.14***
***B″* (response bias) slope**	+	–.01	–.11	–.08
AV outcomes				
RT mean	+	**.11***	.04	**.10**
RT standard deviation	+	**.11***	.06	.01
% lapses	+	**.11***	.07	**.08**
RT mean slope	+	**.17****	.06	–.01
RT *SD* slope	+	.08	.02	–.05
% lapses slope	+	**.18****	.06	–.01
ID outcomes [a]				
% interference in RT	+	.02	.02	.03
% errors interference	+	–.01	.02	.05

Note. n = 113. ADHD = Attention-deficit/hyperactivity disorder. H_1 = Alternative hypothesis. cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms. aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms. ASRS-5 = Adult ADHD Self-Report Screening Scale for DSM-5. ANTI = Attention Network Test for Interaction. EV = Executive vigilance. AV= Arousal Vigilance. ID = Irrelevant distraction. RT = Reaction time. Asterisks indicate significance before Benjamini-Hochberg procedure. Significant correlations after corrections for multiple comparisons ($q < .2$) are shown in bold. Corrections were independently applied to each group of multiple comparisons, which corresponded to our nine distinct theoretical hypotheses (i.e., ANTI overall performance; the three attentional networks; the two types of vigilance, both overall scores and performance over time; and the irrelevant distraction). Statistical hypotheses may be directional (positive or negative) or nondirectional.

[a] n = 102.

*$p < .05$. **$p < .01$. No other $p < .05$ appeared with exploratory two-tailed tests.

SUPPLEMENTAL FIGURE 1: Q-Q Plots Comparing ADHD Symptom Distribution in our Sample With Symptom Distribution in an Estimated Normative Sample for the cBAARS-IV (Panel A) and the aBAARS-IV (Panel B)



Note. The normative, equally-sized sample was obtained by extracting 120 quantiles from a large bootstrapped sample (*N* = 10,000) that fits the percentile values available in Barkley (2011). **Panel A**: cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms. **Panel B**: aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms.

Study 3

Attention-Deficit/Hyperactivity Disorder Symptoms as Function of Arousal and Executive Vigilance: Testing the Halperin and Schulz's Neurodevelopmental Model in an Adult Community Sample

This work has been submitted for publication as:

Coll-Martín, T., Carretero-Dios, H., & Lupiáñez, J. (2023). Attention-deficit/hyperactivity disorder symptoms as function of arousal and executive vigilance: Testing the Halperin and Schulz's neurodevelopmental model in an adult community sample. *Collabra: Psychology*.

Current state: Minor reviews required.

Abstract

Halperin and Schulz's neurodevelopmental model postulates that the onset of attention-deficit/hyperactivity disorder (ADHD) in childhood is due to subcortical alterations, whereas the disorder trajectory into adulthood depends on the development of executive functions. Based on a dimensional framework of ADHD, Coll-Martín et al. (2021) found support for the model in an adult community sample assessed in arousal and executive vigilance. The present study is a preregistered (https://osf.io/tkdq7) close replication of Coll-Martín et al. with stricter control of statistical error rates to test the two hypotheses of the model. A sample of university students (*N* = 292 valid; 49% women) completed self-reports of ADHD symptoms in childhood (retrospectively) and adulthood and performed the online version of an attentional task (the ANTI-Vea). Our preregistered hypotheses achieved an acceptable statistical power for the effects of interest, even after accounting for measurement error. Despite this, only the unexpected negative correlation between executive vigilance and symptoms in childhood was significant, therefore refuting the theoretical predictions. Similarly, neither multiverse nor exploratory analyses supported the dissociation pattern proposed by the neurodevelopmental model. ADHD symptoms across the lifespan may be pathophysiologically identical, at least in terms of vigilance. Future studies could include complementary assessment methods and clinical groups.

Materials, data, and analyses that support the findings of this study are openly available at https://osf.io/vqgms/

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental condition characterized by age-inappropriate, persistent, and impairing levels of inattention and/or hyperactivity–impulsivity (American Psychiatric Association [APA], 2013). The disorder is present in about 5% of children and 2.5% of adults (Polanczyk et al., 2007; Simon et al., 2009). Throughout the lifespan, ADHD is a risk factor for several negative outcomes, including educational underachievement, difficulties with employment, and criminality (Faraone et al., 2015; Fletcher, 2014; Loe & Feldman, 2007). Against this backdrop, identifying the neurocognitive mechanisms underlying ADHD symptoms across development is crucial to enhance the approach to the disorder (Castellanos & Tannock, 2002; Luo et al., 2019; Sonuga-Barke et al., 2023)

Neurodevelopmental Model of ADHD

Numerous theoretical models have formulated different explanations of ADHD based on underlying impairments in single (Barkley, 1997; Sergeant, 2005; Sonuga-Barke et al., 1992) or multiple (Durston et al., 2011; Sonuga-Barke, 2003) neurocognitive pathways. In an attempt to account for the developmental trajectory of the disorder, Halperin and Schulz (2006) elaborated a *neurodevelopmental model of ADHD* (also referred here as “neurodevelopmental model”). The authors postulate a double dissociation in which the onset of ADHD in childhood is due to subcortical brain dysfunctions that remain stable over the lifespan, while the reduction of symptom severity with age is dependent on the development of the prefrontal cortex. In this sense, executive functions, mediated by the prefrontal cortex, are not central to the early emergency of ADHD, but influence on its developmental course into adulthood.

The neurodevelopmental model generates testable predictions in measures of cognitive tasks. First, the subcortical brain dysfunctions should be observable in measures

with minimal or no executive load, such as basic reaction time (RT) or RT variability in simple RT task. These measures should distinguish ADHD children from controls across the lifespan, regardless of adolescent or adult status. Second, measures of executive function, such as target identification or inhibition tasks, should be dimensionally related to the severity of ADHD symptomatology, particularly in adulthood. Importantly, the model considers the phenomenon of late-onset ADHD (Asherson & Agnew-Blais, 2019), which would be product of either early or late lesions in the prefrontal cortex subserving executive processes.

Initial support for the neurodevelopmental model came from a prospective longitudinal study of 98 children with ADHD who were reassessed after 10 years (Halperin et al., 2008). Compared to matched controls, the adolescents diagnosed with ADHD in childhood showed deficient response variability, irrespective of their current clinical status. However, only adolescents with persistent—but not remitting—ADHD exhibited poorer inhibition and working memory than controls. Despite the promising findings of this study, subsequent research has yielded mixed results (Coghill, Hayward, et al., 2014; Coll-Martín et al., 2021; Gmehlin et al., 2016; Leenders et al., 2021; McAuley et al., 2014; Rommel et al., 2015). Furthermore, a systematic review found no support for the model, as the patterns of neurocognitive deficits in individuals with ADHD were similar for high- and low-executive-load measures (van Lieshout et al., 2013).

Although the current literature is not very encouraging with the neurodevelopmental model, some gaps and methodological issues are worth considering. First, most of these studies consisted of case–control comparisons that were underpowered to detect a range of effects that could be considered theoretically relevant (e.g., 0.35 ≥ Cohen's d_s ≥ 0.2). In contrast, population-based or community samples can better fit the dimensional nature of ADHD (Coghill & Sonuga-Barke, 2012; Hilger et al., 2020) and are more efficient in collecting well-powered sample sizes. Second, studies that included low-executive-load

outcomes rarely used tasks with no executive component (e.g., Coll-Martín et al., 2021; Gmehlin et al., 2016). In this sense, even RTs or omission errors in tasks such as the continuous performance test (CPT) are influenced by the executive processes and the response criterion involved in the task demands. The next section will address this issue.

Measuring Executive and Arousal Vigilance

Impaired vigilance is central to the phenotypic and neurocognitive characterization of ADHD (Huang-Pollock et al., 2012; Wilding, 2005; Willcutt et al., 2005). The construct is generally defined as the attentional capacity to maintain performance over time. Given the diversity of terms and measures linked to vigilance, some authors deem it as a nonunitary concept (Langner & Eickhoff, 2013; Luna et al., 2018; Sturm et al., 1999). In this vein, Luna et al. distinguish two components of vigilance: *executive vigilance* (EV) and *arousal vigilance* (AV).

EV is the ability to detect infrequent but critical signals among nonsignal stimuli. It is measured with tasks derived from the CPT paradigm such as the AX-CPT (Rosvold et al., 1956) or the Test of Variables of Attention (TOVA; Greenberg & Waldman, 1993). This type of taskf seems to involve executive mechanisms of sustained attention for stimuli discrimination and goal-oriented response selection. Notably, response-inhibition CPTs (e.g., Conners's CPT [Conners, 2000], Sustained Attention to Response Task [SART; Robertson et al., 1997]), the most common CPT variant in ADHD research, also require motor suppression of the preponderant response in the presence of the target stimulus. Key measures of EV are hits (inverse of omission errors) and false alarms, both tending to decrease with time on task (for a discussion about false alarms, see Thomson et al., 2016).

AV is the capacity to sustain a rapid reactivity to any environmental stimulus—without implementing any control over the selection of the response executed. This form

of vigilance is measured in simple RT tasks, such as the Psychomotor Vigilance Test (PVT; Lim & Dinges, 2008) and the WAFA test of the Vienna Test System (Schuhfried, 2013). These tasks seem to record an arousal component of vigilance, a mechanism that could be more related to physiological levels of excitability. The main indices of AV are the mean and variability of the RT and the attentional lapses. Contrary to EV, this vigilance decrement manifests as an increase of the measures during the task.

In order to evaluate both vigilance components simultaneously, Luna et al. (2018) designed the *Attentional Networks Test for Interaction and Vigilance—Executive and Arousal Components* (ANTI-Vea). This task is based on the Attention Networks Test for Interaction (ANTI; Callejas et al., 2004), which combines an Eriksen flanker paradigm with spatial cues and warning signals to assess the three attentional networks (M. I. Posner & Petersen, 1990). In ANTI trials, which are the bulk of the ANTI-Vea, participants must respond to the direction pointed by the central arrow. To assess EV, in a small percentage of trials that central arrow appears vertically displaced for participants to detect and respond to it, thereby suppressing their preponderant response to ANTI trials. To measure AV, in another small percentage of trials, a salient stimulus (i.e., a red down counter) is displayed for participants to stop it as fast as possible. Of note, the length of the task (~33 min) successfully induce a decrement in all vigilance measures (Luna et al., 2018).

Several studies have found that EV and AV are dissociable in the ANTI-Vea, both as a result of experimental manipulations (Feltmate et al., 2020; Hemmerich et al., 2023; Sanchis et al., 2020) and in relation to individual differences (Cásedas et al., 2022; Román-Caballero et al., 2021). In the context of ADHD, Coll-Martín et al. (2021) conducted a study in which we administered the ANTI-Vea to 113 university undergraduates in a

laboratory setting.[11] They assessed ADHD symptom severity retrospectively in childhood and concurrently in adulthood through self-reports. In line with the neurodevelopmental model, the authors found that symptoms in childhood correlated with higher lapses (AV), while adult symptoms were associated to a greater decrease in hits during the task (EV). Although promising, these results came from an exploratory study with several outcomes and need to be replicated with a larger sample and a stricter control of false positive rates and some potential confounders.

The Present Study

This study aimed to test the neuropsychological predictions of Halperin and Schulz's (2006) neurodevelopmental model of ADHD from a dimensional framework. For this purpose, a community sample of university students was assessed for childhood and adult symptoms of ADHD and performed the online version of the ANTI-Vea to measure AV and EV. This procedure can be considered a close replication of Coll-Martín et al. (2021), with the main difference being the setting where the ANTI-Vea was administered (but see Luna, Roca, et al., 2021, for the remarkable psychometric similarities between the lab and online versions). Unlike the original study, the larger sample size and the more specific contrasts selected in this study provides a reasonably acceptable informational value to the statistical tests performed.

According to the neurodevelopmental model and the results of the original study (Coll-Martín et al., 2021), we preregistered the following hypotheses (https://osf.io/tkdq7): (H1) childhood ADHD symptoms would predict higher lapses (AV); and (H2) adult ADHD symptoms would predict lower hits (EV), even after accounting for symptoms in childhood (i.e., after controlling for baseline to focus on later development). As primary outcomes,

[11] Technically, both the cited study and the study we are presenting in this manuscript used a version of the ANTI-Vea that incorporates some random ANTI trials with irrelevant distractors. The added trials represented less than 10% of the total, and they did not affect the normal functioning of the rest of the task indices or the reliability scores (Coll-Martín et al., 2021). For the sake of simplicity, in this paper we refer to this version of the task as "ANTI-Vea" and omit details of those additional trials in the description.

we focused on the measures of vigilance decrement, which has been considered the core feature of the construct (Huang-Pollock et al., 2012; L. Tucha et al., 2017) and was related to ADHD in both vigilance measures in the original study (Coll-Martín et al., 2021). However, we also calculated the overall vigilance scores as secondary outcomes, since these are the measures typically reported in ADHD research and are more reliable (Cásedas et al., 2022; Coll-Martín et al., 2021; Huang-Pollock et al., 2012).

Methods

In order to report the severity of the tests transparently, the study design and analysis plan were publicly preregistered with the Preregistration for Quantitative Research in Psychology Template (PRP-QUANT; (Bosnjak et al., 2022) at https://osf.io/tkdq7 (for a report of deviations from the preregistration, see **Supplemental Table 1**). Consequently, the main statistical hypotheses and additional analyses will be referred to with the same label as in the preregistration document.

Sample Selection and Study Procedure

Figure 1 illustrates the sample selection process. In the first phase of the study, we collected a total of 2,003 responses to a 15-min online survey via LimeSurvey (https://www.limesurvey.org). Participants were recruited through advertisements on the virtual distribution list of our university. In exchange for completing the survey, participants had the opportunity to win two prizes of €200 in a raffle. In addition to the self-reports of ADHD symptoms, the survey included sociodemographic questions and other psychological variables (see the preregistration document for further details). The appearance order of the two main self-reports of ADHD symptoms in childhood and adulthood was randomized. After exclusions, there were 1,540 eligible participants (72.7% women, 26.1% men, 1.23% nonbinary; 18–35 years, *M* = 22.5, *SD* = 3.7).

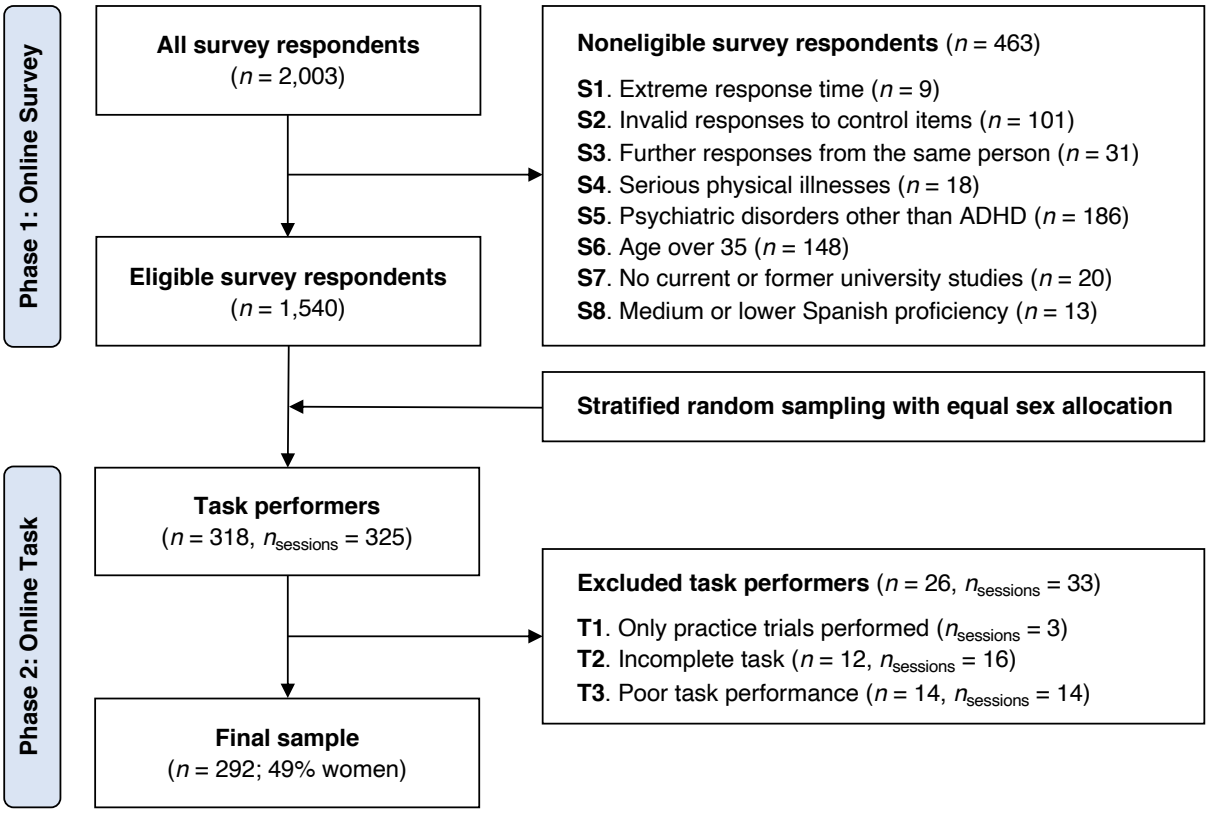


Figure 1. Selection Process of Participants Through Each Stage of the Study. S1–S8 are the exclusion criteria of the survey respondents. S1 based on the mean plus/minus three standard deviations cut-off. S2 based on three attention check items distributed throughout the survey. S4–S8 based on the information reported by participants. S4–S5 referred to diagnosed conditions. S4 limited to diseases that may largely impact task performance (e.g., motor paralysis). Note that there were participants qualified for exclusion by more than one survey criteria. T1–T3 are the exclusion criteria of the cognitive task performers. Note that only one task session per participant was included in the final sample.

In the second phase, eligible participants were randomly invited to perform the online version of the cognitive task (i.e., the ANTI-Vea). Invitations were sent via email until approximately 300 responses were collected. We stratified by sex to ensure a representative distribution of the population in that factor. In addition to the ANTI-Vea, this phase included a brief set of questions about the task (i.e., questions related to the task experience, which are not part of this study, and control items) and a reassessment of ADHD symptoms with the same instruments as in the initial survey. The entire duration of

the second phase was about 50 minutes. Participants were compensated €6 for their voluntary participation.[12]

A total of 318 participants (50% women, 50% men) enrolled in the second phase of the study. They received the link to the web-based version of the ANTI-Vea with the following instructions before starting the task: (a) sit in a comfortable place without distractions; (b) keep entertainment devices (i.e., television, radio, mobile phone, etc.) out of reach; (c) set the computer's sound level at 75% and do not minimize the automatic full-screen mode of the task; (d) if necessary, wear glasses or contact lenses; and (e) if necessary, solve any particular issue before starting so that the task can be completed without any breaks. The stimuli sequence and correct responses for each type of trial are depicted in **Figure 2** (for a more detailed description of the task with audiovisual material, see Method section of the ANTI-Vea website at https://anti-vea.ugr.es/method.html). The ANTI-Vea comprises three types of trials: ANTI (60%), EV (20%), and AV (20%). Participants were encouraged to respond as quickly and accurately as possible while keeping their eyes on the fixation point until the finalization of the task. The ANTI-Vea started with a practice phase, in which instructions and feedback were given so that participants could gradually familiarize themselves with each type of trial.

After the practice phase, participants performed the task itself, which consisted of six seamless blocks of 80 pseudorandomised trials each (48 ANTI, 16 EV, and 16 AV). In ANTI trials, a central arrow (i.e., the target) with two flankers on each side appeared pointing to the left or right. Participants had to discriminate the direction of the target (by pressing either "C" key for leftward direction or "M" key for rightward direction) while ignoring the direction of the flanking arrows, which could point to the same or the opposite direction as the target with equal probability. These stimuli could be preceded by an

[12] For 67 participants (17.5% of the final sample), ADHD symptoms were not assessed in the second phase of the study.

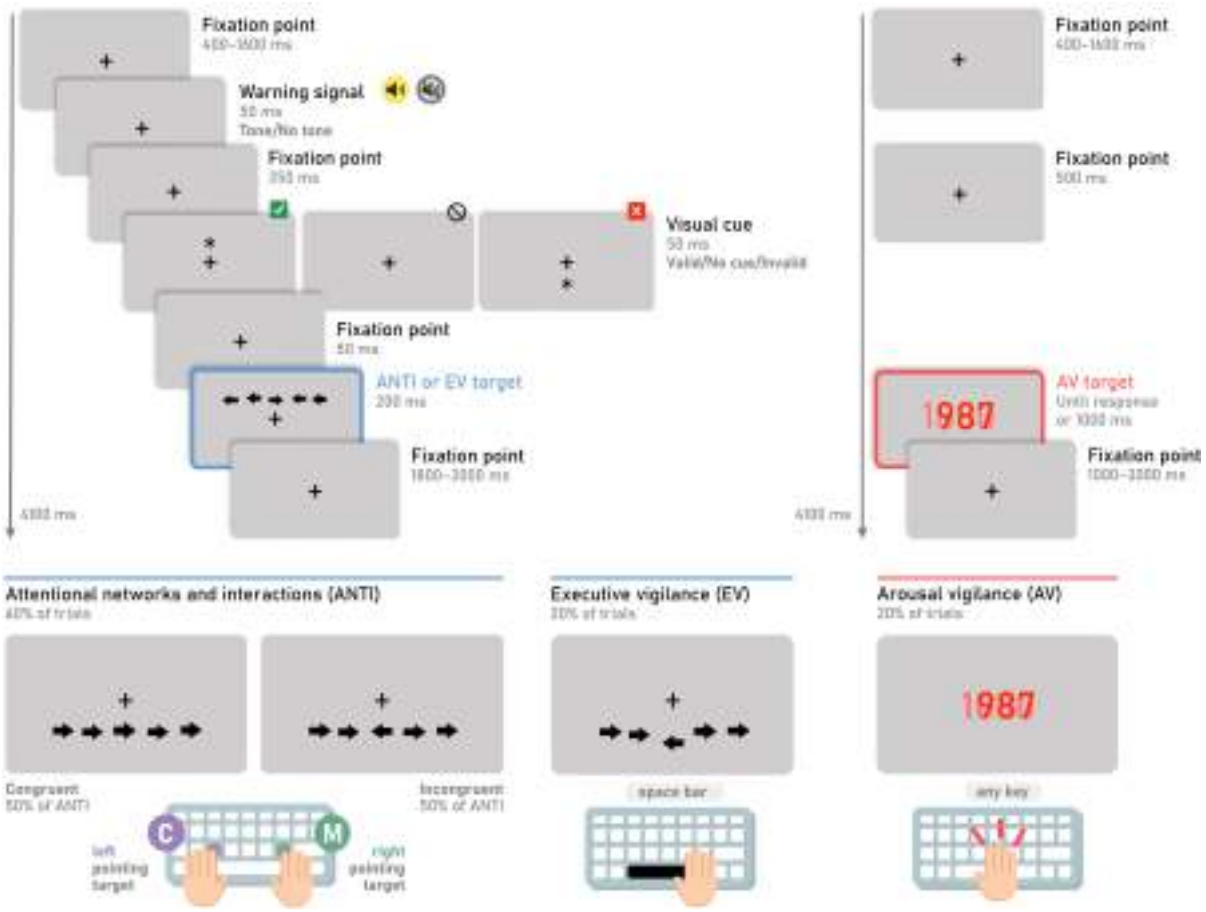


Figure 2. Attention Network Test for Interaction and Vigilance—Executive and Arousal Components (ANTI-Vea) Procedure. The top left shows the temporal sequence of the ANTI and EV trials. Target and flankers could appear above or below the fixation cross and point to the left or the right side with equal probability. The warning signal appeared in half of these trials. The visual cue had an equal chance of appearing in the same location as the target, in the opposite location, or not appearing. The bottom left and middle shows the correct response based on whether the target is vertically aligned with the flankers (ANTI trials) or displaced (EV trials). Note that the five arrows are slightly vertically displaced at random by ± 2 px to generate some noise in ANTI and EV trials, while the target is substantially vertically displaced by ± 8 px in EV trials. The right part shows the temporal sequence and correct response of the AV trials. The duration of each task interval appears next to its corresponding box. Note that, although every trial lasted 4,100 ms, the sequence of events appeared at a variable interval within each trial.

asterisk (i.e., a visual spatial cue) and/or a tone (i.e., a warning signal), thereby allowing the conditions to assess the three attentional networks. EV were similar to ANTI trials, with the exception that here the target appeared vertically displaced from the flankers, so that participants must detect the displacement by pressing the space bar. In contrast, AV trials only displayed a red millisecond down counter (starting at 1,000 ms) for participants to stop it by pressing any key as fast as possible.

After the practice phase, participants performed the task itself, which consisted of six seamless blocks of 80 pseudorandomised trials each (48 ANTI, 16 EV, and 16 AV). In ANTI trials, a central arrow (i.e., the target) with two flankers on each side appeared pointing to the left or right. Participants had to discriminate the direction of the target (by pressing either “C” key for leftward direction or “M” key for rightward direction) while ignoring the direction of the flanking arrows, which could point to the same or the opposite direction as the target with equal probability. These stimuli could be preceded by an asterisk (i.e., a visual spatial cue) and/or a tone (i.e., a warning signal), thereby allowing the conditions to assess the three attentional networks. EV were similar to ANTI trials, with the exception that here the target appeared vertically displaced from the flankers, so that participants must detect the displacement by pressing the space bar. In contrast, AV trials only displayed a red millisecond down counter (starting at 1,000 ms) for participants to stop it by pressing any key as fast as possible.

All data collection took place from June 2021 to June 2022 entirely online. The research project was approved by our institutional ethics.

Instruments

Barkley Adult ADHD Rating Scale-IV: Childhood and Current Symptoms

The self-reports of the Barkley Adult ADHD Rating Scale-IV (BAARS-IV; Barkley, 2011) include two scales to assess ADHD symptoms: retrospectively in childhood (cBAARS-IV) and concurrently in adulthood (aBAARS-IV). Each scale is composed of 18 items, nine of inattention (e.g., “Difficulty sustaining my attention in tasks for fun activities”) and nine of hyperactivity–impulsivity (e.g., “Shift around excessively or feel restless or hemmed in”), in a Likert scale ranged from 1 (*never or rarely*) to 4 (*very often*). While the cBAARS-IV refers to behaviours between 5 and 12 years of age, the aBAARS-IV refers to the last 6 months. Since the items are based on the *Diagnostic and Statistical Manual*

of Mental Disorders (4th ed.; DSM-IV; APA, 1994), we used the Spanish version of the manual for the translation (APA, 1994/1995), as we did in the previous study (Coll-Martín et al., 2021). In our final sample, Cronbach's alpha reliability scores were .90 and .86 for cBAARS-IV and aBAARS-IV, respectively, close to the .95 and .92 of the original BAARS-IV (Barkley, 2011). Barkley proposed the 95th percentile of these scales as a cut-off to identify individuals at high risk of ADHD.

Adult ADHD Self-Report Screening Scale for DSM-5

The Adult ADHD Self-Report Screening Scale for DSM-5 (ASRS-5; Ustun et al., 2017) assesses the adult-specific presentation of ADHD symptoms based on DSM-5 conceptualization (APA, 2013). It includes six items (e.g., "How often do you put things off until the last minute?") in a 5-point Likert scale (0 = *never* to 4 = *very often*). As in the aBAARS-IV, the questions in this scale refer to behaviours that have occurred over the last 6 months. We used the Spanish version of the ASRS-5 that was administered in the original study (Coll-Martín et al., 2021). Cronbach's alpha reliability scores in our final sample were .60, which is close to the .64 from our previous study. Ustun et al. established a threshold of 14 points in the ASRS-5 as the preferred bound for screening purposes.

ANTI-Vea

The ANTI-Vea (Luna et al., 2018) is a cognitive task that provides measures of the functioning of the three attentional networks (alerting, orienting and, executive attention), along with measures of AV and EV (overall performance and decrement across time on task). The simultaneous assessment of these processes controls the order effect bias. The main characteristics of the task are depicted in **Figure 2** and described in Sample Selection and Study Procedure section. We used the online version of the ANTI-Vea, which is freely available in multiple languages at https://anti-vea.ugr.es (Coll-Martín, Román-Caballero, et al., 2023). The ANTI-Vea has been psychometrically validated in lab

and online settings with no substantial differences between the scores analysed in each task version (Luna, Roca, et al., 2021).

In the present study, we focused on two types of ANTI-Vea measures. For AV, we assessed lapses, defined as trials with an excessively slow RT (i.e., RT > 600 ms) or no response to the red down counter. For EV, we measured hits, defined as trials in which the infrequent displacement of the central arrow is correctly detected. For each of the two measures, we considered both the overall task performance and the slope of the vigilance decrement across the six blocks of the task. This decrement manifests as an increase in the lapse rate and a decrease in the hit rate across blocks of trials. The internal consistency scores of the four indices were estimated using a permutation-based split-half approach (Parsons et al., 2019) with 10,000 random splits. The Spearman-Brown (SB) corrected coefficients in our final sample (see **Table 2**) were arguably close to the corresponding .96, .94, and .78 obtained in the original study (Coll-Martín et al., 2021) for lapse overall, hit overall, and lapse slope, respectively. The exception was the slope of hits, for which the reliability score in our sample (r_{SB} = .61) was substantially higher than in the original (r_{SB} = .27).

Data Analysis

As preregistered, data were preprocessed and analysed based on the original study (Coll-Martín et al., 2021). The entire workflow has been run and documented in three reproducible R scripts (Version 4.2.1; R Core Team, 2022). We used the *tidyverse* collection of R packages (Wickham et al., 2019) for most of the data treatment and output visualization.

For the selection of ADHD symptom measures, we considered the period elapsed from the initial survey until the completion of the task. If this time interval was 90 days or less (44.2% participants of final sample), we used the questionnaires from the first survey.

Conversely, if the period was longer than 90 days (53.8% participants of final sample), we used the ADHD self-reports they completed in the second phase of the study.[13] The score for each ADHD symptom scale (i.e., the cBAARS-IV, the aBAARS-IV, and the ASRS-5) was the sum of its items. Applying the procedure of the original study (Coll-Martín et al., 2021), the distribution of the cBAARS-IV and the aBAARS-IV scores were compared with a simulated normative sample.

Figure 1 shows the task sessions and participants excluded from the ANTI-Vea. Compared to the final sample, the level of ADHD symptoms among the participants excluded due to poor performance (i.e., more than 25% errors in ANTI trials) was negligibly lower for symptoms in childhood ($d_{\text{cBAARS-IV}}$ = –0.16) and negligibly to slightly higher for adult symptoms ($d_{\text{aBAARS-IV}}$ = 0.03; $d_{\text{ASRS-5}}$ = 0.22). We calculated the percentage of lapses for AV stimuli and the percentage of hits for EV stimuli for each participant. Of the total number of lapses, 87.2% were due to excessively slow RT, while the remaining 12.8% were due to no response. For both lapse and hit percentages, vigilance decrement was calculated by estimating the linear slope of the outcome across the six blocks of the task. We used the *plyr* R package (Wickham, 2011) to compute the reliability of EV and AV measures.

Regarding our preregistered statistical hypotheses, we distinguished between primary and secondary vigilance outcomes. Primary outcomes were the slopes of vigilance decrement across time on task, while secondary outcomes were the overall vigilance scores. Therefore, since the slope of lapses is positive while that of hits is negative, we hypothesized a positive correlation between the slope of lapses and childhood symptom severity measured with the cBAARS-IV (H1), as well as a negative correlation between the slope of hits and adult symptom severity measured with the

[13] There were six participants (2.0% of final sample) for whom more than 90 days elapsed from the initial survey to the task (*Mdn* = 144), but a second assessment of ADHD symptoms was not available. For these participants, the symptom measures collected in the baseline survey were used.

ASRS-5 (H2). In the same vein, given that more lapses reflect poorer AV and fewer hits indicate poorer EV, we hypothesized a positive correlation between the overall percentage of lapses and the cBAARS-IV (Sc1), as well as a negative correlation between the overall percentage of hits and the ASRS-5 (Sc2). To focus more closely on the association with late-developing symptoms, H2 and Sc2 were partial correlations controlled for the cBAARS-IV. In parallel, we also tested H1′, H2′, Sc1′, and Sc2′, which represented the set of opposite statistical hypotheses, that is, those hypotheses relating each vigilance component to ADHD symptom severity in the age period opposite to that established in the neurodevelopmental model.[14] **Figure 3** illustrates all the statistical hypotheses tested in our study.

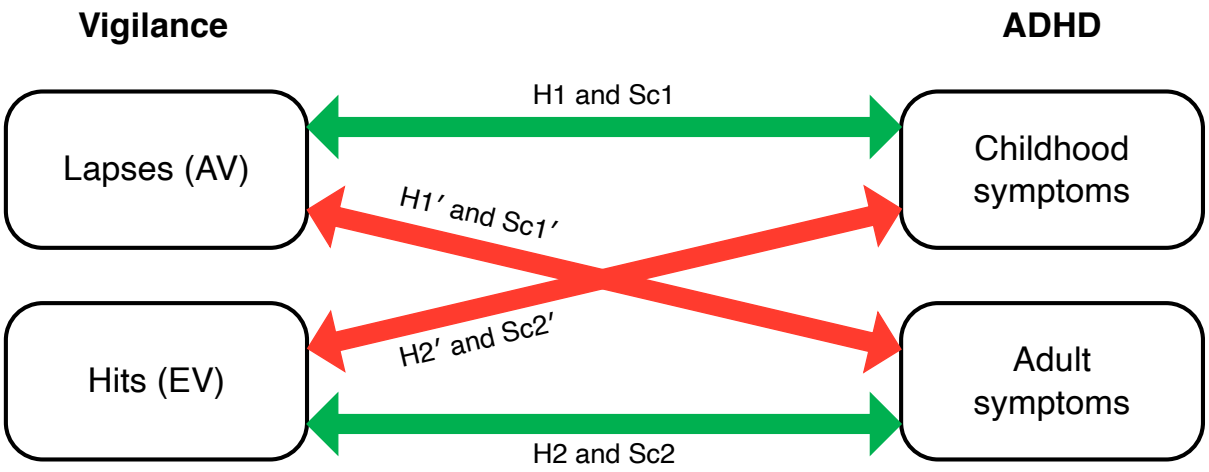


Figure 3. Statistical Hypotheses Tested in Our Study. H1, H2, H1′, and H2′ are primary hypotheses measuring vigilance decrement, while Sc1, Sc2, Sc1′, and Sc2′ are secondary hypotheses measuring overall vigilance. All hypotheses are correlations between the two elements linked by the arrow. Correlations are positive in hypotheses involving lapses (AV) and negative in hypotheses involving hits (EV). Correlations involving symptoms in adulthood are controlled for symptoms in childhood. Green arrows represent statistical hypotheses derived from Halperin and Schulz's (2006) neurodevelopmental model, while red arrows are their opposite statistical hypotheses. AV = Arousal vigilance; EV = Executive vigilance; ADHD = Attention-deficit/hyperactivity disorder.

Since bivariate normality was violated for H1 and H2 (both *p*s < .001), we based on Kendall's rank-order correlation coefficients with the *correlation* R package (Makowski et

[14] The neurodevelopmental model does not rule out the possibility that minimal impairment in executive functions could be related to ADHD symptoms in childhood. However, any such relationship should be substantially lower than nonexecutive dysfunctions (Halperin, 2016; Halperin & Schulz, 2006).

al., 2020) to test the hypotheses. We conducted one-tailed contrasts at a significance level of α = .05 for primary statistical hypotheses (H1 and H2) and their opposite counterparts (H1′ and H2′), as well as α = .025 for secondary statistical hypotheses (Sc1 and Sc2) and counterparts (Sc1′ and Sc2′) to control for multiple comparisons. Moreover, we based on the one-tailed limit of the 95% confidence interval (CI; corrected for multiple comparisons when applicable) to test whether the effect sizes we obtained were significantly lower, in absolute terms, than the effect sizes of interest[15] (for the effects of interest, see Table 1 and Supplemental Table 2). Additionally, we analysed the robustness of the results by performing a multiverse analysis (Steegen et al., 2016) for each statistical hypothesis. Finally, we explored the reach of the neurodevelopmental model to the ANTI-Vea core indices that measure arousal or executive processes.

Sample Size Justification

For the sample size justification, we followed Lakens's (2022) guidelines (see **Supplemental Text 1** for a more detailed report prior to data analysis). The sample size collected in the first and second phases of the study (see **Figure 1**) was determined by the financial resources provided by our funders for participant payment. Our final sample consisted of 292 university students (49.0% women, 51.0% men; 18–30 years, *M* = 21.7, *SD* = 2.7; see **Figure 4A**). Most of the participants were Spanish (95.2%) and current university students (92.1%).[16] Five individuals (1.7%) reported a prior diagnosis of ADHD.

To appraise the informational value of the sample size for our preregistered hypotheses, we considered two types of effect size: the smallest effect size of interest (SESOI) and the expected effect size. As preregistered, we determined a true SESOI of ρ = | .2 | based on a combination of influential expert opinions (Willcutt et al., 2005),

[15] In statistical notation, this tests H_0: $|\rho| \geq |\text{effect of interest}|$ versus H_1: $|\rho| < |\text{effect of interest}|$.
[16] Technically, 22 participants of the final sample (7.5%) were former university students. Additionally, due to an investigator error, one participant was neither pursuing nor had any previous university studies.

measurement error issues (Gignac & Szodorai, 2016), and recognition of heterogeneity in ADHD etiopathophysiology (Luo et al., 2019). According to recent considerations on measurement error in cognitive-behavioural tasks (Parsons et al., 2019), we based on the observed SESOI, which for correlations is obtained as follows:

$$r = \rho \sqrt{\text{reliability}(x) \times \text{reliability}(y)}, \quad (1)$$

where *x* and *y* are the two measures of the correlation. This formula provided the observed SESOIs as Pearson's correlation coefficients (*r*) for each of the four statistical hypotheses tested in our sample.

For the expected effect sizes, we based on the corresponding four Kendall's τ values from the original study (Coll-Martín et al., 2021). To counteract the potential overinflation of the effect sizes in the original study, we applied the Perugini et al.'s (2014) correction for replications. Following Gilpin's (1993) formula, we transformed Kendall's τ values into Pearson's *r* values. These Pearson's coefficients, along with the reliability of the measures in the original study, were used to estimate the true expected effect sizes through the Spearman's (1904) correction for attenuation formula—which is a rearrangement of **Equation 1**. Finally, each true expected effect was input into **Equation 1** to obtain the *r* values that would be expected to be observed in our sample.

After estimating the two types of effects of interest, namely the SESOIs and the expected effects, we conducted simulations to compute the statistical power achieved to detect these effects across each of our four statistical hypotheses. Using the *faux* R package (DeBruine, 2021), we input the eight Pearson's coefficients and run 10,000 simulations to conduct the corresponding hypotheses tests for Kendall's coefficients.[17] The minimal

[17] Although H2 and Sc2 are partial correlations controlled for the cBAARS-IV, they were simulated as zero-order correlations. This was to avoid that a null correlation between the cBAARS-IV and EV might overestimate the size of the partial correlation between the ASRS-5 and EV.

statistically detectable effect (i.e., the critical τ value) was also estimated for the primary and the secondary statistical hypotheses.

The results of the power analysis for the effect sizes of interest are shown in **Table 1** (for the power analysis of the opposite statistical hypotheses, see **Supplemental Table 2**). With one exception, the statistical power to detect at least one of the two effects of interest was greater than 80% for each statistical hypothesis. Only for Sc2 the power achieved was at best slightly suboptimal (1 – β = .69). Furthermore, the SESOIs of all the hypotheses were higher than the critical τ value. Taking together, our sample size provides an arguably acceptable informational value with respect to the statistical hypotheses derived from the Halperin and Schulz's (2006) neurodevelopmental model.

Table 1

Effect Sizes of Interest and Achieved Power to Detect Them Across Each Statistical Hypothesis

Hypothesis	Smallest effect of interest			Expected effect (from Coll-Martín et al., 2021)		
	r	τ	Power (1 – β)	*r*	τ	Power (1 – β)
H1	.16	**.10**	.82	.19	**.12**	.94
H2	–.12	**–.08**	.62	–.16	**–.10**	.83
Sc1	.19	**.12**	.87	.10	.06	.36
Sc2	–.15	**–.10**	.69	.11	.07	0

Note. N = 292. H1 and H2 are the primary statistical hypotheses, while Sc1 and Sc2 are the secondary statistical hypotheses. Kendall's τ values come from the Pearson's *r* values used to conduct the 10,000 simulations for power analysis (see Gilpin, 1993, for the formula to transform the correlation coefficients). Statistical power corresponds to one-tailed tests for Kendall's coefficients, with α = .05 for primary statistical hypotheses and α = .025 for secondary statistical hypotheses. Kendall's τ values above the minimal statistically detectable effect (τ = |.06| for primary hypotheses; τ = |.08| for secondary hypotheses) are in bold.

Results

ADHD Self-Reports

Figure 4B–D shows the distribution of ADHD symptoms in the three scales, and **Figure 4B–C** also compares it to an estimated normative sample (for a detailed procedure and statistical report, **see Supplemental Text 2**). Taking together, ADHD symptoms in our sample of young university students was higher than in the general population of adults. Even so, the spread and variability within each scale did not seem to differ from those observed in an estimated normative sample.

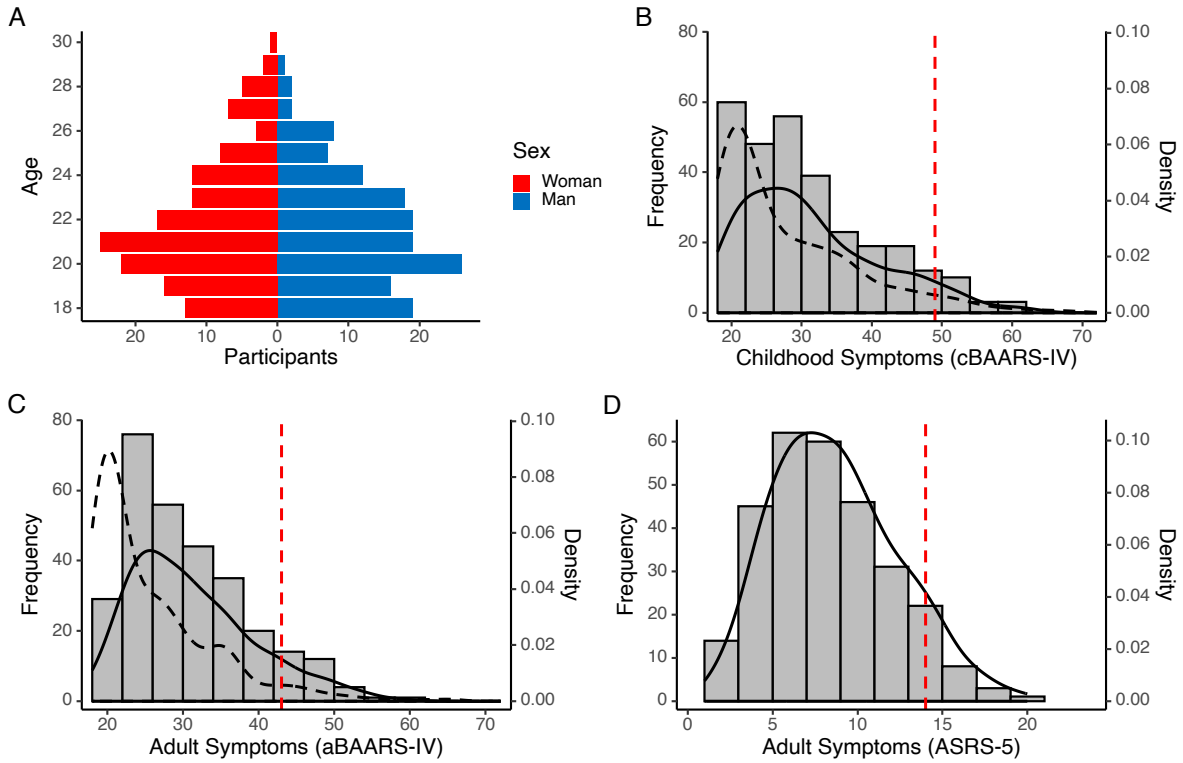


Figure 4. Basic Demographics and Distribution of Total ADHD Symptom Scores for Each of the Three Scales Compared to an Estimated Normative Sample. *N* = 292. **Panel A**: Age-sex pyramid. **Panels B–D**: Histogram and black solid line represent the frequency and density curve of ADHD total scores in the study sample. The vertical dashed red line represents a threshold to identify individuals at risk of ADHD. **Panels B and C**: The dashed black line represents the density curve of ADHD total scores in an estimated normative sample. This normative and representative sample was obtained by extracting 292 quantiles from a large simulated sample (*N* = 10,000) from the percentile values available in (Barkley, 2011). ADHD = Attention-deficit/hyperactivity disorder; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current symptoms; ASRS-5 = Adult ADHD Self-Report Screening Scale for DSM-5.

Unsurprisingly, the cBAARS-IV (M = 31.37, SD = 9.82), the aBAARS-IV (M = 31.16, SD = 8.14), and the ASRS-5 (M = 8.74, SD = 3.66) showed significant positive correlations among them, with effect sizes that are considered large in the field (Gignac & Szodorai, 2016). Concretely, for the cBAARS-IV with the aBAARS, $r(290) = .53$, $p < .001$, for the cBAARS-IV with the ASRS-5, $r(290) = .34$, $p < .001$, and for the aBAARS-IV with the ASRS-5, $r(290) = .75$, $p < .001$. As expected, the correlation between the two measures of symptoms in adulthood was higher than those between these measures and the one of symptoms in childhood (both ps < .001). Additionally, the correlation with childhood symptoms was stronger when the adult symptom items also corresponded to DSM criteria, as in the case of the aBAARS-IV, than when they were intended to reflect the adult-specific presentation of ADHD symptoms, as with the ASRS-5 ($z = 5.11$, $p < .001$).

Task Performance

Table 2 shows the means and standard deviations of the percentages of lapses and hits for both the vigilance decrement and the overall performance. In line with the original study (Coll-Martín et al., 2021), we found a positive linear slope of lapses, $t(291) = 7.18$, $d_z = 0.42$, and a negative linear slope of hits, $t(291) = –9.83$, $d_z = –0.58$, across time on task. Specifically, the lapses increased from 7.96% in Block 1 to 13.38% in Block 6, while the hits decreased from 78.94% in Block 1 to 68.90% in Block 6. Interestingly, the correlation between lapses and hits was not significant for either the overall scores ($r = –.09$, $p = .10$) or the decrement slopes ($r = –.11$, $p = .07$), revealing a dissociation between both vigilance components.

Preregistered Hypotheses

Table 2 shows the results of the preregistered hypotheses. For the primary outcomes (i.e., vigilance decrement), none of the statistical hypotheses—namely H1, H2, H1′, and H2′—were statistically significant (all ps > .194). Even more so, each of these

Table 2

Descriptive Statistics, Internal Consistency, and Kendall's Correlation Coefficients With ADHD Symptoms for Arousal Vigilance (AV) and Executive Vigilance (EV) According to Preregistered Hypotheses

Measure of vigilance	*M*	*SD*	r_{SB}	Correlation with ADHD symptom severity			
				Neurodevelopmental model		Opposite statistical hypotheses	
				Childhood ADHD	Adult ADHD	Childhood ADHD	Adult ADHD
Primary				H1	H2	H1′	H2′
% Lapse slope (AV)	1.09	2.60	.67	.00 [.07$^{<EI}$]			.01 [.07$^{<EI}$]
% Hit slope (EV)	–2.17	3.78	.61		–.03 [–.10$^{<EI}$]	.04 [–.03$^{<EI}$]	
Secondary				Sc1	Sc2	Sc1′	Sc2′
% Lapse overall (AV)	10.57	17.69	.98	.07† [.14]			–.00 [.07$^{<EI}$]
% Hit overall (EV)	73.08	18.28	.95		–.04 [–.12]	–.09* [–.16]	

Note. N = 292. Correlation with symptoms in childhood are zero-order correlations with the Barkley Adult ADHD Rating Scale-IV: Childhood symptoms (cBAARS-IV). Correlations with adult symptoms are partial correlations with the Adult ADHD Self-Report Screening Scale for DSM-5 (ASRS-5) after controlling for the cBAARS-IV. Correlational tests are positive for AV and negative for EV. Neurodevelopmental model encompasses the preregistered hypotheses derived from the Halperin and Schulz's (2006) model. Opposite statistical hypotheses are included for comparison purposes. One-tailed, 95% (primary outcomes) or 97.5% (secondary outcomes) limits of confidence intervals are in brackets. ADHD = Attention-deficit/hyperactivity disorder; r_{SB} = Spearman-Brown split-half reliability coefficient.

$^{\dagger}p_{uncorrected} < .05$, one-tailed. $^{*}p_{corrected} < .05$, one-tailed. $^{<EI}p_{corrected} < .05$, one-tailed inferiority test: the limit of the confidence interval excludes at least one of the two effects of interest (EIs; for the EIs, see Table 1 and **Supplemental Table 2**).

four correlation coefficients was significantly smaller in size than at least one of the two effects of interest (all *p*s < .05). Specifically, all correlation sizes, except that of H2, were lower than their corresponding SESOI. Moreover, the coefficients from H1 and H2 were smaller than their expected effect sizes.

Regarding the secondary outcomes (i.e., overall vigilance scores), the expected positive correlation between the cBAARS-IV and the percentage of lapses (Sc1) was only significant before correcting for multiple comparisons, $\tau = .07$, $p_{uncorrected} = .047$, $p_{corrected} = .094$. In addition, the hypothesised negative correlation between the ASRS-5 and the percentage of hits (Sc2) was not significant, $\tau = -.04$, $p_{uncorrected} = .128$. As expected, the correlation between the ASRS-5 and the percentage of lapses (Sc1′) was not significant, $\tau = -.00$, $p_{uncorrected} = .523$, and its effect size was smaller than the SESOI ($\tau_{SESOI} = .10$; $p_{corrected} < .05$). Surprisingly, the negative correlation between the cBAARS-IV and the percentage of hits (Sc2′) was significant, $\tau = -.09$, $p_{corrected} = .033$.

Multiverse Analyses

To generate the multiverse of analyses, we identified five decision points in the analytical process with more than one apparently reasonable choice, the first being the preregistered option: participants with more than 90 days from survey to task (*n* = 6; retained vs. excluded), poor task performers (*n* = 14; excluded vs. retained), task threshold to compute the vigilance indices (absolute vs. relative), correlation coefficient (Kendall vs. Spearman vs. Pearson) and type of correlation (zero-order vs. partial[18]). Furthermore, we incorporated the aBAARS-IV as a secondary measure of symptoms in adulthood (preregistered as Ss2). This provided 48 valid specifications to analyse the correlation between each of the four vigilance indices and each of the three ADHD measures (i.e., 576 total estimates). The distribution of the estimates within the four multiverses is

[18] For the ASRS-5, partial correlation was the preregistered option.

illustrated in **Figure 5** (for a more comprehensive report of the method and results of these analyses, see **Supplemental Table 4**).

Figure 5A–B shows that the lack of significant findings in the preregistered H1 and H2 was robust across the different analytical options, including the use of a secondary measure of ADHD symptoms in adulthood (i.e., the aBAARS-IV). Regarding their opposite counterparts, we found that 20 out of the 96 analytical scenarios in H1′ (20.8%) were statistically significant. A closer inspection revealed that most of these significant findings (90.0%) computed the lapse index based on a relative threshold (i.e., trials with an RT higher than the participant's mean plus one standard deviation or trials with no response). This type of lapse index computation exhibited very low reliability scores, albeit probably biased upwards when poor performers were retained for the analyses (see **Supplemental Figure 1**). On the contrary, for the analytical combinations that employed an absolute threshold to compute lapses, the rate of significant findings was below chance (2.1%). As for H2′, the null results were consistent across all analytical choices.

In contrast to the primary hypotheses, secondary hypotheses (**Figure 5C–D**) showed a rather mixed pattern of statistical significances across analytical scenarios. Sc1 yielded 8.3% significant contrasts, which doubled to 16.7% when only analyses with an absolute threshold for lapses were considered. Regarding Sc2, the percentage of statistically significant scenarios dramatically varied from 16.7% in the ASRS-5 to 95.8% in the aBAARS-IV. This difference between adult measures of ADHD symptoms was also manifested in Sc1′, where 20.8% and 54.2% analytical combinations were significant for the ASRS-5 and the aBAARS-IV, respectively. Sc2′, the only statistical hypothesis whose preregistered contrast was significant, yielded 35.4% scenarios with positive findings. Since most of our analytical options do not follow the linear regression model, the robustness of the secondary hypotheses could not be inferentially evaluated with available approaches (e.g., Simonsohn et al., 2020).

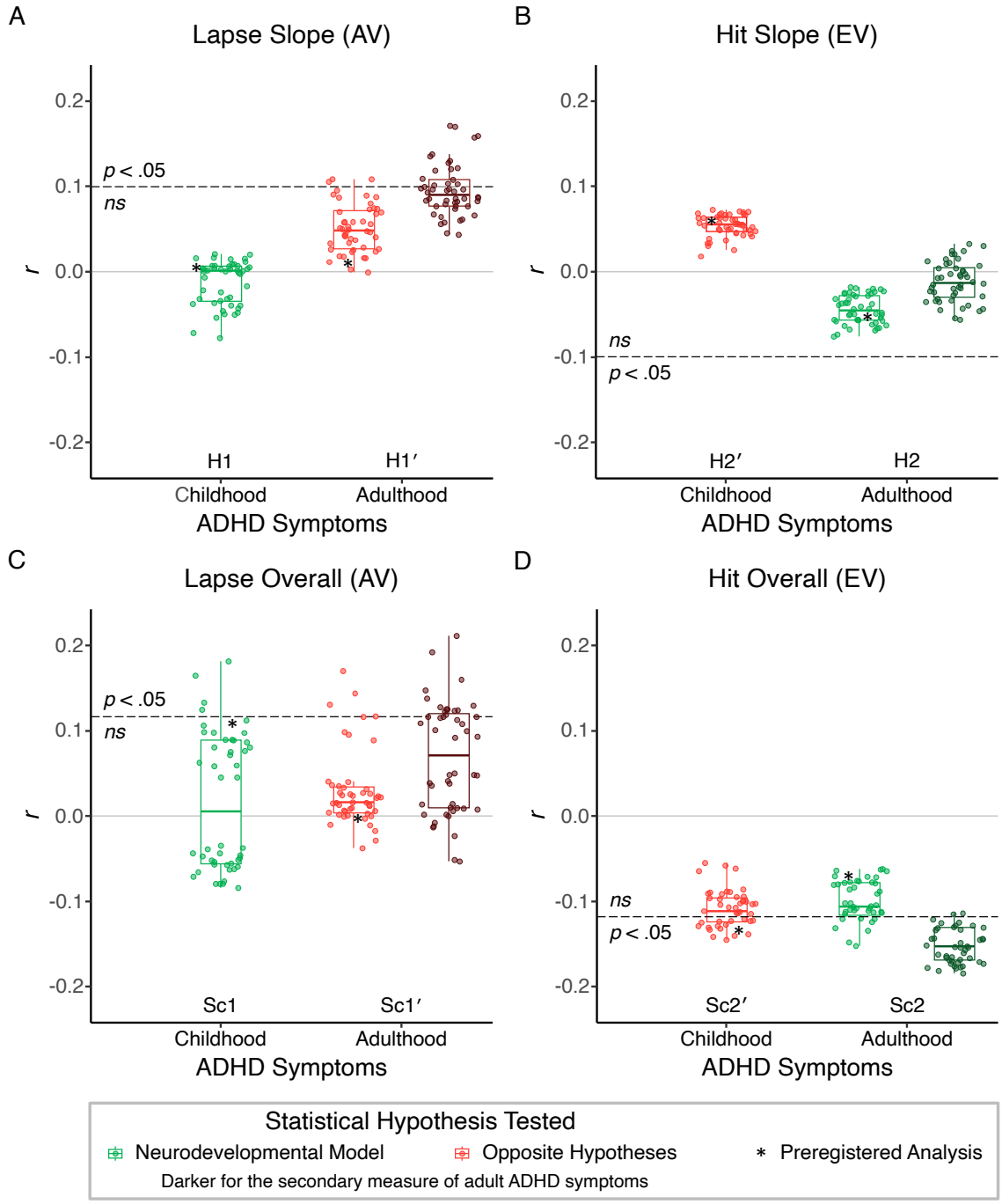


Figure 5. Distribution and Box Plot of Correlation Coefficients of Arousal Vigilance (AV) and Executive Vigilance (EV) With ADHD Symptoms Across the Multiverse of Reasonable Analytical Options. *N* = 292. Kendall and Spearman correlation coefficients were transformed into Pearson's *r* values to allow for comparison of effects (formulas based on Gilpin, 1993). ADHD symptoms in childhood and adulthood are measured with the Barkley Adult ADHD Rating Scale-IV: Childhood symptoms (cBAARS-IV) and the Adult ADHD Self-Report Screening Scale for DSM-5 (ASRS-5), respectively. The secondary measure of ADHD symptoms in adulthood refers to the Barkley Adult ADHD Rating Scale-IV: Current symptoms (aBAARS-IV). The horizontal dotted grey lines represent the significance threshold for the correlations. This significance threshold is based on one-tailed tests (positive for AV and negative for EV), corrected for multiple comparisons in secondary statistical hypotheses (i.e., **Panels C and D**). **Panel D:** Due to the variety of statistical approaches, one statistically significant coefficient in the ASRS-5 (*r* = –.117) and two in the aBAARS-IV (*r*s = –.116 and –.117) appear above the significance threshold line, while one nonsignificant coefficient in the

Exploratory Analyses of all Arousal and Executive Task Indices

To analyse the relationships between ADHD symptoms and both arousal and executive processes more broadly, we computed all ANTI-Vea indices of these neurocognitive domains (see **Supplemental Table 5** for descriptive statistics and correlations among them). They included eight measures of arousal: lapses in AV trials (slope and overall), mean RT in AV trials (slope and overall), standard deviation of the RT in AV trials (slope and overall), and the alerting index (i.e., no tone minus tone conditions) in ANTI trials (RT and percentage of errors). They also included six executive measures: hits in EV trials (slope and overall), false alarms in EV trials (slope and overall), and the congruency index (i.e., incongruent minus congruent conditions) in ANTI trials (RT and percentage of errors).

The set of 14 ANTI-Vea indices was used to predict each of the three measures of ADHD symptoms (i.e., the cBAARS-IV, the ASRS-5, and the aBAARS-IV) through multiple linear regression models. To select the predictors of each model, we employed a bidirectional stepwise regression method aimed at minimising the Akaike information criterion (AIC). The results (**Table 3**) showed that three, six, and five ANTI-Vea indices were selected to predict the cBAARS-IV, the ASRS-5, and the aBAARS-IV, respectively. The measure of symptoms in childhood was also entered and selected in both models of adult symptoms. Crucially, both arousal and executive task indices uniquely predicted ADHD symptoms across the three models.

Discussion

Are alterations in AV associated with higher ADHD symptoms in childhood (H1), while deficits in EV are related to late-developing ADHD symptoms in adulthood (H2)? This dissociation, predicted by the Halperin and Schulz's (2006) neurodevelopmental

Table 3

Multiple Regressions of Three Models of ADHD Symptoms as a Function of Arousal and Executive Task Indices

Predictor	Model 1: Childhood (cBAARS-IV)		Model 2: Adulthood (ASRS-5)		Model 3: Adulthood (aBAARS-IV)	
	***B* (*SE*)**	**β**	***B* (*SE*)**	**β**	***B* (*SE*)**	**β**
Arousal						
Lapse overall	0.10 (0.03)	.18**	0.05 (0.02)	.23*		
RT *M* overall			–0.01 (0.00)	–.18		
RT *SD* overall					0.04 (0.01)	.15**
Alerting RT			0.01 (0.01)	.08	0.03 (0.01)	.13**
Executive						
Hit overall	–0.07 (0.03)	–.12*	–0.02 (0.01)	–.11*		
FA slope	0.89 (0.33)	.15**			–0.49 (0.23)	–.10
FA overall			0.12 (0.04)	.17**		
Congruency RT			0.01 (0.01)	.11*	0.03 (0.02)	.11*
Congruency errors					0.15 (0.10)	.07
R^2	.08					
ΔR^2			.07		.07	

Note. *n* = 289 (three participants from the final sample were dropped due to an incorrect task setting for FAs). The predictors of each model were selected from a set of 14 relevant task indices (eight of arousal and six executive) through a bidirectional stepwise regression method based on the Akaike information criterion (AIC). The two models predicting adult symptoms also include symptoms in childhood (i.e., the cBAARS-IV) as a predictor variable. As such, the incremental variance (ΔR^2) of these models indicates the proportion of explained variance above and beyond that accounted for by a model only including childhood symptoms. ADHD = Attention-deficit/hyperactivity disorder; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood symptoms; ASRS-5 = Adult ADHD Self-Report Screening Scale for DSM-5; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current symptoms; RT = Reaction time; FA = False alarm.

*$p < .05$, one-tailed (left-tailed for hit overall and right-tailed for the rest of predictors, as per Coll-Martín et al., 2021). **$p < .01$, one-tailed.

model, was supported in Coll-Martín et al. (2021). Here we designed a preregistered, closed replication of that study specifically aimed at testing the model. Based on a dimensional framework of ADHD, we assessed retrospective and current self-reported symptoms in a community sample of university students who performed an online cognitive task: the ANTI-Vea. The final sample size (N = 292) allowed our preregistered hypotheses to achieve an arguably acceptable statistical power for the effects of interest. Despite this, our main results failed to replicate the previous study: Indeed, only the unexpected (i.e., opposite to the neurodevelopmental model) negative correlation between EV and symptoms in childhood was significant. Although multiverse and exploratory analyses yielded some significant findings, they neither supported the dissociation pattern proposed by the neurodevelopmental model.

Our unsuccessful replication of Coll-Martín et al. (2021) was rather clear and consistent. Not only were our correlation coefficients for H1 and H2 not significantly different from zero, but they also were significantly smaller in size than an attenuated estimation of the effect sizes in the original study. Even testing H2 as a simple correlation, as conducted in the original study, was not significant. The minimal conceptual differences between the original study and this replication—namely, the task setting (lab vs. online) and the composition of the university sample (mostly women from the same degree subject vs. sex-balanced sample from the entire student community)—were unlikely to account for the huge discrepancies in the results.[19] Instead, it is far more plausible that the findings of H1 and H2 in the original study were false positives, given its exploratory approach and lower control of the Type I error. This highlights the importance of close replications with adequate control of both types of error rates to improve the reliability of a given literature.

[19] We tested H1 and H2 in our subsample of women. None of the Kendall's coefficients were greater in size than the minimal statistically detectable effect in the full final sample. The same was true when H2 was tested as a simple correlation, as in the original study.

In contrast to Coll-Martín et al. (2021), our study failed to support the neurodevelopmental model. Notably, unlike most previous empirical research, our study selected a clear and valid nonexecutive measure to test the first prediction of the model. Specifically, the AV indices from the ANTI-Vea are thought to reflect the noradrenergic mechanisms of the hindbrain mediating arousal (Luna et al., 2018). According to the neurodevelopmental model, alterations in this subcortical system are a potential cause of ADHD and remain stable throughout the lifetime. The conceptual and empirical dissociation of AV from EV in the ANTI-Vea—as evidenced by the low correlation found in our data—support the task as a useful tool in the neurocognitive research on ADHD.

Another measurement issue, in this case related to the second prediction of the neurodevelopmental model, was the use of two conceptually different scales of ADHD symptoms in adulthood. Our primary measure was the ASRS-5, which assess the adult presentation of ADHD, including adult-specific items (e.g., frequency of difficulties in unwinding and relaxing when having time to oneself). In addition, we administered the aBAARS-IV as a secondary measure, which consists of traditional DSM items with identical behaviour and wording (e.g., frequency of fidgeting with hands or feet or squirming in seat) as its childhood counterpart (i.e., the cBAARS-IV). While the debate between using adult-specific versus DSM-based items for ADHD diagnosis has been addressed elsewhere (e.g., Sibley et al., 2012), differences in sensitivity of these measures to neurocognitive variables has not been formally studied. Although descriptively, our multiverse and exploratory analyses suggest that DMS-based items could be somewhat more sensitive to attentional functioning than adult-specific items, especially considering that the former measure is potentially more affected when controlling for childhood symptoms. Alternatively, the higher reliability and skewness of the aBAARS-IV compared to the ASRS-5 could explain such differences in the size of the correlations.

Is impaired vigilance decrement core to ADHD?

We used measures of vigilance decrement for our primary hypotheses (H1 and H2) and their opposite counterparts (H1′ and H2′). Compared to overall vigilance scores, the decline in performance over time has been less studied in ADHD, despite being considered by some as the defining feature of sustained attention (Huang-Pollock et al., 2012; L. Tucha et al., 2017). Surprisingly, our four correlation coefficients were significantly smaller in size than at least one of their corresponding effects of interest. Furthermore, the lack of a correlation statistically different from zero in the expected direction was robust across the sets of reasonable analytical options, as shown in the multiverse analyses. Only one exploratory analysis in our stepwise multiple linear regression, namely the positive association between the rate of false alarms and childhood symptoms, was significant.

Considering that our analyses had an acceptable statistical power, our lack of significant results for the relationship between vigilance decrement and ADHD symptoms was theoretically unexpected. However, they are in line with other clinical and community studies that used moderate to large samples (i.e., $N > 150$; Aduen et al., 2020; Huang-Pollock et al., 2020; Huang-Pollock et al., 2012). In contrast, one study comparing ADHD children with controls ($n > 200$ per group) found that the former had a higher vigilance decrement in response speed and consistency (each $d_s > 0.3$; Weyandt et al., 2017). While this discrepancy needs to be solved, one possibility might be that vigilance decrement is not substantially related to ADHD symptomatology. On the contrary, momentary attentional fluctuations, the other component of vigilance (Esterman & Rothlein, 2019), could be behind the impaired sustained attention in ADHD. In our study, preliminary support for this idea comes from the association between a higher rate of lapses and higher ADHD symptom severity in 30.1% of the multiverse scenarios with an absolute lapse threshold as well as in the exploratory multiple regression models.

Is the neurodevelopmental model a useful theory?

Looking at the big picture, the most consistent pattern across our results was the failure to support the neurodevelopmental dissociation proposed by the Halperin and Schulz's (2006) model. In our preregistered analyses, we found that the rate of hits (EV) negatively correlated with symptoms in childhood but not in adulthood, which is the opposite to the theoretical prediction. Furthermore, our exploratory regression analyses were far from suggesting any dissociation: Both arousal and executive processes independently predicted ADHD symptoms in childhood and adulthood. Although contrary to Coll-Martín et al. (2021), our lack of a dissociation supporting the model is in line with several studies (Coghill, Hayward, et al., 2014; Coll-Martín, Sonuga-Barke, et al., 2023; Gmehlin et al., 2016; McAuley et al., 2014; van Lieshout et al., 2013).

Going further, some of the core neurocognitive phenomena for which the neurodevelopmental model was formulated are arguably better explained by alternative accounts. For example, the neurodevelopmental model overemphasises the prefrontal cortex subserving executive functions as the only area responsible for changes in ADHD symptoms across the lifespan. This contrasts with evidence suggesting that executive functions are implemented in distinct neural networks involving multiple brain regions (e.g., (Dosenbach et al., 2008). Furthermore, the neurodevelopmental model proposal of brain lesions or insults as causes or developmental moderators of ADHD is inconsistent with the current neurodiversity model, which considers ADHD as the extreme expression of a temperamental trait (Sonuga-Barke & Kostyrka-Allchorne, 2023; Sonuga-Barke & Thapar, 2021). The latter model can provide a more straightforward explanation for why brain stimulation through modern neurotherapeutics or computer-based cognitive training has at best limited effects on core ADHD symptoms (Rubia, 2022; Westwood et al., 2023). In addition, the heterogeneity of neurocognitive alterations associated to ADHD symptoms

within each stage of development and its change trajectories is better accounted for by models of multiple developmental pathways (Nigg et al., 2005; Sonuga-Barke, 2005).

Taking together, it can be stated that the Halperin and Schulz's (2006) neurodevelopmental model in its current form has substantially lower explanatory capacity than other alternative theories in the field. More broadly, assuming that there is no developmental dissociation in the neurocognitive processes underlying ADHD symptoms has important implications. If late-developing symptoms are pathophysiologically similar to symptoms in childhood, then late-onset ADHD cases—or a substantial proportion of them—would be close in nature to child-onset ADHD. Therefore, translational interventions for ADHD symptoms should be designed to target the same underlying neurocognitive processes regardless of the age of individuals and impairment onset.

Limitations

We have identified three main limitations in the design of our study. The first regards the generalization of our findings. Although our community sample was sex balanced, it was mainly made up of university students, which is not fully representative of the young or general adult population. Indeed, ADHD symptoms in childhood have been associated with lower educational attainment in adulthood (Galéra et al., 2012; Pingault et al., 2011). Despite this sampling bias, our statistical analyses did not suggest that ADHD symptom scores were lower or more homogeneous in our sample than in a representative community sample. In any case, replication of our findings with a more representative sample is warranted. Additionally, although the dimensional framework assumes that the neurocognitive correlates of ADHD symptoms remain constant throughout a continuous trait, the empirical extension of our results to clinical populations is crucial.

Second, our assessment of ADHD symptoms in childhood and adulthood relied solely on self-reports, with the measurement of childhood symptoms being retrospective.

Although self-reported measures of ADHD symptoms may capture a broader dimension, they seem to be less sensitive than parent-reports to some neurocognitive outcomes (Du Rietz et al., 2016; Riglin et al., 2022). In addition, the validity of retrospective reports of ADHD symptoms has been questioned due to potential recall bias, with longitudinal studies finding a modest correlation between prospective and retrospective parent ratings of symptoms in childhood (e.g., r = .39; von Wirth et al., 2021). However, given the fluctuating trajectory of ADHD symptoms throughout development (Sibley et al., 2022; Stern et al., 2020), part of the mismatch between both measures may be better explained by differences in the time span assessed by each symptom scale (e.g., last 6 months vs. whole childhood) rather than recall bias. Of note, Lundervold et al. (2021) found a 7-year test–retest reliability score of .89 for a retrospective self-report measure of ADHD symptoms in childhood. Looking on the bright side, our study design held constant the rater and the time of assessment, thereby controlling for biases related to these factors. In any case, the integration of distinct but complementary assessment methods is fundamental to advance the understanding of the neurocognitive processed associated to ADHD symptoms across lifespan.

The third limitation is about the statistical conclusion validity of our preregistered hypotheses. Our sample size was relatively large in this literature. Even so, it was far from achieving a reasonable statistical power to test for differences between correlations, the most pertinent analysis for contrasting the hypotheses of the neurodevelopmental model in our design. In fact, we would have needed around twice our sample size to perform such a test. Descriptively, none of the differences in the correlation coefficients between the ADHD measures of each vigilance outcome (e.g., τ in H1 minius τ in H1′; see Table 2) is higher in size than any SESOI (see Table 1 and Supplemental Table 2). Despite this constraint that prevented us from comparing correlations inferentially, our study employed a thoughtful power analysis for single nonparametric correlations, accounting for

measurement error to estimate the effects of interest. This approach not only allowed for transparent and accurate reporting of statistical power for each preregistered statistical hypothesis, but also enabled that all our primary hypotheses were statistically conclusive—by comparing each CI limit with the effects of interest. Although these practices are essential to evaluate the informational value of an empirical study, they are rarely implemented (Lakens, 2022; Parsons et al., 2019).

Conclusion

Assuming a dimensional framework, our unsuccessful preregistered close replication of Coll-Martín et al. (2021) did not support the Halperin and Schulz's (2006) neurodevelopmental model of ADHD: Only one unpredicted correlation was significant. Neither our exploratory findings were in line with the developmental dissociation hypothesized by the model: Both arousal and executive task indices uniquely predicted both ADHD symptoms in childhood and adulthood. Based on our findings, translational interventions for ADHD symptoms should target the same underlying attentional deficits regardless of the age of individuals and onset of the impairment. Future studies should include complementary assessment methods of ADHD symptoms, clinical groups, and other neurocognitive domains to qualify and extend our tentative recommendations.

Supplemental Materials

SUPPLEMENTAL TEXT 1: Sample Size Justification Report Prior to Conduct the Data Analysis

The following text has been obtained through the Shiny app that accompanies Lakens' (2022) article titled "Sample Size Justification". Note that the output was generated on June 17th, 2022, prior to data analysis. At that time, we did not know the reliability scores of the measures in our sample, and thus the power calculation was based on the values obtained in the original study. Furthermore, we did not know that the normality assumption had been violated, and therefore, the power calculation was based on Pearson's correlation coefficients instead of Kendall's rank-order correlation coefficients, as we ultimately did.

Below are four sections of the sample size justification. Part A contains a description of the population, as well as a description of the resource constraints that determine how much of the population can be sampled. In Part B a description of which effect sizes are of interest is provided. In Part C an overview of the inferential goal of the study is specified. In Part D the sample size that will be collected is reported, and the informational value of the study is evaluated.

A: Sample Description

Description of the population.

```
We have recruited 318 university students (50% women). They were initially
recruited through advertisements on the distribution list of our universit
y. Concretely, a total of 2003 respondents completed an online survey in ex
change for the opportunity to win two prizes of 200 € in a raffle. We exclu
ded participants with invalid responses (i.e., extreme response times or ca
reless responses), serious diseases, psychiatric disorders or conditions ot
her than ADHD, age over 35, no current or former university studies, or rep
eated responses. After exclusions, we invited participants to collaborate i
n the second phase of the research consisting in performing an online cogni
tive task. Invitations were sent out by email until nearly 300 responses we
re collected. They received 6 € as compensation for their voluntary partici
pation. We stratified for sex to have equal representation in our final sam
```

```
ple. The population we want to generalize our findings consists of general
young adults.
```

Can you collect data from the entire population?

```
no
```

Description of resource constraints.

```
The maximum sample size we could collect (N ≈ 300) was determined by the pe
cuniary amount the funder could provide us for participants payment.
```

B: Effects of Interest

Information about the Smallest Effect Size of Interest. The smallest effect size of interest size is specified as a correlation of 0.2. The following details were provided about the smallest effect size of interest:

```
For our planned hypothesis tests H1 and H2 we specify the smallest effect s
ize of interest (SESOI). In the context of ADHD theories based on neurocogn
itive processes, an influential meta-analysis on executive functions in ADH
D considered a moderate difference (ds ≈ 0.5; equivalent to r = 0.24; and t
his, in turn, equivalent to a true correlation (ρ) = .32, Gignac & Szodora
i, 2016) as insufficient to validate the proposed mechanism as central in t
he disorder (Willcutt et al., 2005). Given the heterogeneity in ADHD etiopa
thophysiology, we have set a less exigent SESOI of ρ = .2. Below this thres
hold, the theory would be too imprecise, and probably other neurocognitive
predictors should be added to reframe the theory and make it relevant.


Notably, based on current considerations in cognitive-behavioural measureme
nts (Parsons et al., 2019), we will focus on the observed SESOI, which for
correlations would be obtained as follows:


Observed SESOI (r) = True SESOI (ρ) * √(Reliability (x) * Reliability (y).


Although the final observed SESOI will be computed from the reliability of
the measures in the present study, we provide an estimation from our previo
us study using a virtually identical task (Coll-Martín et al., 2021), wher
e: SESOI would be r = .167 in H1 and r = −.083 in H2 (for the zero-order co
rrelation). Finally, in the likely event that the bivariate normality assum
ption is violated for either H1 or H2, we will use Kendall's τ coefficient
as a measure of both effect sizes. The equivalent coefficient will be obtai
ned from Gilpin (1993).
```

Information about the Minimal Statistically Detectable Effect. The minimal statistically detectable effect is specified as a correlation of 0.10. The following details were provided about the minimal statistically detectable effect:

```
In our previous study we had 5.83% of invalid participants. To be conservat
ive, and given that the present study is online, we plan for 10% of invalid
participants, so 318−32 = 286 valid responses. In G*Power (Version 3.1.9.6)
we compute the critical r-value:


Exact - Correlation: Bivariate normal model
Options: exact distribution
Analysis: Sensitivity: Compute required effect size
Input: Tail(s) = One
Effect direction = r ≥ ρ
α err prob = 0.05
Power (1-β err prob) = 0.8
Total sample size = 286
Correlation ρ H0 = 0
Output: Lower critical r = 0.0974574
Upper critical r = 0.0974574
Correlation ρ H1 = 0.1464436
```

Information about the Expected Effect Size. The expected effect size is based on a previous study. The expected effect size is a Correlation of NA [since there is more than one expected effect, no specific value appears]. An evaluation of the similarity of the previous study with the planned study, a citation of the previous study, and details about the effect size from the previous study are provided below:

```
We based on our previous study (Coll-Martín et al., 2021) to estimate the e
xpected effect size for the present study. Indeed, this planned study can r
easonably be considered a close replication. Both studies recruited partici
pants from the same university, although the original study was more restri
cted to students of specific degrees. Similarly, both studies employed the
same type of questionnaires for ADHD symptoms and a virtually identical tas
k to measure vigilance, being the main difference in the setting where the
task was performed: laboratory (original) or virtual (present replication).
Finally, for both studies the procedure consisted of a first phase where th
e questionnaires were answered, followed by a second phase where the cognit
ive task was carried out.
```

An evaluation of the uncertainty in the effect size estimate in the previous study, and how this is dealt with (e.g., choosing a more consevative estimate) is provided below:

```
In the original study, the effect size was r = .215, 95% CI [.032, .385] fo
r H1 and r = —.225 95% CI [—.379, —.023] for H2. The next section will atte
mpt to deal with uncertainty, along with publication bias.
```

An evaluation of whether the effect size estimate of the previous is unbiased, and if not, any approach to correct for bias, or decisions about the use of a more conservative effect size estimate is provided below:

```
The effects sizes of the original study are likely to be inflated, as they
were the main significant findings that could lead to the publication of th
e manuscript. Applying the suggestion of Perugini et al. (2014) for replica
tion studies, a better estimation of the expected effect size is the closer
-to-zero-bound of the 60% confidence interval in the original study. Theref
ore, the expected effect size for the present study would be r = 137 for H1
and r = —.132 for H2.
```

The following information about the Sensitivity Power Analysis has been provided:

```
A sensitivity analyses with our expected final sample size (N = 286) indica
tes that, for α = .05, a power of 80% correspond to an r-value = |.146| for
one-tailed tests.


Exact - Correlation: Bivariate normal model


Options:    exact distribution


Analysis:   Sensitivity: Compute required effect size
Input:      Tail(s)                     =   One
            Effect direction         =  r ≤ ρ
            α err prob                  =   0.05
            Power (1-β err prob)        =   0.8
            Total sample size           =   286
            Correlation ρ H0            =   0
Output: Lower critical r            =   -0.0974574
            Upper critical r            =   -0.0974574
            Correlation ρ H1            =   -0.1464436
```

```
This effect size for which we have sufficient power to detect is lower than
the SESOI in H1 (r = .167), slightly higher than our expected effect sizes
in H1 (r = .137) in H2 (r = −.132), and far higher than the SESOI in H2 (r
= −.083; for zero-order correlation).
```

C: Inferential Goal

The following information about the inferential goal related to statistical power has been provided.

The inferential goal is to perform a hypothesis test with a certain statistical power, computed by a sensitivity power analysis. The chosen alpha level is 0.05 A justification for the chosen alpha level and desired power (or for a sensitivity power analysis, the achieved power for effects of interest), and details of the power calculation (preferably in reproducible code) is provided below:

```
We have collected a total of 318 participants. In the previous study, we ha
d a 5.83% of invalid responses (due to poor task accuracy) for the main ana
lyses. Here we apply a conservative estimation of a 10% of invalid response
s, leaving a total of (318 − 32) 286 participants with valid answers. Given
this final sample size, and considering a critical α = .05, we achieve the
following statistical power for the effects of interest in one-tailed corre
lations:

-For the SESOI, our power is 88.43% for H1 (r = .167) and 40.40% for H2 (r
= −.083; for zero-order correlation).

-For our expected effect size, our power is 75.15% for H1 (r = .137) and 7
2.35% for H2 (r = −.132).
```

D: Informational Value of the Study

Based on the resource constraints, the effects of interest, and the inferential goals, the following evaluation of the informational value of the study has been provided.

Given the following resource constraints:

```
The maximum sample size we could collect (N ≈ 300) was determined by the pe
cuniary amount the funder could provide us for participants payment.
```

and given a smallest effect size of interest size of Correlation = 0.2, a minimal statistically detectable effect of Correlation = 0.10, an expected effect size of Correlation

= NA, an evaluation of effects one has sufficient power to detect based on a sensitivity power analysis as described below:

```
A sensitivity analyses with our expected final sample size (N = 286) indica
tes that, for α = .05, a power of 80% correspond to an r-value = |.146| for
one-tailed tests.


Exact - Correlation: Bivariate normal model


Options:    exact distribution


Analysis:   Sensitivity: Compute required effect size
Input:      Tail(s)                     =   One
            Effect direction         =  r ≤ ρ
            α err prob                  =   0.05
            Power (1-β err prob)        =   0.8
            Total sample size           =   286
            Correlation ρ H0            =   0
Output: Lower critical r            =   -0.0974574
            Upper critical r            =   -0.0974574
            Correlation ρ H1            =   -0.1464436


This effect size for which we have sufficient power to detect is lower than
the SESOI in H1 (r = .167), slightly higher than our expected effect sizes
in H1 (r = .137) in H2 (r = —.132), and far higher than the SESOI in H2 (r
= —.083; for zero-order correlation).
```

and given the inferential goal based on a sensitivity power analysis with an alpha level of 0.05, the sample size in the planned study will consist a total of 286 participants, each contributing 1 observations. The following additional details about the sample size were provided:

```
The estimation of the final sample size (N = 286; after exclusion of partic
ipants with poor accuracy) from the initial sample size of 328 participants
is based on a protective expectation of 10% invalid responses (almost twice
as much as in the original study).


Moreover, based on current considerations in cognitive-behavioural measurem
ents, our effect of interest considers measurement error as per the followi
ng formula:
```

```
Observed SESOI (r) = True SESOI (ρ) * √(Reliability (x) * Reliability (y)).
```

An explanation of the informational value of the sample size that will be collected, given any resource constraints, the effects of interest, and the inferential goal, is provided below:

```
With an estimated final sample of 286 participants, we expect to reach a de
cent power to test two relevant predictions derived from the Halperin and S
chulz's (2006) neurodevelopmental model. In the case of H1, we are likely t
o have sufficient power for the SESOI (88.43%), which is higher than the ex
pected effect size. As per H2, although our study is rather underpowered fo
r the SESOI (40.40%), the power is only slightly suboptimal for the expect
effect size (72.35%). Considering that this latter estimation has followed
a conservative strategy (called safeguard power analysis), the achieved pow
er can be deemed as acceptable. Taking together, we think that our sample s
ize has sufficient informational value with respect to both statistical hyp
otheses linked to the theory of Halperin and Schulz. Ultimately, examine th
is account in a community sample can extend this ADHD theory to subclinical
and general population.
```

SUPPLEMENTAL TEXT 2: Attention-deficit/hyperactivity disorder (ADHD) Symptom Distribution in our Study Compared to Normative Thresholds and Distributions

Procedure

We calculated the total scores (i.e., sum of the items) of ADHD symptoms for each of the three self-reports measures: the Barkley Adult ADHD Rating Scale-IV: Childhood symptoms (cBAARS-IV), the Barkley Adult ADHD Rating Scale-IV: Current symptoms (aBAARS-IV), and the Adult ADHD Self-Report Screening Scale for DSM The percentage of individuals scoring above the preliminary clinical cutoff was analyzed for each measure. For the cBAARS-IV and the aBAARS-IV, this cutoff was set to the 95th percentile, (Barkley, 2011) while for the ASRS-5, the cutoff was set to a direct score of 14 points (Ustun et al., 2017).

In addition, for the cBAARS and the aBAARS, we compared the distribution of the scores in our sample with an estimation of a Barkley's (2011) normative distribution obtained from a large representative sample of adults from the United States. Barkley reported the values corresponding to 22 and 20 percentiles for the cBAARS and the aBAARS, respectively. Based on the values of these percentiles, we generated a sample of 10,000 simulated total scores for the cBAARS and the aBAARS using a Monte Carlo simulation approach. Percentiles of the simulated sample fitted Barkley's percentiles for each ADHD symptom scale, and they may be compared with our study percentiles in Supplemental Table 3. Finally, we extracted 292 theoretical quantiles from the simulated normative sample, which resulted in a sample of equal size to our study sample. This equally sized sample was used for comparison purposes with the sample from our study.

Results

Binomial tests indicated that while the percentage of participants above the 95th percentile cutoff in the cBAARS-IV (7.5%) was not significantly different from the

normative 5% (p = .058), such percentage in the aBAARS-IV (11.0%) was higher than that preliminary clinical cutoff (p < .001). Regarding the ASRS-5, the percentage of participants above the cutoff of 14 points (11.6%) was not statistically different from the normative 11.2% (p = 781).[20] Two-sample Kolmogorov-Smirnov tests indicate that ADHD symptom distributions in the cBAARS-IV and the aBAARS-IV were significatively different from their corresponding normative sample (both p < .001). Indeed, a visual comparison in Figure 3 shows that the normative distributions are clearly more left-skewed than their respective sample distributions. Compared to the 50th percentile in the normative sample for the cBAARS-IV (*Mdn* = 23) and the aBAARS-IV (*Mdn* = 22), Wilcoxon Signed-Ranks Tests indicated that the medians of the cBAARS-IV (*Mdn* = 29) and the aBAARS-IV (*Mdn* = 30) in our sample were significatively higher (both p < .001). Levene's tests for homogeneity of variances showed no significant differences between the variability of the scores of the cBAARS-IV and the aBAARS-IV between our sample and the normative one (both ps > .430).

[20] Note that the criterion of 11.2% comes from a sample of adults with 8.2% of ADHD cases (Ustun et al., 2017), which is a higher percentage than in the general population.

SUPPLEMENTAL TABLE 1: List of Deviations From the Preregistration

Deviation no.	Description
1	We preregistered that for participants in whom a period of more than 90 days elapsed from the initial survey until the completion of the task, we would use the ADHD symptom self-reports completed during the second phase of the study. However, at that time, we did not expect that seven of those participants (six valid) had not completed the questionnaires the second time. We decided to retain these participants for the preregistered analyses and add this decision point (i.e., retain vs. exclude) into the multiverse analyses. No changes in the pattern of statistical significances of the preregistered statistical hypotheses were found when these participants were excluded.
2	At the time of preregistration, we were not aware of the importance of controlling the inflation of the Type I error rate in secondary statistical hypotheses (Sc1 and Sc2). Since both refer to the same theoretical hypothesis as their corresponding primary statistical hypotheses (H1 and H2), in the final manuscript we decided to halve the statistical significance threshold for the secondary hypotheses (i.e., $\alpha/2 = .025$). The same correction was applied for the opposite counterparts of the secondary statistical hypotheses (Sc1′ and Sc2′).
3	We preregistered an exploratory analysis (E) that consisted of examining the mediating role of cognitive control in the decrement of vigilance associated with ADHD symptoms. However, since the association between ADHD symptoms and decrement of vigilance was smaller in size than the effects of interest, we decided not to conduct this mediation analysis.
4	As a sensitivity analysis to be included in the multiverse (Ss1), we preregistered an alternative approach to estimate late-developing ADHD symptoms. This consisted of computing the result of subtracting symptoms in childhood from symptoms in adulthood. However logical concerns with the use of change variables at different timepoints (Shahar & Shahar, 2012), along with a potential floor effect at least in the cBAARS-IV, made us realise the inadequacy of this approach. When change scores are computed for statistical hypotheses involving late-developing symptoms (i.e., H2, Sc2, H1′, Sc1′), the pattern of statistical significances is the same as in the preregistered analyses (i.e., all nonsignificant).
5	After the preregistration, we identified two decision points with more than one reasonable analytical option (for a more detailed rationale, see Supplemental Table 4): the correlation coefficient (Kendall vs. Spearman vs. Pearson) and the type of correlation (zero-order vs. partial). Both were included in the multiverse.

Note. ADHD = Attention-deficit/hyperactivity disorder; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood symptoms.

SUPPLEMENTAL TABLE 2: Effect Sizes of Interest and Achieved Power to Detect Them Across Each Opposite Statistical Hypothesis

Hypothesis	Smallest effect of interest			Expected effect (from Coll-Martín et al., 2021)		
	r	τ	Power (1 – β)	*r*	τ	Power (1 – β)
H1′	.13	**.08**	.67	–.09	–.06	0
H2′	–.15	**–.09**	.78	–.17	**–.11**	.88
Sc1′	.15	**.10**	.70	.05	.03	.11
Sc2′	–.18	**–.12**	.85	.09	.06	0

Note. N = 292. H1′ and H2′ are the opposite counterparts of the primary statistical hypotheses, while Sc1′ and Sc2′ are the opposite counterparts of the secondary statistical hypotheses. Kendall's τ values come from the Pearson's *r* values used to conduct the 10,000 simulations for power analysis (see Gilpin, 1993, for the formula to transform the correlation coefficients). Statistical power corresponds to one-side tests for Kendall's coefficients, with α = .05 for opposite primary statistical hypotheses and α = .025 for opposite secondary statistical hypotheses. Kendall's τ values above the minimal statistically detectable effect (τ = |.06| for opposite primary hypotheses; τ = |.08| for opposite secondary hypotheses) are in bold.

SUPPLEMENTAL TABLE 3: Percentiles Values of the cBAARS-IV and the aBAARS-IV in Original and Estimated Normative Samples Compared to the Sample of the Present Study

	cBAARS-IV total scores		aBAARS-IV total scores	
Percentile	**Normative sample**	**Study sample**	**Normative sample**	**Study sample**
99	60 (61)	58	54 (55)	53
98	55	54	49	50
97	52 (53)	52	46	50
96	51	52	44	48
95	49	51	43	48
94	47 (48)	49	42 (41)	47
93	46	49	39	45
92	45	47	38	44
91	44	47	37	43
90	43	46	36	43
89	42	46		
88	41 (40)	45		
87	39	45		
85	38	43	35	40
84	37	42		
83			34	39
82	36	41	33	38
81			32	38
79			31	37
77	35	38	30	37
76	34	38		
75	33	38	29	36
51	24 (23)	29	23 (22)	30
50	23	29	22	30
1	18	18	18	19

Note. Percentiles in the normative sample were extracted from Barkley (2011), which is based on a large representative sample of adults (ages 18–39) from the United States. These percentiles were used to generate a simulated normative sample through a Monte Carlo simulation approach (N = 10,000). The values of the simulated sample are in parentheses when they differ from the original normative sample. cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current symptoms.

SUPPLEMENTAL TABLE 4: Multiverse Analysis of the Correlations Between Arousal Vigilance (AV) and Executive Vigilance (EV) with ADHD Symptoms for Each Reasonable Combination of Analytical Decisions

Universe	Analytical decision					Correlation with ADHD symptoms		
	Long respondents	Poor task performers	Task index computation	Correlation coefficient	Correlation type	cBAARS-IV	ASRS-5	aBAARS-IV
				Primary vigilance outcome: Lapse slope (AV)				
1	**Retained**	**Excluded**	**Absolute**	**Kendall**	**Zero-order**	**.00**	–.00	.03
2	*Retained*	*Excluded*	*Absolute*	*Kendall*	*Partial*	.00	*.01*	.05
3	Retained	Excluded	Absolute	Spearman	Zero-order	.00	.00	.04
4	Retained	Excluded	Absolute	Spearman	Partial	.00	.01	.07
5	Retained	Excluded	Absolute	Pearson	Zero-order	.01	.02	.08
6	Retained	Excluded	Absolute	Pearson	Partial	.00	.02	.09
7	Retained	Excluded	Relative	Kendall	Zero-order	.00	.03	.06
8	Retained	Excluded	Relative	Kendall	Partial	–.02	.04	.08*
9	Retained	Excluded	Relative	Spearman	Zero-order	.00	.04	.08
10	Retained	Excluded	Relative	Spearman	Partial	–.03	.05	.12*
11	Retained	Excluded	Relative	Pearson	Zero-order	–.02	.04	.07
12	Retained	Excluded	Relative	Pearson	Partial	–.04	.05	.09
13	Retained	Retained	Absolute	Kendall	Zero-order	.01	.02	.04
14	Retained	Retained	Absolute	Kendall	Partial	.00	.01	.05
15	Retained	Retained	Absolute	Spearman	Zero-order	.01	.03	.06
16	Retained	Retained	Absolute	Spearman	Partial	.00	.02	.08
17	Retained	Retained	Absolute	Pearson	Zero-order	–.02	.03	.06
18	Retained	Retained	Absolute	Pearson	Partial	–.03	.04	.08
19	Retained	Retained	Relative	Kendall	Zero-order	–.00	.05	.07*
20	Retained	Retained	Relative	Kendall	Partial	–.03	.06	.10**
21	Retained	Retained	Relative	Spearman	Zero-order	–.00	.07	.10*
22	Retained	Retained	Relative	Spearman	Partial	–.05	.08	.15**
23	Retained	Retained	Relative	Pearson	Zero-order	–.05	.06	.06
24	Retained	Retained	Relative	Pearson	Partial	–.07	.08	.10*
25	Excluded	Excluded	Absolute	Kendall	Zero-order	.01	.01	.04
26	Excluded	Excluded	Absolute	Kendall	Partial	.01	.02	.05
27	Excluded	Excluded	Absolute	Spearman	Zero-order	.02	.02	.05
28	Excluded	Excluded	Absolute	Spearman	Partial	.01	.03	.08
29	Excluded	Excluded	Absolute	Pearson	Zero-order	.02	.04	.10

Universe	Analytical decision					Correlation with ADHD symptoms		
	Long respondents	Poor task performers	Task index computation	Correlation coefficient	Correlation type	cBAARS-IV	ASRS-5	aBAARS-IV
				Primary vigilance outcome: Lapse slope (AV)				
30	Excluded	Excluded	Absolute	Pearson	Partial	.00	.03	.10*
31	Excluded	Excluded	Relative	Kendall	Zero-order	.00	.04	.06
32	Excluded	Excluded	Relative	Kendall	Partial	–.02	.05	.09*
33	Excluded	Excluded	Relative	Spearman	Zero-order	.01	.05	.09
34	Excluded	Excluded	Relative	Spearman	Partial	–.03	.07	.13*
35	Excluded	Excluded	Relative	Pearson	Zero-order	–.02	.05	.07
36	Excluded	Excluded	Relative	Pearson	Partial	–.04	.06	.10*
37	Excluded	Retained	Absolute	Kendall	Zero-order	.01	.03	.05
38	Excluded	Retained	Absolute	Kendall	Partial	.00	.03	.06
39	Excluded	Retained	Absolute	Spearman	Zero-order	.02	.05	.08
40	Excluded	Retained	Absolute	Spearman	Partial	.01	.04	.09
41	Excluded	Retained	Absolute	Pearson	Zero-order	–.01	.07	.08
42	Excluded	Retained	Absolute	Pearson	Partial	–.03	.07	.10*
43	Excluded	Retained	Relative	Kendall	Zero-order	.00	.06	.08*
44	Excluded	Retained	Relative	Kendall	Partial	–.03	.07*	.11**
45	Excluded	Retained	Relative	Spearman	Zero-order	.00	.09	.11*
46	Excluded	Retained	Relative	Spearman	Partial	–.05	.10*	.16**
47	Excluded	Retained	Relative	Pearson	Zero-order	–.04	.09	.08
48	Excluded	Retained	Relative	Pearson	Partial	–.08	.11*	.12*
				Primary vigilance outcome: Hit slope (EV)				
1	*Retained*	*Excluded*	*Absolute*	*Kendall*	*Zero-order*	*.04*	–.01	.01
2	**Retained**	**Excluded**	**Absolute**	**Kendall**	**Partial**	.03	**–.03**	–.01
3	Retained	Excluded	Absolute	Spearman	Zero-order	.05	–.02	.02
4	Retained	Excluded	Absolute	Spearman	Partial	.05	–.05	–.01
5	Retained	Excluded	Absolute	Pearson	Zero-order	.03	–.03	.00
6	Retained	Excluded	Absolute	Pearson	Partial	.04	–.04	–.01
7	Retained	Excluded	Relative	Kendall	Zero-order	.05	–.01	.02
8	Retained	Excluded	Relative	Kendall	Partial	.04	–.04	–.02
9	Retained	Excluded	Relative	Spearman	Zero-order	.07	–.02	.03
10	Retained	Excluded	Relative	Spearman	Partial	.06	–.06	–.02
11	Retained	Excluded	Relative	Pearson	Zero-order	.04	–.03	.00
12	Retained	Excluded	Relative	Pearson	Partial	.05	–.04	–.02
13	Retained	Retained	Absolute	Kendall	Zero-order	.04	–.01	.00
14	Retained	Retained	Absolute	Kendall	Partial	.04	–.04	–.02
15	Retained	Retained	Absolute	Spearman	Zero-order	.05	–.02	.01

Universe	Analytical decision					Correlation with ADHD symptoms		
	Long respondents	Poor task performers	Task index computation	Correlation coefficient	Correlation type	cBAARS-IV	ASRS-5	aBAARS-IV
				Primary vigilance outcome: Hit slope (EV)				
16	Retained	Retained	Absolute	Spearman	Partial	.05	–.05	–.02
17	Retained	Retained	Absolute	Pearson	Zero-order	.04	–.03	–.01
18	Retained	Retained	Absolute	Pearson	Partial	.06	–.05	–.03
19	Retained	Retained	Relative	Kendall	Zero-order	.04	–.01	.01
20	Retained	Retained	Relative	Kendall	Partial	.05	–.04	–.03
21	Retained	Retained	Relative	Spearman	Zero-order	.06	–.02	.01
22	Retained	Retained	Relative	Spearman	Partial	.07	–.06	–.04
23	Retained	Retained	Relative	Pearson	Zero-order	.05	–.03	–.00
24	Retained	Retained	Relative	Pearson	Partial	.06	–.05	–.03
25	Excluded	Excluded	Absolute	Kendall	Zero-order	.03	–.02	.01
26	Excluded	Excluded	Absolute	Kendall	Partial	.03	–.03	–.01
27	Excluded	Excluded	Absolute	Spearman	Zero-order	.05	–.02	.01
28	Excluded	Excluded	Absolute	Spearman	Partial	.04	–.06	–.01
29	Excluded	Excluded	Absolute	Pearson	Zero-order	.02	–.04	–.01
30	Excluded	Excluded	Absolute	Pearson	Partial	.03	–.05	–.02
31	Excluded	Excluded	Relative	Kendall	Zero-order	.04	–.02	.01
32	Excluded	Excluded	Relative	Kendall	Partial	.04	–.04	–.02
33	Excluded	Excluded	Relative	Spearman	Zero-order	.06	–.03	.02
34	Excluded	Excluded	Relative	Spearman	Partial	.06	–.06	–.03
35	Excluded	Excluded	Relative	Pearson	Zero-order	.03	–.04	–.00
36	Excluded	Excluded	Relative	Pearson	Partial	.05	–.05	–.02
37	Excluded	Retained	Absolute	Kendall	Zero-order	.03	–.02	–.00
38	Excluded	Retained	Absolute	Kendall	Partial	.03	–.04	–.03
39	Excluded	Retained	Absolute	Spearman	Zero-order	.05	–.03	–.00
40	Excluded	Retained	Absolute	Spearman	Partial	.05	–.07	–.03
41	Excluded	Retained	Absolute	Pearson	Zero-order	.03	–.05	–.02
42	Excluded	Retained	Absolute	Pearson	Partial	.05	–.07	–.04
43	Excluded	Retained	Relative	Kendall	Zero-order	.04	–.02	.00
44	Excluded	Retained	Relative	Kendall	Partial	.04	–.05	–.04
45	Excluded	Retained	Relative	Spearman	Zero-order	.06	–.04	.00
46	Excluded	Retained	Relative	Spearman	Partial	.06	–.07	–.05
47	Excluded	Retained	Relative	Pearson	Zero-order	.04	–.05	–.01
48	Excluded	Retained	Relative	Pearson	Partial	.06	–.07	–.04
				Secondary vigilance outcome: Lapse overall (AV)				
1	**Retained**	**Excluded**	**Absolute**	**Kendall**	**Zero-order**	**.07**	.00	.08*

Universe	Analytical decision					Correlation with ADHD symptoms		
	Long respondents	Poor task performers	Task index computation	Correlation coefficient	Correlation type	cBAARS-IV	ASRS-5	aBAARS-IV
				Secondary vigilance outcome: Lapse overall (AV)				
2	*Retained*	*Excluded*	*Absolute*	*Kendall*	*Partial*	.06	*–.00*	.06
3	Retained	Excluded	Absolute	Spearman	Zero-order	.10	.01	.12*
4	Retained	Excluded	Absolute	Spearman	Partial	.08	.00	.10
5	Retained	Excluded	Absolute	Pearson	Zero-order	.18**	.17**	.21***
6	Retained	Excluded	Absolute	Pearson	Partial	.13*	.12*	.14*
7	Retained	Excluded	Relative	Kendall	Zero-order	–.02	.00	.01
8	Retained	Excluded	Relative	Kendall	Partial	–.03	.02	.03
9	Retained	Excluded	Relative	Spearman	Zero-order	–.04	.01	.01
10	Retained	Excluded	Relative	Spearman	Partial	–.04	.03	.04
11	Retained	Excluded	Relative	Pearson	Zero-order	–.03	.03	–.01
12	Retained	Excluded	Relative	Pearson	Partial	–.05	.04	.01
13	Retained	Retained	Absolute	Kendall	Zero-order	.06	.01	.08*
14	Retained	Retained	Absolute	Kendall	Partial	.04	.01	.07
15	Retained	Retained	Absolute	Spearman	Zero-order	.09	.03	.12*
16	Retained	Retained	Absolute	Spearman	Partial	.06	.02	.11
17	Retained	Retained	Absolute	Pearson	Zero-order	.11	.13*	.16**
18	Retained	Retained	Absolute	Pearson	Partial	.07	.10	.12*
19	Retained	Retained	Relative	Kendall	Zero-order	–.03	.01	.01
20	Retained	Retained	Relative	Kendall	Partial	–.04	.02	.03
21	Retained	Retained	Relative	Spearman	Zero-order	–.05	.01	.01
22	Retained	Retained	Relative	Spearman	Partial	–.06	.03	.05
23	Retained	Retained	Relative	Pearson	Zero-order	–.08	–.04	–.05
24	Retained	Retained	Relative	Pearson	Partial	–.07	–.01	–.01
25	Excluded	Excluded	Absolute	Kendall	Zero-order	.06	–.00	.07
26	Excluded	Excluded	Absolute	Kendall	Partial	.05	–.01	.06
27	Excluded	Excluded	Absolute	Spearman	Zero-order	.09	.00	.11
28	Excluded	Excluded	Absolute	Spearman	Partial	.07	–.01	.09
29	Excluded	Excluded	Absolute	Pearson	Zero-order	.16**	.14*	.19**
30	Excluded	Excluded	Absolute	Pearson	Partial	.12*	.10	.13*
31	Excluded	Excluded	Relative	Kendall	Zero-order	–.03	–.00	.00
32	Excluded	Excluded	Relative	Kendall	Partial	–.04	.01	.02
33	Excluded	Excluded	Relative	Spearman	Zero-order	–.05	.00	.00
34	Excluded	Excluded	Relative	Spearman	Partial	–.05	.02	.03
35	Excluded	Excluded	Relative	Pearson	Zero-order	–.05	.02	–.02
36	Excluded	Excluded	Relative	Pearson	Partial	–.06	.03	.00

Universe	Analytical decision					Correlation with ADHD symptoms		
	Long respondents	Poor task performers	Task index computation	Correlation coefficient	Correlation type	cBAARS-IV	ASRS-5	aBAARS-IV
				Secondary vigilance outcome: Lapse overall (AV)				
37	Excluded	Retained	Absolute	Kendall	Zero-order	.05	.02	.08*
38	Excluded	Retained	Absolute	Kendall	Partial	.03	.00	.07
39	Excluded	Retained	Absolute	Spearman	Zero-order	.08	.03	.12*
40	Excluded	Retained	Absolute	Spearman	Partial	.04	.01	.10
41	Excluded	Retained	Absolute	Pearson	Zero-order	.10	.12*	.15*
42	Excluded	Retained	Absolute	Pearson	Partial	.06	.09	.11
43	Excluded	Retained	Relative	Kendall	Zero-order	–.04	.01	.01
44	Excluded	Retained	Relative	Kendall	Partial	–.05	.02	.03
45	Excluded	Retained	Relative	Spearman	Zero-order	–.06	.01	.01
46	Excluded	Retained	Relative	Spearman	Partial	–.07	.04	.05
47	Excluded	Retained	Relative	Pearson	Zero-order	–.08	–.03	–.05
48	Excluded	Retained	Relative	Pearson	Partial	–.08	.00	–.01
				Secondary vigilance outcome: Hit overall (EV)				
1	*Retained*	*Excluded*	*Absolute*	*Kendall*	*Zero-order*	*–.09**	–.07	–.11**
2	**Retained**	**Excluded**	**Absolute**	**Kendall**	**Partial**	–.08*	**–.04**	–.08*
3	Retained	Excluded	Absolute	Spearman	Zero-order	–.13*	–.10	–.15**
4	Retained	Excluded	Absolute	Spearman	Partial	–.12*	–.07	–.12*
5	Retained	Excluded	Absolute	Pearson	Zero-order	–.14*	–.15*	–.18**
6	Retained	Excluded	Absolute	Pearson	Partial	–.10	–.11	–.12*
7	Retained	Excluded	Relative	Kendall	Zero-order	–.09*	–.07	–.11**
8	Retained	Excluded	Relative	Kendall	Partial	–.08*	–.05	–.08*
9	Retained	Excluded	Relative	Spearman	Zero-order	–.13*	–.11	–.16**
10	Retained	Excluded	Relative	Spearman	Partial	–.12*	–.07	–.12*
11	Retained	Excluded	Relative	Pearson	Zero-order	–.14*	–.15**	–.18**
12	Retained	Excluded	Relative	Pearson	Partial	–.10	–.11	–.12*
13	Retained	Retained	Absolute	Kendall	Zero-order	–.07	–.07	–.11**
14	Retained	Retained	Absolute	Kendall	Partial	–.06	–.05	–.09*
15	Retained	Retained	Absolute	Spearman	Zero-order	–.11	–.11	–.16**
16	Retained	Retained	Absolute	Spearman	Partial	–.09	–.07	–.14*
17	Retained	Retained	Absolute	Pearson	Zero-order	–.10	–.13*	–.15**
18	Retained	Retained	Absolute	Pearson	Partial	–.06	–.10	–.12*
19	Retained	Retained	Relative	Kendall	Zero-order	–.08*	–.08	–.11**
20	Retained	Retained	Relative	Kendall	Partial	–.07	–.05	–.09*
21	Retained	Retained	Relative	Spearman	Zero-order	–.11	–.11	–.16**
22	Retained	Retained	Relative	Spearman	Partial	–.10	–.07	–.14*

Universe	Analytical decision					Correlation with ADHD symptoms		
	Long respondents	Poor task performers	Task index computation	Correlation coefficient	Correlation type	cBAARS-IV	ASRS-5	aBAARS-IV
	Secondary vigilance outcome: Hit overall (EV)							
23	Retained	Retained	Relative	Pearson	Zero-order	–.11	–.13*	–.16**
24	Retained	Retained	Relative	Pearson	Partial	–.06	–.10	–.12*
25	Excluded	Excluded	Absolute	Kendall	Zero-order	–.08*	–.07	–.11**
26	Excluded	Excluded	Absolute	Kendall	Partial	–.07	–.04	–.08*
27	Excluded	Excluded	Absolute	Spearman	Zero-order	–.12*	–.10	–.16**
28	Excluded	Excluded	Absolute	Spearman	Partial	–.11	–.06	–.12*
29	Excluded	Excluded	Absolute	Pearson	Zero-order	–.12*	–.12*	–.16**
30	Excluded	Excluded	Absolute	Pearson	Partial	–.09	–.08	–.11
31	Excluded	Excluded	Relative	Kendall	Zero-order	–.08*	–.07	–.11**
32	Excluded	Excluded	Relative	Kendall	Partial	–.08	–.04	–.09*
33	Excluded	Excluded	Relative	Spearman	Zero-order	–.12*	–.10	–.16**
34	Excluded	Excluded	Relative	Spearman	Partial	–.11	–.06	–.13*
35	Excluded	Excluded	Relative	Pearson	Zero-order	–.13*	–.12*	–.17**
36	Excluded	Excluded	Relative	Pearson	Partial	–.09	–.09	–.12*
37	Excluded	Retained	Absolute	Kendall	Zero-order	–.07	–.07	–.11**
38	Excluded	Retained	Absolute	Kendall	Partial	–.06	–.05	–.10*
39	Excluded	Retained	Absolute	Spearman	Zero-order	–.10	–.11	–.17**
40	Excluded	Retained	Absolute	Spearman	Partial	–.08	–.08	–.14*
41	Excluded	Retained	Absolute	Pearson	Zero-order	–.09	–.11	–.14*
42	Excluded	Retained	Absolute	Pearson	Partial	–.05	–.09	–.11
43	Excluded	Retained	Relative	Kendall	Zero-order	–.07	–.08*	–.12**
44	Excluded	Retained	Relative	Kendall	Partial	–.06	–.05	–.10*
45	Excluded	Retained	Relative	Spearman	Zero-order	–.10	–.11	–.17**
46	Excluded	Retained	Relative	Spearman	Partial	–.08	–.08	–.15*
47	Excluded	Retained	Relative	Pearson	Zero-order	–.09	–.12*	–.15*
48	Excluded	Retained	Relative	Pearson	Partial	–.06	–.09	–.12*

Note. N = 292. Long respondents (n = 7; n = 6 after excluding poor task performers) are participants for whom more than 90 days elapsed between the completion of the ADHD self-reports of the task and the task itself. These participants did not complete the second administration of ADHD questionnaires (but note that the measures of ADHD symptoms in adulthood considers the last 6 months (i.e., twice our cut-off point). Poor task performers (n = 14; n = 13 after excluding long respondents) are participants with more than 25% errors in Attention Network Test for Interaction (ANTI) trials of the cognitive task. Note that the options of long respondents and poor performers slightly affect the sample size and therefore the statistical power. We control for this statistical confounder

by directly inputting a sample size of 292 to the formula to obtain the *p* value from the correlation coefficient. The task index computation can be based on an absolute (i.e., reaction time [RT] higher than 600 ms or no response for lapses; correct detection of the displacement for hits) or relative (i.e., RT higher than the participant's mean plus one standard deviation or no response for lapses outcomes; correct detection of the displacement within 2.5 standard deviations plus/minus each participant's mean RT) threshold. Both poor task performers and task index computation were preregistered with the label (Ss3). The correlation coefficients include the nonparametric Kendall's rank-order correlation coefficient and Spearman rank-order correlation, but also the parametric Pearson product–moment correlation coefficient. Although the correlation of the pre-registered hypotheses violated the normalization assumption, McDonald (2014) recommends using the Pearson correlation even in this type of scenario. In partial correlations, the associations involving the cBAARS-IV are controlled for the ASRS-5, while correlations involving the ASRS-5 or the aBAARS-IV are accounted for by the cBAARS-IV. Note that controlling for symptoms in childhood by a later variable (i.e., symptoms in adulthood) may be considered problematic by some due to potential collision bias (Rohrer, 2018). The rows with the set of preregistered analytical decisions for each statistical hypothesis derived from Halperin and Schulz's (2006) neurodevelopmental model, along with their corresponding correlation, are in bold. The rows with the set of preregistered analytical decisions for each opposite statistical hypothesis (i.e., hypotheses relating each vigilance component to ADHD symptom severity in the age period opposite to that established in the neurodevelopmental model), along with their corresponding correlation, are italicized. ADHD = Attention-deficit/hyperactivity disorder; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current symptoms; ASRS-5 = Adult ADHD Self-Report Screening Scale for DSM-5.

$^{*}p < .05$, one-tailed. $^{**}p < .01$, one-tailed. $^{*}p < .001$, one-tailed. All contrasts are corrected for multiple comparisons in hypotheses involving secondary vigilance outcomes.

SUPPLEMENTAL TABLE 5: Means, Standard Deviations, and Pearson Correlations Among Arousal and Executive ANTI-Vea Indices

Index	*M*	*SD*	1	2	3	4	5	6
Arousal								
1. Lapse slope (AV)	1.09	2.60	—					
2. Lapse overall (AV)	10.57	17.69	.22***	—				
3. RT *M* slope (AV)	2.07	10.12	.77***	.21***	—			
4. RT *M* overall (AV)	491.75	95.44	.18**	.88***	.15**	—		
5. RT *SD* slope (AV)	4.33	9.41	.54***	.26***	.49***	.23***	—	
6. RT *SD* overall (AV)	78.73	32.79	.37***	.63***	.35***	.51***	.44***	—
7. Alerting RT	37.20	31.60	.07	–.03	.06	.03	.02	–.02
8. Alerting errors	2.46	4.60	.18**	.08	.13*	.10	.14*	.15*
Executive								
9. Hits slope (EV)	–2.17	3.78	–.10	–.01	–.08	.02	–.12*	–.11
10. Hits overall (EV)	73.08	18.28	–.07	–.11	–.06	–.11	–.07	–.18**
11. FA slope (EV) [a]	–0.51	1.71	–.01	.04	–.08	.05	.01	.08
12. FA overall (EV) [a]	5.41	5.19	.02	.09	.07	.10	.06	.07
13. Congruency RT	42.54	27.71	.07	.09	.02	.09	.12*	.16**
14. Congruency errors	0.92	4.18	.13*	–.08	.03	–.09	.07	.07

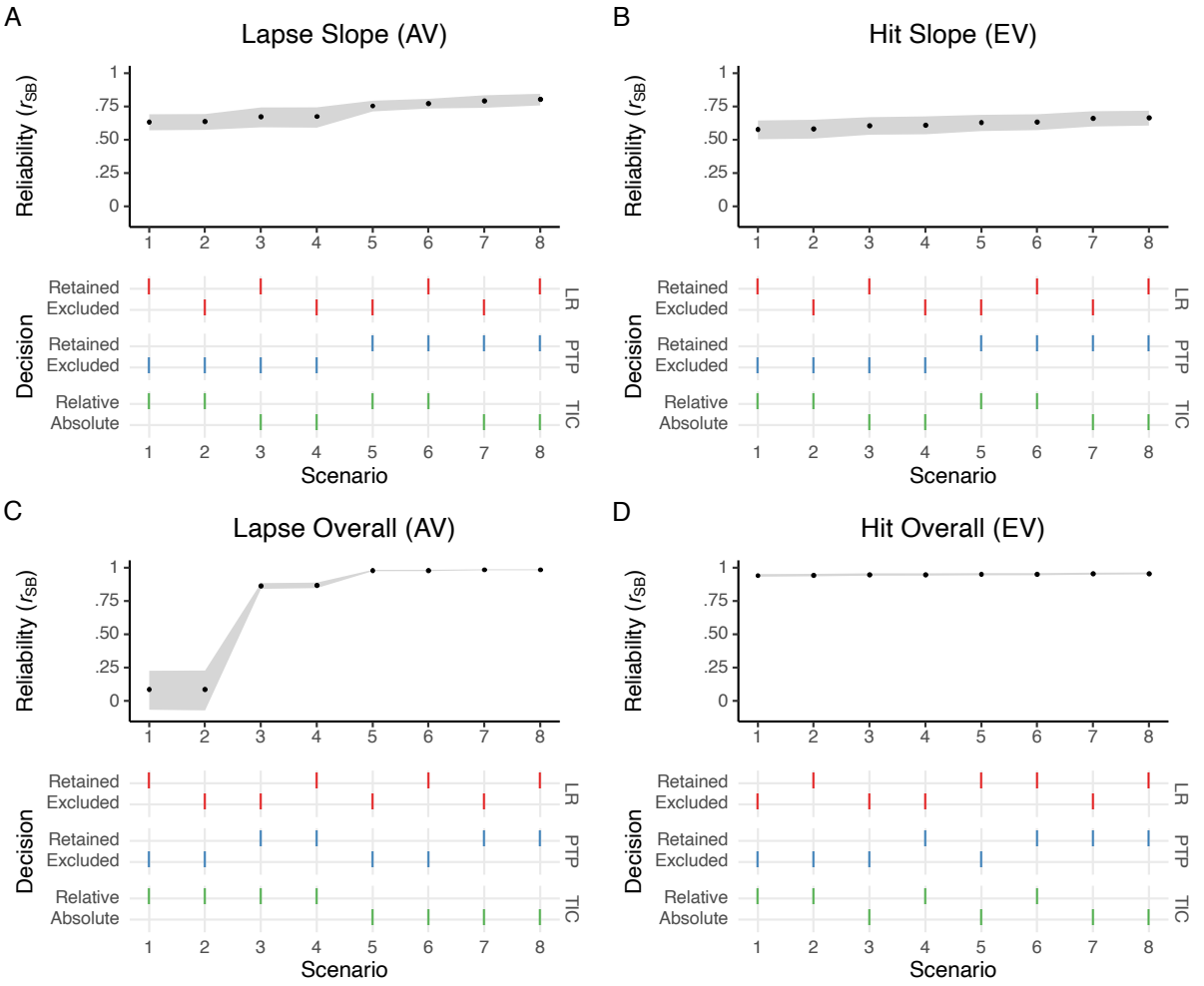
Index	7	8	9	10	11	12	13	14
Arousal								
1. Lapse slope (AV)								
2. Lapse overall (AV)								
3. RT *M* slope (AV)								
4. RT *M* overall (AV)								
5. RT *SD* slope (AV)								
6. RT *SD* overall (AV)								
7. Alerting RT	—							
8. Alerting errors	.05	—						
Executive								
9. Hits slope (EV)	.03	.02	—					
10. Hits overall (EV)	.12*	.03	.41***	—				
11. FA slope (EV) [a]	.01	–.02	.07	-.04	—			
12. FA overall (EV) [a]	.22***	–.01	.21***	.27***	–.21***	—		
13. Congruency RT	–.10	.07	–.13*	–.31***	.10	–.22***	—	
14. Congruency errors	–.10	.05	–.11	–.45***	.14*	–.32***	.35***	—

Note. N = 292. These indices come from the Analysis section of the website at https://anti-vea.ugr.es/analysis.html (for a more detailed description of the analyses, see Luna, Barttfeld, et al., 2020). ANTI-Vea = Attention Network Test for Interactions and Vigilance—executive and arousal components; RT = Reaction time; AV =Arousal vigilance; EV = Executive vigilance; FA = False alarm.

[a] *n* = 289.

*$p < .05$. **$p < .01$. ***$p < .001$.

SUPPLEMENTAL FIGURE 1: Internal Consistency Reliability Scores for Indices of Arousal Vigilance (AV) and Executive Vigilance (EV) Across the Multiverse of Reasonable Analytical Options



Note. *N* = 292 (LR retained and PTP excluded; as preregistered); *N* = 306 (LR retained and PTP retained); *N* = 299 (LR excluded and PTP retained); *N* = 286 (LR excluded and PTP excluded). Internal consistency scores were estimated using a permutation-based split-half approach (Parsons et al., 2019) with 10,000 random splits, and then applying the Spearman–Brown prophecy formula. The black dots represent the median scores of each set of iterations, while the grey area represents their corresponding 95% confidence interval (based on 2.5th and 97.5th percentiles of each set of iterations). r_{SB} = Spearman-Brown split-half reliability coefficient; LR = Long respondents; PTP = Poor task performers; TIC = Task index computation.

Study 4

Are Attentional Processes Differentially Related to Symptoms of ADHD in Childhood, Adulthood, and Late Onset? Evidence From a Multi-Sample of Community Adults

This work is in preparation as:

Coll-Martín, T., Sonuga-Barke, E. J. S., Carretero-Dios, H., & Lupiáñez, J. Are attentional processes differentially related to symptoms of ADHD in childhood, adulthood, and late onset? Evidence from a multi-sample of community adults [Manuscript in preparation]. Department of Behavioural Sciences Methodology, University of Granada.

Abstract

The recent discovery of late-onset attention-deficit/hyperactivity disorder (ADHD) has challenged the neurodevelopmental notion of the disorder. Understanding the differences in neurocognitive processes underlying ADHD between distinct age ranges and at different onset times is crucial for guiding its approach. Based on a dimensional conceptualization of ADHD, the present study aims to analyze the association between attentional processes and ADHD symptoms in childhood and adulthood among community adults. A total of 462 university students from three samples were analyzed. Participants completed self-reports of ADHD symptoms in childhood (retrospectively) and adulthood and performed the ANTI-Vea: a novel task that simultaneously assesses the functioning of attentional networks and vigilance, distinguishing between arousal and executive components. Using mixed-effect models, each task index was computed and included in a regression model to be predicted from the severity of symptoms in childhood and adulthood. Alterations in arousal and executive measures were similarly predicted by both childhood and adult symptoms. Albeit with some change in task indices, predictions made by late-onset symptoms (i.e., adult symptoms after controlling for baseline in childhood) were close to those of childhood and adult symptoms. These observed similarities do not support the idea of different neurocognitive impairments in adult or late-onset ADHD, as compared to child-onset disorder. Translational interventions for ADHD symptoms should target the same underlying attentional deficits across different stages of development, regardless of the age of disorder onset. Future studies could include complementary assessment methods and clinical groups.

Materials, data, and analyses that support the findings of this study are openly available at https://osf.io/53ubs/

Introduction

Conventionally, attention-deficit/hyperactivity disorder (ADHD) has been conceptualized as a child-onset neurodevelopmental disorder (Sonuga-Barke et al., 2023). Indeed, the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association, 2013) sets as a diagnostic criterion that several ADHD symptoms must have been present prior to age 12. However, this neurodevelopmental notion of the disorder has been recently challenged by longitudinal studies from different countries suggesting that a substantial proportion of adults with ADHD did not meet the diagnostic—nor "several" symptoms—in childhood (Agnew-Blais et al., 2016; Caye et al., 2016; Moffitt et al., 2015). This so-called late-onset ADHD, estimated to constitute around 1%–2% of the population and half of the all adults with ADHD, presents life impairment that demands clinical attention (Asherson & Agnew-Blais, 2019).

While late-onset ADHD is increasingly recognized, the controversy centers around the extent to which these symptomatic manifestations reflect the same etiopathophysiology as the conventional form of disorder (Asherson & Agnew-Blais, 2019; Polanczyk et al., 2019). Focused on the underlying mechanisms, are the neurocognitive alterations linked to ADHD symptoms different between child- and late-onset ADHD? Moffit et al. (2015) found that the performance in adults with child-onset ADHD was impaired across measures of working memory, processing speed, flexibility, and sustained attention; whereas individuals with late-onset ADHD only exhibited deficits in a specific index of this last measure. However, other studies with similar designs found no differences between child- and adult-onset ADHD in working memory, inhibition, reaction time (RT) variability, sustained attention, or timing (Cooper et al., 2018; Ilbegi et al., 2021; Riglin et al., 2022).

In the present study, we aimed at addressing the above question from a different design and approach. Based on a dimensional framework of ADHD (Coghill & Sonuga-Barke, 2012; Hilger et al., 2020), we collapsed three independent samples of community adults that completed self-reports of ADHD symptoms in childhood (retrospectively) and adulthood and performed a comprehensive attentional task: the *Attentional Networks Test for Interaction and Vigilance—Executive and Arousal Components* (ANTI-Vea; Coll-Martín, Román-Caballero, et al., 2023). The ANTI-Vea assesses the functioning of three independent attentional networks (alerting, orienting, and executive control; M. I. Posner & Petersen, 1990), along with the dissociable executive and arousal components of vigilance (Luna et al., 2018). This design allowed for analysis of the association of different attentional processes with the severity of ADHD symptoms in both childhood and adulthood. Furthermore, by controlling for the association between adult and childhood symptoms, it became possible to estimate the relationship of attentional processes with late-onset ADHD symptoms.

Although at the expense of other constraints, our design offers the following advantages over previous studies. First, analyzing all participants across different community samples addresses the lack of statistical power that most previous longitudinal designs faced due to the small size of the late-onset ADHD group ($n < 35$; Cooper et al., 2018; Ilbegi et al., 2021; Moffitt et al., 2015). Second, while most previous studies included only measures of executive functions or tasks with a certain executive demand—with Ilbegi et al. (2021) being an exception—our attentional task assesses both executive and arousal processes. This distinction, particularly relevant in ADHD models (Barkley, 1997; Halperin & Schulz, 2006; Sergeant, 2005), has been validated in experimental and individual difference studies using the ANTI-Vea (Cásedas et al., 2022; Hemmerich et al., 2023). Finally, our study employed self-reports for both childhood and adulthood measures of ADHD symptoms, thus avoiding the methodological bias related with rater

change that is present in some longitudinal studies of this literature (Asherson & Agnew-Blais, 2019).

The comparison of the pattern of associations between attentional processes and ADHD symptoms in (a) childhood, (b) adulthood, and (c) late onset serves to examine two competing accounts. For the *quantitative difference models*, late-onset ADHD symptoms reflect the same neurocognitive alterations as childhood symptoms and only differ in the time period in which they are manifested (e.g., Faraone & Biederman, 2016). From this perspective, the pattern of attentional associations across the three symptom expressions should not show substantial variation. Conversely, the *qualitative difference models* consider that the underlying mechanisms of late-onset symptoms are substantially distinct from those in childhood. For example, applying the Halperin and Schulz's (2006) neurodevelopmental model, ADHD symptoms in childhood would be related to arousal alterations, while late-onset symptoms would associate with executive impairments. Alternatively, it could be the case that late-onset ADHD represents a nondevelopmental disorder with no neurocognitive deficits (Moffitt et al., 2015).

Methods

Description of the Samples and Study Procedure

Initially we recruited data from 493 Spanish university students coming from three independent samples (see **Table 1** for details). All participants followed a similar procedure. Firstly, they were invited to take part in the study through the communication channels of the university in exchange for course credits or financial reward. The three studies had the ethics committee approval, and participants of each one completed an informed consent form. In each study, participants completed a survey that included measures of ADHD symptoms in childhood (retrospectively) and adulthood. Finally,

participants underwent a comprehensive assessment of their attentional functioning through a computerized cognitive task: the ANTI-Vea. Both the survey and attentional task phases were carried out individually by each participant (i.e., no group sessions), with appropriate instructions provided to facilitate their correct understanding.

To contextualize, Sample 1 (*Original*; Coll-Martín et al., 2021) belonged to the seminal study in which the relationship between ADHD symptoms and ANTI-VEA indices was analyzed for the first time. Sample 2 (*Replication*; Coll-Martín, Carretero-Dios, & Lupiáñez, 2023), consisted of a preregistered close replication of the previous one. Sample 3 (*Multiple*; Lupiáñez et al., 2022) was part of a sleep deprivation study in which participants performed the ANTI-Vea 11 times. For the present study we included only the seven baseline sessions of the task (i.e., those in which participants were not sleep deprived).

Table 1

Characteristics of the Three Samples Included in the Present Study

Sample and study	*N* valid [a] (*N* recruited)	Type of participants	% Men	*M* Age (*SD*)	Study setting	Task sessions
1: *Original* (Coll-Martín et al., 2021)	113 (120)	Only psychology undergraduates	18.6	20.2 (1.92)	Lab	1
2: *Replication* (Coll-Martín, Carretero-Dios, & Lupiáñez, 2023)	292 (318)	University students	51.0	21.7 (2.71)	Online	1
3: *Multiple* (Lupiáñez et al., 2022)	57 (58)	University students	19.3	22.6 (3.39)	Online	7
Total	462 (493)		39.2	21.5 (2.74)		

Note. Six participants (1.30%, none of them in Sample 3), reported a prior diagnosis of attention-deficit/hyperactivity disorder.

[a] *N* valid refers to the number of participants after removing those with incomplete or poor task performance.

Instruments

Barkley Adult ADHD Rating Scale-IV: Childhood and Current Symptoms

The self-report of the Barkley Adult ADHD Rating Scale-IV (BAARS-IV; Barkley, 2011) includes two scales to assess ADHD symptoms: retrospectively in childhood (cBAARS-IV) and concurrently in adulthood (aBAARS-IV). Each scale is composed of 18 items, nine of inattention and nine of hyperactivity–impulsivity, on a Likert scale ranging from 1 (*never or rarely*) to 4 (*very often*). Since these items are based on the DSM-IV (APA, 1994), the Spanish version of the manual was used for translation purposes (APA, 1994/1995). Across the valid participants of each sample, Cronbach's alpha reliability scores ranged from .89 to .90 for the cBAARS-IV and from .80 to .86 for the aBAARS-IV.

ANTI-Vea

The ANTI-Vea (Coll-Martín, Román-Caballero, et al., 2023) is a cognitive task that measures the functioning of the three attentional networks (alerting, orienting, and executive control), along with the executive and arousal components of vigilance. To do so, a pseudorandomized, interleaved sequence of 80 trials per block is presented on the computer screen for participants to provide the appropriate response. In our samples, six seamless blocks of the ANTI-Vea were presented per session, resulting in 480 trials over around 33 min. **Figure 1** illustrates the general characteristics of the three types of ANTI-Vea trials, namely (a) attentional networks and interactions (or simply attentional network trials), derived from the Attention Network Test (Fan et al., 2002); (b) executive vigilance, a signal-detection task or continuous performance test paradigm; and (c) arousal vigilance, a simple RT task similar to the Psychomotor Vigilance Test (Lim & Dinges, 2008).

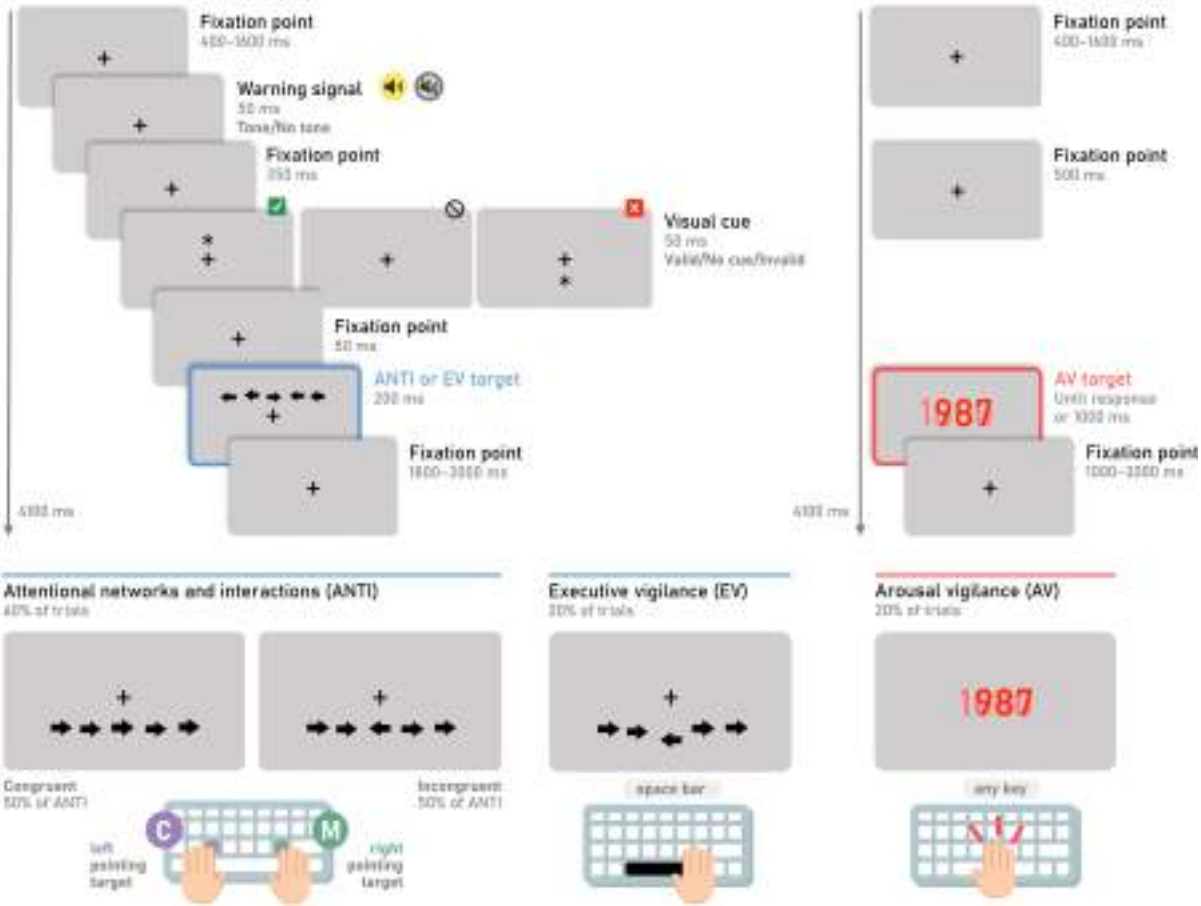


Figure 1. Attention Network Test for Interaction and Vigilance—Executive and Arousal Components (ANTI-Vea) Procedure. Upper left: Attentional network and executive vigilance trials share the same stimuli sequence and response target, namely the central arrow. For attentional network trials, the alerting network is assessed with an auditory warning signal presented in half of trials, the orienting network via a visual cue that may appear at the same (valid) or opposite (invalid) location as the target with equal probability, and the executive control network via the congruency between the target and the flankers. Bottom left: Attentional network and executive vigilance trials differ in their correct answer to the target. In attentional network trials, which constitute the bulk of the task, participants must respond to the direction of the target, while in executive vigilance trials participant must detect its infrequent vertical displacement by pressing the space bar. Right: Procedure for arousal vigilance trials, in which a red millisecond down counter appears for participants to stop it by pressing any key as quickly as possible. For a more detailed description of the task with audiovisual material, see Method section of the ANTI-Vea website at https://anti-vea.ugr.es/method.html

The ANTI-Vea core indices encompass eight scores of attentional networks and 10 of vigilance. Attentional networks indices include the overall performance on mean RT and error percentage across attentional network trials, as well as the modulation of this performance by each of the three networks: alerting (i.e., trials with no acoustic tone minus

trials with acoustic tone, both within trials with no visual cue), orienting (i.e., trials with invalid cue minus trials with valid cue), and executive control (i.e., incongruent minus congruent trials). Vigilance indices are divided into executive and arousal. The former comprises hits (i.e., percentage of trials the displacement of the central arrow is correctly detected by pressing the space bar) and false alarms (i.e., percentage of trials the space bar is pressed when there is a minor, but no substantial, displacement of the central arrow[21]). Arousal vigilance indices include mean RT, standard deviation of the RT, and lapses (i.e., percentage of trials with an RT higher than 600 ms or no response) to stop the millisecond counter (**Figure 1**). In addition to the overall scores, the vigilance indices include the slope of decrement over task blocks with time on task. This vigilance decrement manifest as a decrease in executive vigilance indices (hits and false alarms) and as an increase in arousal vigilance indices (mean RT, standard deviation RT and lapses).

Luna, Roca, et al. (2021) provided evidence supporting that the ANTI-Vea can be administered in both lab and online settings, with no substantial difference in the reliability of its indices. In the present study, we estimated the internal consistency scores of the ANTI-Vea core indices for each sample. To do so, we used a permutation-based split-half approach (Parsons et al., 2019) with 5,000 random splits. The resulting Spearman-Brown corrected coefficients are presented in **Table 2**. Compared to Luna, Roca, et al., the reliability scores we obtained were similar in Sample 1 and Sample 2 but higher in Sample 3.

Data Analysis

Of the 493 participants recruited, we excluded 31 (6.29%), 12 due to an incomplete task session (all from Sample 2) and 19 due to having more than 25% errors in attentional

[21] The five arrows are slightly vertically displaced at random by ± 2 px to generate some noise, while the target is substantially vertically displaced by ± 8 px in executive vigilance trials. The index of false alarms only considers those attentional network trials with more than 2 px of displacement from the target to at least one of its two adjacent flankers. They have been referred to as difficult false alarms.

network trials.[22] This left us with a final sample of 462 participants (39.2% men, 18–32 years, see **Figure 3A**). ANTI-Vea RT measures only considered correct trials that were within 200–1500 ms for attentional network indices and equal to or lower than 2000 ms for arousal vigilance indices. The score for each ADHD symptom scale (i.e., the cBAARS-IV and the aBAARS-IV) was the sum of its items. Applying the procedure of our primary studies (Coll-Martín, Carretero-Dios, & Lupiáñez, 2023; Coll-Martín et al., 2021), we compared the statistics and distribution of both ADHD scores in our final sample with a normative sample.

To capture the dependency structure in our data (see **Figure 2**), we employed a two-step approach with linear mixed-effects models (for additional details, see **Supplemental Text 1**). In the first step, we fitted a general linear mixed model for each ANTI-Vea index based on RT, and a logistic mixed model for each index based on percentage. For indices derived from task manipulations, namely the three attentional networks or the vigilance slopes, the model included the slope of task condition or block, respectively, as a fixed effect. We followed Barr et al.'s (Barr et al., 2013) guidelines for confirmatory hypothesis testing by including the maximal random effects structure justified by the design. Therefore, both participant and task session were included as crossed random intercepts and, when applicable, crossed random slopes of the manipulation effect. In line with recommendations for handling random effects with few units (Harrison et al., 2018), the variable sample was transformed into two dummy variables, which were introduced as fixed factors to control for their main effect and, when applicable, interactions with the task manipulation.

In the second step, we derived the best linear unbiased predictor (BLUP) for the by-participant random intercepts or, when applicable, slopes (Robinson, 1991; but see

[22] In Sample 3 (Multiple), there were 5 participants who did not perform Task Session 1, although the entire sample completed the rest of the sessions. We excluded one participant with poor performance in all task sessions. For the rest of the participants in this sample, we only excluded nine single sessions with poor performance.

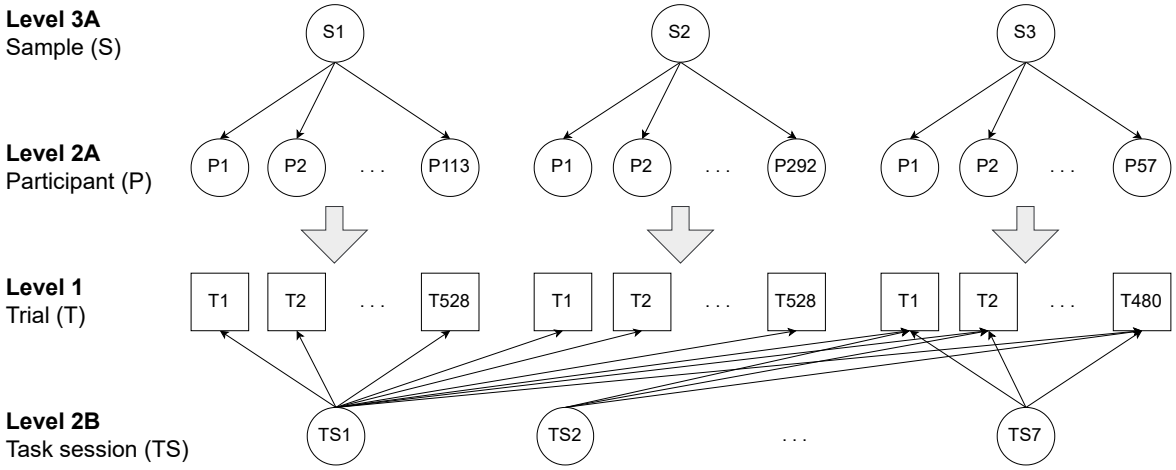


Figure 2. Dependency Structure of the Study Data in Random Factors. Observations and clusters are represented by squares and circles, respectively. Arrows depict the hierarchical relationship between units across different levels, with thick arrows indicating that all units at the higher level relate to all units at the lower level within the cluster. Units omitted from each factor by ellipses maintain the same hierarchical relationships as their two horizontally adjacent units. Participants from S1 and S2 performed a version of the task with 48 irrelevant distraction trials, not analyzed in this study, that did not affect the rest of the task indices or the reliability scores (Coll-Martín et al., 2021). For S3, each participant completed 480 trials per session over 7 sessions.

Hadfield et al., 2010, for a cautious interpretation of this technique in certain contexts). The BLUP of each ANTI-Vea index was employed as the outcome of three multiple linear regression models that differed in the measure of ADHD symptoms used as a predictor of interest: the cBAARS-IV (childhood), the aBAARS-IV (adulthood), or the aBAARS-IV after accounting for the cBAARS-IV ("adulthood unique" or "late onset"). All these models controlled for the main effects and interactions of sex and age with ADHD symptoms, along with the interaction between the two dummy variables of sample and symptoms of ADHD. Critical for the research question, in our sample the minimal statistical effect these regressions could detect was—on average[23]—as small as $\beta \approx 0.09$ for $\alpha = .05$, assuming no multicollinearity.

[23] Assuming $R^2 = .026$ for the entire multiple regression model, which was the mean size found in our analyses.

Transparency and Openness

Study information is provided following the Journal Article Reporting Standards for Quantitative Research in Psychology (JARS–Quant; Appelbaum et al., 2018). The entire workflow of the study has been run and documented in three reproducible R scripts (Version 4.2.1). These scripts, along with all data sets and codebooks, are publicly available at https://osf.io/53ubs/. The ANTI-Vea is freely accesible at https://anti-vea.ugr.es/. The study was not preregistered.

Results

ADHD Self-Reports

Figure 4B shows the distribution of the cBAARS-IV and the aBAARS-IV for each sample. **Figure 4C–D** compares the distribution of the scores obtained in our sample for each scale to that from an estimated normative sample (for a detailed procedure and statistical report, see **Supplemental Text 2**). Taken together, the scores of ADHD symptoms in our sample of young university students were higher than in the general population of adults. Even so, the spread and variability within each scale did not significantly differ from those observed in the estimated normative sample. The correlation between the cBAARS-IV (M = 30.65, SD = 9.42) and the aBAARS-IV (M = 30.04, SD = 7.85) was statistically significant, $r(460) = .54$, $p < .001$.

ANTI-Vea Indices

Table 2 shows the means and standard deviations of each ANTI-Vea index across samples (see **Supplemental Table 2** and **3** for these descriptive statistics per sample and per session within Sample 3, respectively; see **Supplemental Table 4** for correlations among indices). It also includes the reliability scores and correlation coefficients with both

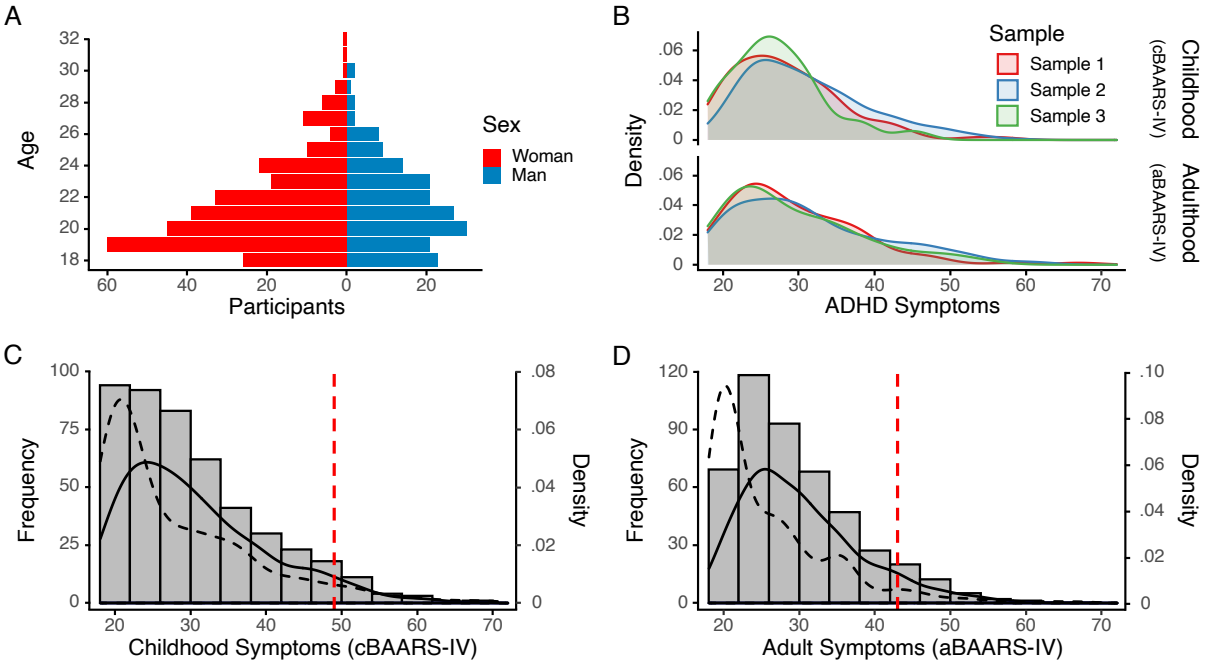


Figure 3. Basic Demographics and Distribution of Total ADHD Symptom Scores per Sample and Compared to an Estimated Normative Sample. N = 462 (n_1 = 113, n_2 = 292, n_3 = 57). **Panel A**: Age-sex pyramid. **Panel B**: Density curves per sample for each measure of ADHD symptoms. **Panels C–D**: Histogram and black solid line represent the frequency and density curve of ADHD total scores in the sample of the present study. The dashed black line represents the density curve of ADHD scores in an estimated normative sample based on the percentile values reported by Barkley (2011; see Supplemental Table 1). The dashed vertical red line represents the 95th percentile in Barkley's normative sample. ADHD = attention-deficit/hyperactivity disorder; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms.

scales of ADHD symptoms per sample. As expected, the reliability scores from the single session of the task performed in the lab (Sample 1) and online (Sample 2) were similar to each other, and both were lower than the scores from the multiple task sessions (Sample 3). Furthermore, overall scores in attentional networks and vigilance showed higher reliability coefficients than those from difference scores attentional networks and slopes of vigilance decrement, respectively. On another note, the rate of significant correlations was unsurprisingly higher in the largest sample (Sample 2) than in the two others.

The results of mixed-effects models for task manipulations (see **Supplemental Tables 5–12**) were in line with **Table 2**. Across samples, participants, and task sessions, responses were significantly slower and had more errors in no-tone versus tone (alerting

Table 2

Means, Standard Deviations, Reliability Scores and Pearson's Correlation Coefficients With ADHD Symptoms for ANTI-Vea Indices

Index	*M* (*SD*)	Internal consistency (r_{SB})			Correlation with ADHD symptoms (*r*)					
					Childhood (cBAARS-IV)			Adulthood (aBAARS-IV)		
		S1	S2	S3	S1	S2	S3	S1	S2	S3
Attentional network										
Overall RT	598 (88)	.99	.99	1	.07	.04	.06	–.09	–.05	.05
% Overall errors	5.42 (4.04)	.91	.89	.98	.06	.03	–.13	–.08	.19**	–.11
Alerting RT	37 (31)	.23	.20	.70	.00	–.02	–.19	.12	.09	–.04
% Alerting errors	2.43 (4.61)	.31	.13	.66	.11	.00	–.09	.18	.00	–.14
Orienting RT	38 (23)	.33	.26	.53	–.05	.02	.12	–.02	–.11	.03
% Orienting errors	0.77 (3.72)	.25	.32	.51	–.01	.08	.03	–.07	.09	–.13
Congruency RT	41 (27)	.65	.65	.84	.06	.10	.33*	.05	.20***	.03
% Congruency errors	1.04 (4.01)	.60	.62	.81	.14	.07	.22	.05	.13*	–.09
Executive vigilance										
% Hits	72.27 (17.82)	.94	.95	.99	.00	–.14*	–.27*	–.04	–.18*	.01
% False alarms [a]	5.30 (5.13)	.85	.77	.97	–.03	–.07	–.26	–.07	.01	–.11
% Hit slope	–1.98 (3.42)	.27	.61	.79	–.09	.03	–.21	–.10	.00	.02
% False alarm slope [a]	–0.49 (1.58)	.40	.24	.65	.10	.17**	.07	–.13	.02	.06
Arousal vigilance										
***M* RT**	494 (87)	.97	.99	1	.15	.17**	.24	.08	.16**	.27*
***SD* RT**	83 (36)	.83	.78	.96	.16	.17**	–.03	.10	.26***	.05
% Lapses	11.17 (16.82)	.96	.98	1	.14	.18**	.26	.14	.21***	.23
***M* RT slope**	3.18 (10.75)	.82	.76	.84	.25**	.00	–.16	.17	.08	–.16
***SD* RT slope**	4.45 (9.27)	.36	.45	.38	.14	–.01	–.08	.06	.05	–.03
% Lapse slope	1.24 (2.83)	.78	.68	.77	.22*	.01	–.20	.16	.08	–.09

Note. *N* = 462 (S1 = 113, S2 = 292, S3 = 57). Means are weighted per sample size, and standard deviations represent the between-participant variability. For S3, the mean of each task session was calculated for each participant before averaging across participants. RT measures are in milliseconds. ADHD = attention-deficit/hyperactivity disorder; ANTI-Vea = Attention Network Test for Interactions and Vigilance—Executive and Arousal Components; r_{SB} = Spearman-Brown split-half reliability coeficient; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample; RT = reaction time.

effect), invalid versus valid (orienting effect), and incongruent versus congruent (congruency effect) trials conditions (all *p*s < .001). Furthermore, throughout the six task blocks, the slopes of executive vigilance were significantly negative, while those of arousal vigilance were significantly positive (all *p*s < .01). Taken together, the ANTI-Vea worked successfully to generate the indices resulting from task manipulations.

Multi-Sample Association Between Attentional Processes and ADHD Symptoms

Figure 4 displays the association between the 12 ANTI-Vea indices and ADHD symptoms in childhood, adulthood, and late onset (i.e., symptoms in adulthood after controlling for those in childhood). To present a more interpretable effect size, the estimates in the figure have been transformed into semipartial correlations by multiplying the standardized regression coefficients by the square root of their corresponding tolerance values. Tolerance was .92, .95, and .69 in childhood, adulthood, and late-onset models, respectively. **Supplemental Tables 13–30** provide detailed information of the three models for each task index. Higher scores in ANTI-Vea indices reflect worse performance—except for hits and hit slope, where higher scores indicate better performance.

In the overall performance of the attentional networks, none of the three regression models showed a significant prediction of ADHD symptoms on RT (all *p*s > 0.08). In contrast, only ADHD symptoms in adulthood were significantly associated with a higher probability of errors (β = 0.10, *p* = .044), while neither childhood nor late-onset symptoms predicted this outcome (both *p*s > .10). Regarding the alerting network, we found that adult symptoms after controlling for symptoms in childhood (i.e., late-onset symptoms) uniquely predicted a higher RT (β = 0.12, *p* = .032). However, neither the childhood nor the adulthood models for RT showed significant associations with ADHD symptoms (both *p*s > .16), nor did any of the three models for error rate (all *p*s > .34). As for the orienting network, only late-onset symptoms were negatively associated with RT (β = –0.12, *p*

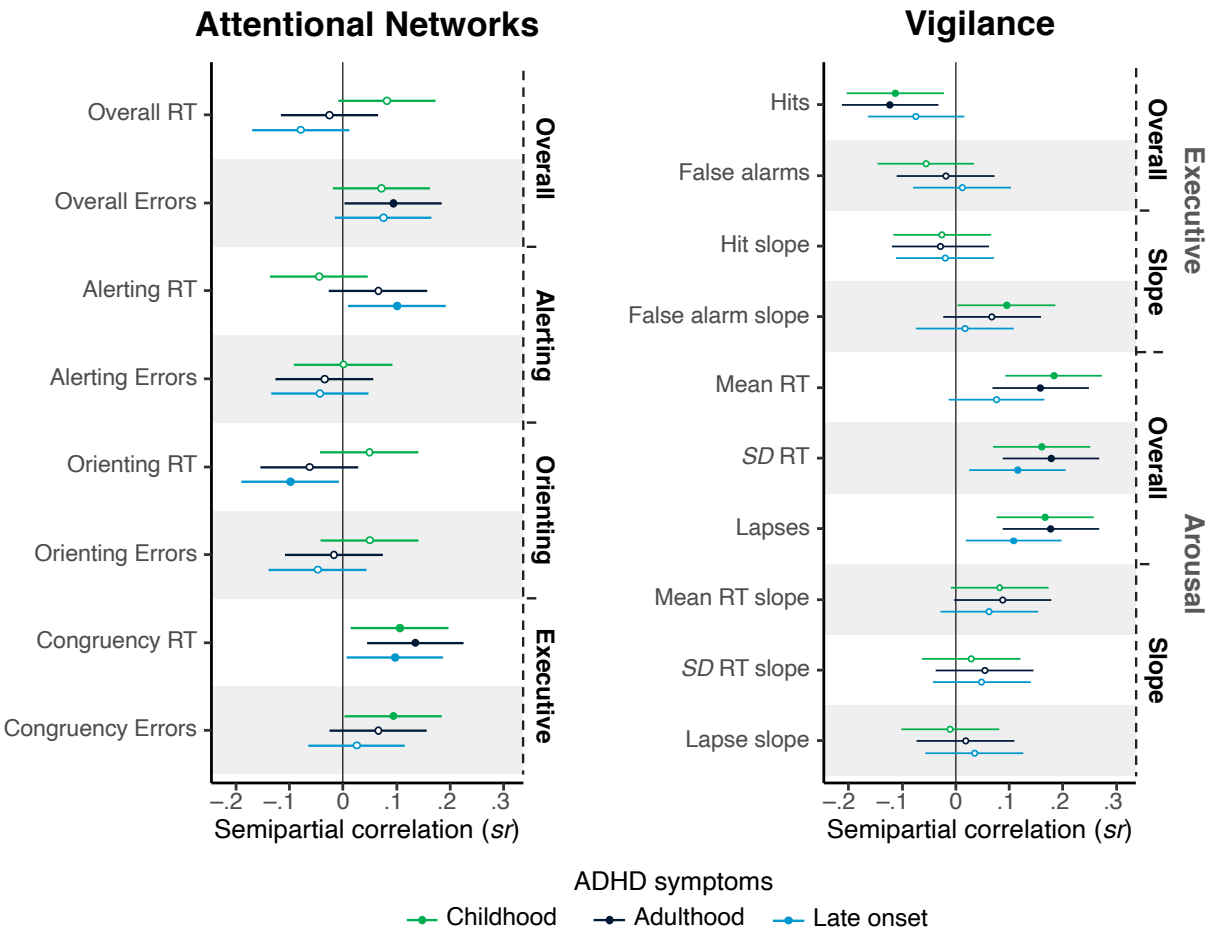


Figure 4. Association Between Attentional Processes (ANTI-Vea Indices) and ADHD Symptoms at Different Ages and Onsets. F *N* = 462 (*n* = 459 for false alarms: overall scores and slopes). Task indices are to the left of each panel, while their corresponding attentional processes are to the right of the panel. Higher scores reflect worse performance (except for hits and hit slope, where higher scores indicate better performance). The position of each circle on the horizontal axis indicates the size of the semipartial correlation, with filled circles representing statistically significant effects and unfilled circles indicating nonsignificant effects. The horizontal lines represent the 95% confidence intervals. The predictor of interest included in each model is ADHD symptoms in (a) childhood retrospectively assessed, (b) adulthood, or (c) late onset—namely symptoms in adulthood after adjusting for those in childhood. All models control for sex, age, and sample. ADHD = attention-deficit/hyperactivity disorder; ANTI-Vea = Attention Network Test for Interactions and Vigilance—Executive and Arousal Components.

= .033), while neither of the other two models for RT nor any of the three models for error probability found ADHD symptoms to be significant (all *p*s > .17). In the case of the executive network, childhood (β = 0.11, *p* = .024), adulthood (β = 0.14, *p* = .004), and late-onset symptoms (β = 0.12, *p* = .038) predicted a higher congruency effect for RT, while only symptoms in childhood (β = 0.12, *p* = .045) were associated with higher error rate—but not in adulthood or late onset, both *p*s > .15.

With respect to executive vigilance, symptoms in childhood (β = 0.12, *p* = .015) and adulthood (β = 0.13, *p* = .008), but not in late onset (β = 0.09, *p* = .105), were negatively associated with the probability of hits. Furthermore, neither the overall scores of false alarms (all *p*s > .22) nor the slopes of hits (all *p*s > .52) were associated with ADHD symptoms in any of the models. In the slope of false alarms, childhood symptoms showed a positive relation (β = 0.10, *p* = .043), while neither adulthood nor late-onset symptoms made significant predictions (both *p*s > .15). Regarding arousal vigilance, except for late-onset symptoms for mean RT (β = 0.09, *p* > .09), all symptoms in childhood (βs = 0.17–0.19, all *p*s < .001), adulthood (βs = 0.16–0.18, all *p*s = .001), and late onset (βs = 0.13–0.14, both *p*s = .02) predicted higher overall mean and standard deviation of the RT as well as a higher rate of lapses. Conversely, in none of the models was there a significant association between ADHD symptoms and mean RT, mean SD, and lapse slopes (all *p*s > .06).

Discussion

Combining three samples of community adults, this study analyzed the associations between the attentional processes assessed with the ANTI-Vea—namely the three attentional networks of alerting, orienting, and executive control, as well as the executive and arousal vigilance components—and self-reported ADHD symptoms at different ages and time onsets. We found that the pattern of attentional alterations predicted by retrospective symptoms in childhood was virtually identical to that of adult symptoms: Specifically, of the total 18 ANTI-Vea indices, the only three that showed discrepancies in statistical significance between both ADHD measures had minimal differences in effect sizes. Albeit with some change in task indices, predictions made by late-onset symptoms were close to those of symptoms in childhood and adulthood. Crucially, alterations in both arousal and executive attentional processes were predicted by the three types of ADHD

symptoms, which arguably supports the quantitative difference models of late-onset ADHD.

The similar pattern of impaired attentional processes in both childhood and adult ADHD symptoms is consistent with previous studies with clinical (Arora et al., 2020; Huang-Pollock et al., 2012; Kofler et al., 2013) and community samples (Aduen et al., 2020; Servera & Cardo, 2006). This supports the notion of lifespan stability and homotypic continuity of the neurocognitive mechanisms underlying ADHD symptoms, at least those starting in childhood. Regarding late-onset symptoms, our findings about their association with executive alterations align with the deficits exhibited by the group with late-onset ADHD in a longitudinal cohort (Riglin et al., 2022), although they contradict the lack of that executive impairment found in two cohorts with small group sample sizes (Cooper et al., 2018; Moffitt et al., 2015). Furthermore, our results showed a relation between late-onset symptoms and poor arousal processes, which corroborate initial evidence from a case-control comparison with lower statistical power (Ilbegi et al., 2021).

While we found no task measures associated exclusively with both adult and late-onset ADHD symptoms, two indices were predicted solely by late-onset symptoms: alerting RT and orienting RT. As shown in Table 2, the reliability scores of these two measures were among the lowest in the ANTI-Vea. The positive relation between late-onset symptoms and a higher alerting RT effect (i.e., worse performance) was partially due to a nonsignificant negative correlation of that index with symptoms in childhood. Given that ADHD is theoretically (Berger & Posner, 2000) and empirically (Arora et al., 2020) linked to deficits in the alerting network, and considering the poor reliability of the index, this negative correlation may be more likely attributed to random noise. As for orienting RT, in addition to being the only ANTI-Vea process with no hypothesized link to ADHD (Coll-Martín et al., 2021), its association with late-onset symptoms was negative—that is, higher symptoms predicted a better functioning of the network. Although future

studies should consider both indices as potential specific correlates of late-onset ADHD or state (vs. vulnerability) markers of symptomatology, the described theoretical and statistical caveats call to be cautious at this time.

Neurocognitive Nature of Late-Onset ADHD

Beyond the above-mentioned nuances, our pattern of attentional associations for late-onset ADHD symptoms closely aligned with those for adult and, critically, childhood symptoms. Indeed, the three types of symptoms predicted alterations in both executive and arousal processes. The similar neurocognitive impairments associated with child- and late-onset symptoms are in line with most group comparisons from longitudinal cohorts (Cooper et al., 2018; Ilbegi et al., 2021; Riglin et al., 2022). In the same vein, studies that set the age-of-onset criterion at age 7 reported no differences in neurocognitive deficits between early- and late-onset ADHD (Faraone, Biederman, Doyle, et al., 2006; Guimarães-da-Silva et al., 2012).

Regarding neurocognitive accounts of late-onset ADHD, our results supported the quantitative difference models. That is, late-onset symptoms reflect the same neurocognitive alterations as childhood symptoms and only differ in the temporal dimension they are expressed. This is noteworthy, as our well-powered design included measures of two dissociable processes—namely executive and arousal—to enable the examination of models predicting qualitative differences in the mechanisms underlying late- versus child-onset symptoms. Assuming quantitative rather than qualitative differences implies to reject models of late-onset ADHD based on different neurocognitive alterations (Halperin & Schulz, 2006), lack of neurocognitive deficits (Moffitt et al., 2015), or mimics from conditions with other neurocognitive impairments (Taylor et al., 2022). However, it cannot be ruled out that qualitative differences may be observed in a subset of late-onset ADHD or in neurocognitive pathways that have not yet been analyzed (e.g., delay aversion, Sonuga-Barke et al., 1992).

Within the quantitative difference models, there are two hypotheses that could potentially explain the similar neurocognitive mechanisms in child- and late-onset ADHD. The first points to the *moderating* factors of the relationship between neurocognitive processes and symptoms. According to this view, neurocognitive alterations are relatively stable from birth, but symptoms may remain obscured until later when the external (e.g., supportive family) or internal (e.g., high IQ or, based on our data, a strong orienting network) scaffoldings are removed or insufficient to meet environmental demands (Faraone & Biederman, 2016; Kosaka et al., 2019). The second hypothesis, related to the complex phenotype framework (Caye et al., 2017), focuses on the *etiological* factors of ADHD across lifespan. From this perspective, the array of neurocognitive alterations underpinning ADHD symptoms can also be caused after the age of 12 by interactions between biological (e.g., brain damage, impaired maturation, substance abuse), psychological (e.g., stress, effort or delay aversion), and environmental (e.g., high demands, harsh reactions) variables. Note that the first hypothesis deems ADHD as a neurodevelopmental disorder regardless of age of onset, while in the second hypothesis ADHD is better conceptualized as a non-neurodevelopmental, general mental health disorder. Future studies testing these hypotheses in longitudinal designs are warranted.

The support our findings provide for the quantitative difference models entails practical implications. Consistent with Asherson and Agnew-Blais (2019), we suggest that the treatment of ADHD symptoms in adulthood should not vary based on the reported age of onset. Given our neurocognitive data, this recommendation is especially relevant in the design and implementations of translational interventions for ADHD, namely cognitive training, neurofeedback, or brain stimulation. In these treatments, the neurocognitive targets should be the same irrespective of the individual's age or the onset of the impairing symptoms.

Limitations

We have identified three main limitations in the design of our study. The first regards the composition of our sample. Our community sample was mainly made up of university students, which is not representative of the young adult population. Furthermore, ADHD symptoms in childhood have been associated with lower educational attainment in adulthood (Galéra et al., 2012; Pingault et al., 2011). Despite this sampling bias, our analyses did not suggest that the distribution of ADHD symptom scores in our sample was more restricted or homogeneous than in a normative community sample. In any case, replication of our findings with a more representative sample is warranted. Additionally, although the dimensional framework assumes that the neurocognitive correlates of ADHD symptoms remain constant throughout a continuous trait, the empirical extension of our results to clinical populations is needed.

Second, the validity of retrospective reports of ADHD symptoms has been questioned due to potential recall bias. Indeed, longitudinal studies finding a modest correlation between prospective and retrospective parent ratings of symptoms in childhood (e.g., r = .39; von Wirth et al., 2021). However, given the changing trajectory of ADHD symptoms throughout development (Sibley et al., 2022; Stern et al., 2020), part of the mismatch between both measures may be better explained by differences in the time span assessed by each symptom scale (e.g., last 6 months vs. whole childhood) rather than recall bias. Of note, Lundervold et al. (2021) found a 7-year test–retest reliability score of .89 for a retrospective self-report measure of ADHD symptoms in childhood. Looking on the bright side, our study design held constant the time of assessment, thereby controlling for recalibration bias, which is specific to prospective evaluation. In any case, the integration of distinct but complementary assessment methods is fundamental to advance the understanding of the neurocognitive processed associated to ADHD symptoms across lifespan.

The third limitation is about the statistical conclusion validity of our design. Within the existing literature, the present study has achieved the highest statistical power so far to address our research question. However, the power was still far from optimal to conduct more pertinent statistical analyses such as comparison between the three ADHD estimates within each task index, for which we would need around twice our current sample size. Furthermore, to strictly control the error rates in a severe test for each of our two theoretical models, a preregistered direct replication with sufficient sample size to correct for multiple comparisons is a crucial step. Moreover, the moderate-to-low reliability of some ANTI-Vea indices, which is a typical issue in most neurocognitive tasks (Dang et al., 2020), was likely to substantially reduce the power and effect sizes of their corresponding analyses in our sample. To provide context for our main analyses, particularly regarding promising measures that yielded nonsignificant results (e.g., vigilance slopes), we reported the reliability scores for all task indices (Table 2), which is an essential but uncommon practice in clinical psychological science (Parsons et al., 2019).

Conclusion

In a large multi-sample study of community adults, we found that childhood, adult, and late-onset ADHD symptoms were associated with both executive and arousal attentional deficits. This lends support to the idea that neurocognitive mechanisms underpinning child- and late-onset ADHD are similar. Consequently, translational interventions for late-onset symptoms should be the same as those designed for conventional ADHD. To consolidate and extend our tentative recommendations, future studies incorporating complementary assessment methods of ADHD symptoms, clinical groups, and additional neurocognitive domains are warranted.

Supplemental Materials

SUPPLEMENTAL TEXT 1: Additional Technical Information of the Regression Models Used in the Study

The two-step approach involved a first phase in which 18 mixed-effects models were fitted to compute each of the 18 ANTI-Vea indices. For the indices based on reaction times (RTs), we fitted a general linear mixed model using restricted maximum likelihood estimation and the "nloptwrap" optimizer. Confidence intervals (CIs) and *p*-values were computed using a Wald *t*-distribution approximation. For the indices based on percentages (i.e., error percentage, hit percentage, false alarm percentage, and lapse percentage), we fitted a logistic mixed model using a maximum likelihood estimation via Laplace approximation and the "bobyqa" optimizer. CIs and *p*-values were computed using a Wald *z*-distribution approximation.

The goal of this initial phase was to yield a score for each participant (for each of the 18 models), taking into account all the fixed and random factors included in that model. In doing so, the comparison of scores between participants become more precise than by merely obtaining an average of responses for each participant without considering response type or the dependency structure of the data. Participant scores from the mixed-effects models were obtained by using the best linear unbiased predictor (BLUP) for the by-participant random intercepts or, when applicable (i.e., in ANTI-Vea indices including manipulation of the task conditions or vigilance slope across task blocks), slopes.

The second stage consisted of standard multiple linear regression models with the BLUP of each index as the dependent variable. In total, 54 regression models were run in this phase (18 indices by three types of ADHD predictor). CIs and *p*-values were computed using a Wald *t*-distribution approximation.

To enhance accuracy and avoid convergence issues, we scaled and centered the slopes in all models—this applies to models from both the first and second stages. Our two-step approach was also crucial in facilitating the convergence of the mixed-effects models. When faced with convergence or singularity issues, we adopted different strategies, including the use of various optimizers, increasing the number of iterations, and modifying the type or order of the dummy variables derived from the factor sample. As a result, all the mixed-effects models converged appropriately, although we had to remove the by-task session random slope of orienting errors due to singularity issues with the maximal random structure. Based on Cook's distance, none of the mixed-effects or multiple regression models presented influential observations. No predictor of interest in the three multiple regression models computed for each task index presented multicollinearity problems: variance inflation factors (VIFs) for Model 1 (childhood symptoms), Model 2 (adult symptoms), and Model 3 (adult symptoms) were 1.09, 1.06, and 1.44, respectively.

The regression analyses were executed with the following R packages: *lme4* ((Bates et al., 2015) to fit the mixed-effects models, *sjPlot* (Lüdecke, 2022) to obtain and compute some specific statistics of the models, and *performance* (Lüdecke et al., 2021) to check the fit of the models.

SUPPLEMENTAL TEXT 2: Attention-deficit/hyperactivity disorder (ADHD) Symptom Distribution in our Study Compared to Normative Values and Distributions

Procedure

We calculated the total scores (i.e., sum of the items) of ADHD symptoms for the each of the two self-reported scales of the Barkley Adult ADHD Rating Scale-IV (BAARS-IV; Barkley, 2011): Childhood Symptoms (cBAARS-IV) and Current Symptoms (aBAARS-IV; i.e., symptoms in adulthood). The scores obtained in our sample were compared with the percentile values obtained by Barkley from a large representative sample of young adults from the United States (see Supplemental Table 1). Barkley reported the values corresponding to 22 and 20 percentiles for the cBAARS and the aBAARS, respectively. For each scale in our sample, we analyzed the percentage of individuals scoring above the preliminary cutoff, set to the 95th percentile in Barkley's normative sample. Furthermore, we compared the medians obtained in our sample with the 50th percentile values in Barkley's normative sample.

In addition to the percentile values, we compared the distribution of the scores in our sample with an estimation of the Barkley's (2011) normative distribution. Based on the values of Barkley's percentiles, we generated a sample of 10,000 simulated total scores for the cBAARS and the aBAARS using a Monte Carlo simulation approach. Percentile values of the simulated sample fitted those reported by Barkley for each ADHD symptom scale. From this simulated sample, we extracted 462 theoretical quantiles, which resulted in an estimated normative sample of equal size to our study sample. This equally sized sample was used to compare the distribution and variability of the scores with the sample from our study.

Results

Binomial tests indicated that while the percentage of participants above the 95th percentile cutoff in the cBAARS-IV (6.1%) was not significantly different from the

normative 5% (p = .285), such percentage in the aBAARS-IV (8.6%) was higher than that preliminary clinical cutoff (p < .001). Compared to the 50th percentile in the normative sample for the cBAARS-IV (*Mdn* = 23) and the aBAARS-IV (*Mdn* = 22), Wilcoxon Signed-Ranks Tests indicated that the medians of the cBAARS-IV (*Mdn* = 28) and the aBAARS-IV (*Mdn* = 29) in our sample were significatively higher (both p < .001). Two-sample Kolmogorov-Smirnov tests indicated that ADHD symptom distributions in the cBAARS-IV and the aBAARS-IV were significatively different from their corresponding normative sample (both p < .001). Indeed, a visual comparison in Figure 3 shows that the normative distributions are more left-skewed than their respective sample distributions. Finally, Levene's tests for homogeneity of variances showed no significant differences between the variability of the scores of the cBAARS-IV and the aBAARS-IV between our sample and the normative one (both ps > .240).

SUPPLEMENTAL TABLE 1: Percentiles Values of the cBAARS-IV and the aBAARS-IV in Original and Estimated Normative Samples Compared to the Sample of the Present Study

Percentile	cBAARS-IV total scores		aBAARS-IV total scores	
	Normative sample	Study sample	Normative sample	Study sample
99	60 (61)	58	54 (55)	53
98	55	54	49	50
97	52 (53)	52	46	48
96	51	51	44	47
95	49	49	43	45
94	47 (48)	48	42 (41)	44
93	46	47	39	43
92	45	46	38	43
91	44	46	37	42
90	43	45	36	41
89	42	44		
88	41 (40)	43		
87	39	42		
85	38	41	35	38
84	37	40		
83			34	37
82	36	39	33	37
81			32	36
79			31	35
77	35	37	30	35
76	34	36		
75	33	36	29	34
51	24 (23)	29	23 (22)	29
50	23	28	22	29
1	18	18	18	18

Note. Percentiles in the normative sample were extracted from Barkley (2011), which is based on a large representative sample of adults (ages 18–39) from the United States. These percentiles were used to generate a simulated normative sample through a Monte Carlo simulation approach (N = 10,000). The values of the simulated sample are in parentheses when they differ from the original normative sample. cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms.

SUPPLEMENTAL TABLE 2: Means and Standard Deviation per Sample for all ANTI-Vea Indices

ANTI-Vea index	Sample 1 (*n* =113)		Sample 2 (*n* = 292)		Sample 3 (*n* = 57) [a]	
	M	***SD***	***M***	***SD***	***M***	***SD***
Attentional network						
Overall RT	600	96	597	92	598	101
% Overall errors	5.75	4.34	5.25	3.97	5.60	3.77
Alerting RT	**35**	**33**	**37**	**32**	**39**	**22**
%Alerting errors	**2.34**	**5.28**	**2.46**	**4.60**	**2.47**	**2.98**
Orienting RT	**35**	**26**	**40**	**24**	**32**	**12**
% Orienting errors	**0.65**	**3.90**	**0.85**	**3.92**	**0.56**	**1.85**
Congruency RT	**40**	**29**	**43**	**28**	**37**	**17**
% Congruency errors	**1.46**	**4.21**	**0.92**	**4.18**	**0.79**	**2.34**
Executive vigilance						
% Hits	68.62	17.29	73.08	18.28	75.34	15.54
% False alarms	5.16	5.09	5.41	5.19	5.08	4.95
% Hit slope	**–1.74**	**3.00**	**–2.17**	**3.78**	**–1.47**	**1.90**
% False alarm slope	**–0.42**	**1.51**	**–0.51** [b]	**1.71** [b]	**–0.50**	**0.93**
Arousal vigilance						
Mean RT	507	61	492	95	478	83
***SD* RT**	92	42	79	33	86	34
% Lapses	12.98	14.35	10.57	17.69	10.68	16.77
Mean RT slope [c]	**6.18**	**13.54**	**2.07**	**10.12**	**2.94**	**5.35**
***SD* RT slope** [c]	**5.24**	**10.63**	**4.33**	**9.41**	**3.49**	**4.46**
% Lapse slope	**2.00**	**3.74**	**1.09**	**2.60**	**0.53**	**1.16**

Note. RT measures are in milliseconds. In bold are the indices resulting from comparing task conditions (i.e., stimulus manipulation in attentional network indices and vigilance decrement in executive and arousal vigilance indices). All one-sample t tests (against zero) of the indices in bold for each sample yielded significant results ($p < .05$), except for orienting RT in Sample 1 ($p = .077$). ANTI-Vea = Attention Network Test for Interactions and Vigilance—Executive and Arousal Components; RT = reaction time.

[a] The mean of each task session was calculated for each participant before averaging across participant scores. [b] $n = 289$. [c] For the calculation of the index, five task sessions (all from different participants) were dropped from the analyses because Block 1 did not contain correct trials in these sessions. This procedure also applied to the results showed in **Table 2**.

SUPPLEMENTAL TABLE 3: Means (With Standard Deviations in Parentheses) per Session in Sample 3 for all ANTI-Vea Indices

Index	TS1 (*n* = 51)	TS2 (*n* = 56)	TS3 (*n* = 54)	TS4 (*n* = 55)	TS5 (*n* = 56)	TS6 (*n* = 57)	TS7 (*n* = 56)
Attentional network							
Overall RT	632 (102)	612 (104)	606 (107)	607 (123)	591 (117)	574 (105)	580 (108)
% Overall errors	6.11 (4.61)	4.50 (3.94)	5.68 (4.79)	6.24 (5.51)	6.36 (5.27)	5.46 (5.08)	3.76 (3.11)
Alerting RT	**44 (37)**	**38 (32)**	**36 (39)**	**38 (30)**	**42 (36)**	**37 (38)**	**32 (27)**
% Alerting errors	**2.08 (4.77)**	**2.57 (4.19)**	**1.66 (5.09)**	**2.61 (5.91)**	**2.90 (5.27)**	**2.78 (4.58)**	**2.31 (4.05)**
Orienting RT	**38 (21)**	**301 (22)**	**38 (20)**	**37 (23)**	**30 (22)**	**35 (21)**	**21 (17)**
% Orienting errors	**1.10 (3.72)**	**–0.15 (3.69)**	**0.60 (3.64)**	**1.02 (3.74)**	**0.76 (3.71)**	**0.99 (3.22)**	**–0.50 (3.51)**
Congruency RT	**37 (32)**	**32 (24)**	**43 (24)**	**44 (25)**	**40 (24)**	**37 (21)**	**27 (19)**
% Congruency errors	**0.34 (4.48)**	**0.19 (3.08)**	**0.44 (4.16)**	**1.65 (3.81)**	**1.18 (3.61)**	**0.66 (3.39)**	**0.83 (2.36)**
Executive vigilance							
% Hits	73.67 (19.77)	74.85 (20.37)	74.38 (21.92)	74.81 (16.71)	76.67 (16.31)	75.49 (16.84)	79.59 (14.15)
% False alarms	6.24 (6.12)	5.24 (6.32)	5.59. (6.66)	5.10 (6.49)	5.14 (5.68)	4.57 (5.44)	3.36 (4.50)
% Hit slope	**–1.38 (4.37)**	**–3.15 (4.13)**	**–1.44 (3.62)**	**–0.98 (3.08)**	**–0.58 (4.70)**	**–0.99 (3.57)**	**–1.65 (2.94)**
% False alarm slope	**–0.90 (2.16)**	**–0.81 (1.84)**	**–0.48 (1.56)**	**–0.13 (2.09)**	**–0.29 (1.76)**	**–0.49 (2.14)**	**–0.48 (1.19)**
Arousal vigilance							
***M* RT**	500 (97)	495 (106)	479 (88)	478 (91)	472 (81)	472 (75)	449 (56)
***SD* RT**	89 (43)	82 (41)	83 (37)	95 (60)	88 (44)	81 (38)	80 (31)
% Lapses	11.42 (16.42)	11.87 (20.67)	10.53 (18.99)	11.40 (19.14)	11.40 (19.96)	10.03 (17.42)	6.72 (10.01)
***M* RT slope** [a]	**3.46 (10.18)**	**1.62 (7.89)**	**2.33 (10.71)**	**1.66 (8.86)**	**3.37 (10.40)**	**2.26 (9.37)**	**6.08 (7.65)**
***SD* RT slope** [a]	**4.74 (10.88)**	**3.35 (8.50)**	**2.54 (9.94)**	**1.84 (9.80)**	**4.54 (13.42)**	**3.71 (8.83)**	**4.06 (6.80)**
% Lapse slope	**1.33 (1.97)**	**0.34 (1.66)**	**0.33 (3.48)**	**0.56 (1.98)**	**0.18 (3.52)**	**0.22 (2.80)**	**1.14 (2.40)**

Note. RT measures are in milliseconds. In bold are the indices resulting from comparing task conditions (i.e., stimulus manipulation in attentional network indices and vigilance decrement in executive and arousal vigilance indices). All one-sample t tests (against zero) of the indices in bold for each session yielded significant results ($p < .05$), except for orienting errors (TS2, TS3, TS5, and TS7), congruency errors (S1, S2, S3, and S6), hit slope (S5), false alarm slope (S4, S5, and S6), mean RT slope in arousal vigilance trials (S2, S3, S4, and S6), standard deviation RT slope in arousal vigilance trials (S3, and S4). ANTI-Vea = Attention Network Test for Interactions and Vigilance—Executive and Arousal Components; TS1 = Task Session 1; TS2 = Task Session 2; TS3 = Task Session 3; TS4 = Task Session 4; TS5 = Task Session 5; TS6 = Task Session 6; TS7 = Task Session 7; RT = reaction time.

SUPPLEMENTAL TABLE 4: Pearson Correlations Among ANTI-Vea Indices

Index	1	2	3	4	5	6	7	8	9
Attentional network									
1. Overall RT	—	.22	.15	.33*	.32*	–.18	.04	–.29*	.45***
2. % Overall errors	.23***	—	.16	.50***	–.03	.08	–.11	.06	–.01
3. Alerting RT	–.00	.11*	—	.28*	.21	–.04	–.28*	–.40**	.10
4. % Alerting errors	.09	.14**	.08	—	.05	–.07	–.06	.04	–.05
5. Orienting RT	–.01	–.11*	.03	.02	—	.08	–.01	–.35**	.26
6. % Orienting errors	–.13**	.10*	.07	.08	.08	—	–.06	.07	–.34*
7. Congruency RT	–.13**	.05	–.09	.15**	.01	.15**	—	.38**	–.20
8. % Congruency errors	–.22***	.22***	–.05	.16**	–.09	.14**	.33***	—	–.59***
Executive vigilance									
9. Hits	.32***	–.17***	.08	–.06	.01	–.38***	–.29***	–.42***	
10. False alarms [a]	.31***	.64***	.13**	–.07	–.16**	–.15**	–.19***	–.25***	.31***
11. Hit slope	.15**	–.01	.06	.01	–.05	–.11*	–.11*	–.11*	.37***
12. False alarm slope [a]	–.03	–.10*	.00	.02	.07	.04	.09	.11*	–.04
Arousal vigilance									
13. *M* RT	.42***	.23***	.06	.14**	–.01	.10*	.09	–.03	–.10
14. *SD* RT	.38***	.36***	.01	.27***	–.07	.11*	.15**	.16**	–.17***
15. % Lapses	.45***	.25***	.03	.16**	–.02	.07	.10*	–.02	–.09
16. *M* RT slope	.22***	.18***	.08	.20***	–.03	.00	.07	.12*	–.09
17. *SD* RT slope	.12*	.20***	–.01	.17***	–.07	.01	.13**	.11*	–.10*
18. % Lapse slope	.20***	.15**	.07	.22***	–.06	–.07	.10*	.16**	–.08

Index	10	11	12	13	14	15	16	17	18
Attentional network									
1. Overall RT	.30*	.03	–.16	.48***	.58***	.43***	.23	.29*	.47***
2. % Overall errors	.65***	.25	–.43***	.32*	.57***	.33*	.11	.21	.01
3. Alerting RT	.15	.12	.16	.30*	.26	.24	.12	.24	.10
4. % Alerting errors	.06	.10	.06	.42**	.73***	.43***	.37**	.40**	.25
5. Orienting RT	.14	.06	–.21	.42**	.19	.32*	–.29*	.01	.19
6. % Orienting errors	–.09	–.09	.04	.04	–.08	–.00	–.18	–.18	–.25
7. Congruency RT	–.20	.01	.03	.01	–.07	.02	.11	–.03	.07
8. % Congruency errors	–.49***	–.14	.32*	–.09	.08	–.00	–.10	–.06	–.24
Executive vigilance									
9. Hits	.47***	.26	–.33*	.02	.07	–.03	.17	.20	.39**
10. False alarms [a]	—	.29*	–.70***	.09	.15	.06	.01	.08	.15
11. Hit slope	.18***	—	.00	.00	.09	.04	.12	.11	–.13
12. False alarm slope [a]	–.19***	.06	—	.06	.04	.10	.04	–.02	–.12
Arousal vigilance									
13. *M* RT	.08	–.01	.05	—	.66***	.94***	–.19	.06	.21
14. *SD* RT	.05	–.12*	.05	.51***	—	.66***	.25	.45***	.43***
15. % Lapses	.08	–.04	.04	.89***	.63***	—	–.12	.05	.16
16. *M* RT slope	.03	–.05	–.08	.22***	.43***	.29***	—	.71***	.54***
17. *SD* RT slope	.01	–.09	–.02	.18***	.42***	.22***	.53***	—	.57***
18. % Lapse slope	–.00	–.08	.01	.25***	.40***	.32***	.80***	.51***	—

Note. The lower triangular section includes the pooled between-participant correlations of Sample 1 (n = 113) and Sample 2 (n = 292), as the indices exhibited similar reliability scores in both samples (see Table 2). The upper triangular section contains the between-participant correlations of Sample 3 (n = 57). The scores of each participant in this sample is the average across all their task sessions. ANTI-Vea = Attention Network Test for Interactions and Vigilance—Executive and Arousal Components; RT = reaction time.

[a] n = 289 in Sample 2.

*p < .05. **p < .01. ***p < .001.

SUPPLEMENTAL TABLE 5: Linear (RTs) and Logistic (Errors) Mixed Model for Overall Performance (Attentional Networks)

Component	Model: Overall RT		Model: Overall errors	
	Value	95% CI	Value	95% CI
Fixed effect				
Intercept	**579*****	**[559, 599]**	**4.13*****	**[3.46, 4.91]**
Sample 1 [a]	–12*	[–22, –2]	–0.16	[–0.48, 0.18]
Sample 2 [a]	–19**	[–31, –6]	–0.38	[–0.74, 0.01]
Random effect				
Residuals (σ^2)	19,947		3.29	
$\tau^2_{0\ participant}$	8,718		0.53	
$\tau^2_{0\ task\ session}$	432		0.04	
ICC	.31		.15	
$N_{participants}$	462		462	
$N_{task\ sessions}$	7		7	
N_{trials}	214,476		227,520	
	Explained variance			
Marginal R^2	.01		.00	
Conditional R^2	.32		.15	

Note. RT values are in milliseconds. The model for error is based on log-odds, although fixed effects are expressed in percentages in this table for interpretation purposes. The row in bold is the fixed estimate from which the by-participant random effects are extracted to be used at the second stage of analysis. Nonbolded fixed estimates are scaled and centered. RT = reaction time; CI = confidence interval; ICC = intra-class correlation.

[a] 0 = out, 1 = in.

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

SUPPLEMENTAL TABLE 6: Linear (RTs) and Logistic (Errors) Mixed Model for Alerting (Attentional Networks)

Component	Model: Alerting RT		Model: Alerting errors	
	Value	95% CI	Value	95% CI
Fixed effect				
Intercept	564***	[545, 584]	2.74***	[2.29, 3.28]
Tone [a]	**37*** **	**[34, 40]**	**2.19*** **	**[1.75, 2.67]**
Sample 1 [b]	–11*	[–22, –1]	–0.14	[–0.42, 0.17]
Sample 2 [b]	–19**	[–32, –5]	–0.30	[–0.61, 0.05]
Tone [a] × Sample 1 [b]	–1	[–4, 2]	–0.03	[–0.19, 0.27]
Tone [a] × Sample 2 [b]	0	[–3, 3]	0.12	[–0.13, 0.38]
Random effect				
Residuals (σ^2)	19,323		3.29	
$\tau^2_{0\ participant}$	8,894		0.66	
$\tau^2_{0\ task\ session}$	376		0.03	
$\tau^2_{1\ participant}$	243		0.14	
$\rho_{01\ participant}$	–.03		–.58	
ICC	.33		.15	
$N_{participants}$	462		462	
$N_{task\ sessions}$	7		7	
N_{trials}	71,834		75,840	
	Explained variance			
Marginal R^2	.02		.03	
Conditional R^2	.34		.17	

Note. RT values are in milliseconds. The model for error is based on log-odds, albeit fixed effects are expressed in percentages in this table for interpretation purposes. The row in bold is the fixed estimate from which the by-participant random effects are extracted for the second stage of analysis. For interpretation purposes, only this row is estimated as a direct score (i.e., not scaled or centered) in this model. In the model used for analysis—which also included the random slopes of task session omitted here due to singularity issues—all error estimates are scaled and centered. RT = reaction time; CI = confidence interval; ICC = intra-class correlation.

[a] 0 = tone, 1 = no tone. [b] 0 = out, 1 = in.

*p < .05. **p < .01. ***p < .001.

SUPPLEMENTAL TABLE 7: Linear (RTs) and Logistic (Errors) Mixed Model for Orienting (Attentional Networks)

Component	Model: Orienting RT		Model: Orienting errors	
	Value	**95% CI**	**Value**	**95% CI**
Fixed effect				
Intercept	561***	[541, 580]	3.93***	[3.24, 4.75]
Cue [a]	**33*** **	**[28, 38]**	**0.54*** **	**[0.28, 0.82]**
Sample 1 [b]	–12*	[–23, –2]	–0.15	[–0.48, 0.22]
Sample 2 [b]	–19**	[–32, –6]	–0.43*	[–0.81, –0.01]
Cue [a] × Sample 1 [b]	–1	[–3, 2]	–0.02	[–0.18, 0.23]
Cue [a] × Sample 2 [b]	1	[–2, 4]	0.10	[–0.12, 0.34]
Random effect				
Residuals (σ^2)	19,684		3.29	
$\tau^2_{0\ participant}$	8,495		0.59	
$\tau^2_{0\ task\ session}$	409		0.05	
$\tau^2_{1\ participant}$	92		0.06	
$\tau^2_{1\ task\ session}$	27			
$\rho_{01\ participant}$	.13		–.17	
$\rho_{01\ task\ session}$	.44			
ICC	.32		.16	
$N_{participants}$	462		462	
$N_{task\ sessions}$	7		7	
N_{trials}	142,642		151,680	
	Explained variance			
Marginal R^2	.02		.00	
Conditional R^2	.33		.16	

Note. RT values are in milliseconds. The model for error is based on log-odds, albeit fixed effects are expressed in percentages in this table for interpretation purposes. The row in bold is the fixed estimate from which the by-participant random effects are extracted to be used at the second stage of analysis. For the sake of interpretation, only this row is estimated as a direct score (i.e., not scaled or centered) in this model. In the model used for analysis—which also included the error random slope of task session omitted here due to singularity issues—all error estimates are scaled and centered. RT = reaction time; CI = confidence interval; ICC = intra-class correlation.

[a] 0 = valid, 1 = invalid. [b] 0 = out, 1 = in.

*p < .05. **p < .01. ***p < .001.

SUPPLEMENTAL TABLE 8: Linear (RTs) and Logistic (Errors) Mixed Model for Congruency (Attentional Networks)

Component	Model: Congruency RT		Model: Congruency errors	
	Value	**95% CI**	**Value**	**95% CI**
Fixed effect				
Intercept	560***	[540, 580]	3.60***	[3.01, 4.32]
Flankers [a]	**39*** **	**[33, 45]**	**0.86*** **	**[0.41, 1.36]**
Sample 1 [b]	–13*	[–24, –2]	–0.20	[–0.50, 0.13]
Sample 2 [b]	–20**	[–33, –7]	–0.37	[–0.72, 0.02]
Flankers [a] **× Sample 1** [b]	1	[–2, 4]	0.12	[–0.16, 0.41]
Flankers [a] **× Sample 2** [b]	2	[–1, 6]	0.05	[–0.28, 0.41]
Random effect				
Residuals (σ^2)	19,459		3.29	
$\tau^2_{0\ participant}$	9,090		0.58	
$\tau^2_{0\ task\ session}$	419		0.04	
$\tau^2_{1\ participant}$	429		0.23	
$\tau^2_{1\ task\ session}$	37		0.00	
$\rho_{01\ participant}$	–.25		–.30	
$\rho_{01\ task\ session}$	.02		.03	
ICC	.32		.16	
$N_{participants}$	462		462	
$N_{task\ sessions}$	7		7	
N_{trials}	214,476		227,520	
	Explained variance			
Marginal R^2	.02		.01	
Conditional R^2	.34		.16	

Note. RT values are in milliseconds. The model for error is based on log-odds, albeit fixed effects are expressed in percentages in this table for interpretation purposes. The row in bold is the fixed estimate from which the by-participant random effects are extracted to be used at the second stage of analysis. For the sake of interpretation, only this row is estimated as a direct score (i.e., not scaled or centered) in this model. In the model used for analysis, all error estimates are scaled and centered. RT = reaction time; CI = confidence interval; ICC = intra-class correlation.

[a] 0 = congruent, 1 = incongruent. [b] 0 = out, 1 = in.

*p < .05. **p < .01. ***p < .001.

SUPPLEMENTAL TABLE 9: Logistic Mixed Model for Hits and False Alarms (Executive Vigilance)

Component	Model: Hits		Model: False alarms	
	Value	**95% CI**	**Value**	**95% CI**
Fixed effect				
Intercept	**77.75*****	**[74.87, 80.39]**	**3.31*****	**[2.71, 4.02]**
Sample 1 [a]	–1.94	[–4.00, 0.01]	–0.17	[–0.50, 0.18]
Sample 2 [a]	–0.37	[–2.82, 1.19]	–0.17	[–0.56, 0.28]
Random effect				
Residuals (σ^2)	3.29		3.29	
$\tau^2_{0\ participant}$	0.90		0.73	
$\tau^2_{0\ task\ session}$	0.01		0.04	
ICC	.22		.19	
$N_{participants}$	462		459	
$N_{task\ sessions}$	7		7	
N_{trials}	75,840		65,611	
	Explained variance			
Marginal R^2	.00		.00	
Conditional R^2	.22		.19	

Note. The two models are based on log-odds, although fixed effects are expressed in percentages in this table for interpretation purposes. The row in bold is the fixed estimate from which the by-participant random effects are extracted to be used at the second stage of analysis. Nonbolded fixed estimates are scaled and centered CI = confidence interval; ICC = intra-class correlation.

[a] 0 = out, 1 = in.

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

SUPPLEMENTAL TABLE 10: Logistic Mixed Model for the Slopes of Hits and False Alarms (Executive Vigilance)

Component	Model: Hit slope		Model: False alarm slope	
	Value	95% CI	Value	95% CI
Fixed effect				
Intercept	82.25***	[79.13, 84.98]	4.04***	[3.23, 5.06]
Block [a]	**–1.61*** **	**[–2.29, –0.95]**	**–0.35*** **	**[–0.51, –0.20]**
Sample 1 [b]	–1.50	[–3.33, 0.20]	–0.11	[–0.44, 0.25]
Sample 2 [b]	0.45	[–1.65, 2.36]		
Block [a] × Sample 1 [b]	–0.06	[–0.37, 0.25]	0.02	[–0.07, 0.11]
Block [a] × Sample 2 [b]	–0.30	[–0.69, 0.08]		
Sample 3 [b]			0.22	[–0.42, 0.98]
Block [a] × Sample 3 [b]			0.00	[–0.15, 0.15]
Random effect				
Residuals (σ^2)	3.29		3.29	
$\tau^2_{0\ participant}$	0.82		0.80	
$\tau^2_{0\ task\ session}$	0.04		0.04	
$\tau^2_{1\ participant}$	0.02		0.01	
$\tau^2_{1\ task\ session}$	0.00		0.00	
$\rho_{01\ participant}$	.02		–.30	
$\rho_{01\ task\ session}$	–.81		–1	
ICC	.23		.20	
$N_{participants}$	462		459	
$N_{task\ sessions}$	7		7	
N_{trials}	75,840		65,611	
	Explained variance			
Marginal R^2	.01		.01	
Conditional R^2	.24		.20	

Note. The two models are based on log-odds, albeit fixed effects are expressed in percentages in this table for interpretation purposes. The row in bold is the fixed estimate from which the by-participant random effects are extracted to be used at the second stage of analysis. For the sake of interpretation, only this row is estimated as a direct score (i.e., not scaled or centered) in this model. In the model used for analysis, all estimates are scaled and centered. CI = confidence interval; ICC = intra-class correlation.

[a] Block 1 = 0 and so on until Block 6 = 5. [b] 0 = out, 1 = in.

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

SUPPLEMENTAL TABLE 11: Linear (RTs) and Logistic (Errors) Mixed Model for Mean RT, Standard Deviation of RT, and Lapses (Arousal Vigilance)

Component	Model: *M* RT		Model: *SD* RT		Model: Lapses	
	Value	95% CI	Value	95% CI	Value	95% CI
Fixed effect						
Intercept	**476*****	**[460, 492]**	**66*****	**[61, 71]**	**4.93*****	**[3.82, 6.35]**
Sample 1 [a]	2	[–8, 12]	0	[–3, 4]	–1.94	[–4.00, 0.01]
Sample 2 [a]	–5	[–17, –7]	–5**	[8, –1]	–0.37	[–2.82, 1.90]
Random effect						
Residuals (σ^2)	8,950		2,732		3.29	
$\tau^2_{0\ participant}$	7,431		625		2.81	
$\tau^2_{0\ task\ session}$	227		19		0.03	
ICC	.46		.19		.46	
$N_{participants}$	462		462		462	
$N_{task\ sessions}$	7		7		7	
N_{trials}	75,540		72,380		75,840	
			Explained variance			
Marginal R^2	.00		.01		.01	
Conditional R^2	.46		.20		.47	

Note. RT values are in milliseconds. The standard deviation (SD) of the trial is the SD of that trial, the two previous trials, and the two subsequent trials. The model for lapses is based on log-odds, albeit fixed effects are expressed in percentages in this table to facilitate interpretation. The row in bold is the fixed estimate from which the by-participant random effects are extracted for the second stage of analysis. Nonbolded fixed estimates are scaled and centered. RT = reaction time; CI = confidence interval; ICC = intra-class correlation.

[a] 0 = out, 1 = in.

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

SUPPLEMENTAL TABLE 12: Linear (RTs) and Logistic (Errors) Mixed Model for the Slopes of Mean RT, Standard Deviation of RT, and Lapses (Arousal Vigilance)

Component	Model: *M* RT slope		Model: *SD* RT slope		Model: Lapse slope	
	Value	95% CI	Value	95% CI	Value	95% CI
Fixed effect						
Intercept	469***	[452, 486]	59***	[54, 64]	3.55***	[2.49, 5.03]
Block [a]	**3****	**[1, 5]**	**3*****	**[1, 4]**	**–0.41****	**[0.15, 0.68]**
Sample 1 [b]	0	[–10, 9]	–1	[–4, 2]	0.62*	[0.04, 1.29]
Sample 2 [b]	–3	[–15, 9]	–5*	[–8, –1]		
Block [a] × Sample 1 [b]	1	[0, 2]	0	[–1, 1]	0.84	[–0.23, 2.23]
Block [a] × Sample 2 [b]	–1	[–2, 1]	0	[–1, 1]		
Sample 3 [b]					0.04	[–0.04, 0.13]
Block [a] × Sample 3 [b]					–0.05	[–0.21, 0.12]
Random effect						
Residuals (σ^2)	8,750		2,612		3.29	
$\tau^2_{0\ participant}$	7,063		605		3.42	
$\tau^2_{0\ task\ session}$	285		22		0.10	
$\tau^2_{1\ participant}$	78		47		0.04	
$\tau^2_{1\ task\ session}$	1.70		0.67		0.00	
$\rho_{01\ participant}$	–.03		–.32		–.38	
$\rho_{01\ task\ session}$	–.62		–.38		–.88	
ICC	.47		.23		.49	
$N_{participants}$	462		462		462	
$N_{task\ sessions}$	7		7		7	
N_{trials}	75,540		72,380		75,840	
			Explained variance			
Marginal R^2	.00		.01		.01	
Conditional R^2	.48		.24		.49	

Note. RT values are in milliseconds. The trial standard deviation is the standard deviation of that trial, the two previous trials, and the two subsequent trials. The model for lapses is based on log-odds, albeit fixed effects are expressed in percentages in this table to facilitate interpretation. The row in bold is the fixed estimate from which the by-participant random effects are extracted for the second stage of analysis. For the sake of interpretation, only this row is estimated as a direct score (i.e., not scaled or centered) in this model. In the model used for analysis, all lapse slope estimates are scaled and centered. RT = reaction time; CI = confidence interval; ICC = intra-class correlation.

[a] Block 1 = 0 and so on until Block 6 = 5. [b] 0 = out, 1 = in.

*p < .05. **p < .01. ***p < .001.

SUPPLEMENTAL TABLE 13: Multiple Linear Regressions for RT in Attentional Network Overall Performance as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.00	[–.09, .10]	.01	[–.09, .10]	.01	[–.09, .10]
cBAARS-IV	**.09**	**[–.01, .18]**			.13*	[.03, .24]
aBAARS-IV			**–.03**	**[–.12, .07]**	**–.10**	**[–.20, .01]**
Sex [a]	.16***	[.07, .26]	.14**	[.05, .23]	.16***	[.07, .25]
Age	.01	[–.08, .10]	.00	[–.09, .10]	.00	[–.09, .10]
cBAARS-IV × sex [a]	.01	[–.09, .10]				
cBAARS-IV × age	–.02	[–.11, .07]				
cBAARS-IV × S1 [b]	–.01	[–.16, .15]				
cBAARS-IV × S2 [b]	–.02	[–.17, .13]				
aBAARS-IV × sex [a]			.03	[–.06, .12]	.03	[–.06, .12]
aBAARS-IV × age			–.04	[–.14, .06]	–.03	[–.13, .07]
aBAARS-IV × S1 [b]			–.07	[–.24, .09]	–.06	[–.23, .11]
aBAARS-IV × S2 [b]			–.06	[–.22, .11]	–.05	[–.21, .12]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. RT = reaction time; ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*$p < .05$. **$p < .01$. ***$p < .001$.

SUPPLEMENTAL TABLE 14: Multiple Linear Regressions for Errors in Attentional Network Overall Performance as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.02	[–.07, .11]	–.02	[–.11, .08]	–.02	[–.11, .08]
cBAARS-IV	**.08**	**[–.02, .17]**			.01	[–.10, .12]
aBAARS-IV			**.10***	**[.00, .19]**	**.09**	**[–.02, .20]**
Sex [a]	.07	[–.03, .16]	.07	[–.03, .16]	.07	[–.03, .16]
Age	.01	[–.08, .10]	.01	[–.08, .11]	.01	[–.08, .11]
cBAARS-IV × sex [a]	.12*	[.03, .21]				
cBAARS-IV × age	–.02	[–.11, .07]				
cBAARS-IV × S1 [b]	.09	[–.07, .24]				
cBAARS-IV × S2 [b]	.11	[–.04, .26]				
aBAARS-IV × sex [a]			.08	[–.01, .17]	.08	[–.01, .17]
aBAARS-IV × age			.03	[–.07, .13]	.03	[–.07, .13]
aBAARS-IV × S1 [b]			.05	[–.11, .22]	.05	[–.11, .22]
aBAARS-IV × S2 [b]			.18*	[.01, .34]	.18*	[.01, .34]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. RT = reaction time; ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*$p < .05$. **$p < .01$. ***$p < .001$.

SUPPLEMENTAL TABLE 15: Multiple Linear Regressions for Alerting RT as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	–.01	[–.10, .08]	–.01	[–.10, .09]	–.01	[–.10, .09]
cBAARS-IV	**–.05**	**[–.14, .05]**			–.10	[–.22, .01]
aBAARS-IV			**.07**	**[–.03, .16]**	**.12***	**[.01, .23]**
Sex [a]	.03	[–.06, .13]	.05	[–.04, .15]	.04	[–.06, .13]
Age	–.03	[–.12, .06]	–.02	[–.11, .07]	–.02	[–.11, .07]
cBAARS-IV × sex [a]	–.02	[–.12, .07]				
cBAARS-IV × age	–.01	[–.10, .08]				
cBAARS-IV × S1 [b]	.15	[–.01, .31]				
cBAARS-IV × S2 [b]	.16*	[.01, .31]				
aBAARS-IV × sex [a]			–.04	[–.13, .06]	–.04	[–.13, .06]
aBAARS-IV × age			–.00	[–.10, .10]	–.01	[–.11, .09]
aBAARS-IV × S1 [b]			.07	[–.10, .24]	.06	[–.11, .23]
aBAARS-IV × S2 [b]			.06	[–.11, .22]	.05	[–.12, .22]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. RT = reaction time; ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*p < .05. **p < .01. ***p < .001.

SUPPLEMENTAL TABLE 16: Multiple Linear Regressions for Alerting Errors as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	–.01	[–.10, .08]	.01	[–.09, .10]	.01	[–.09, .10]
cBAARS-IV	**–.00**	**[–.10, .09]**			.03	[–.08, .14]
aBAARS-IV			**–.04**	**[–.13, .06]**	**–.05**	**[–.16, .06]**
Sex [a]	–.01	[–.11, .08]	–.01	[–.10, .08]	–.01	[–.10, .09]
Age	.03	[–.06, .12]	.02	[–.07, .11]	.02	[–.07, .11]
cBAARS-IV × sex [a]	–.07	[–.16, .03]				
cBAARS-IV × age	.02	[–.06, .11]				
cBAARS-IV × S1 [b]	.01	[–.15, .16]				
cBAARS-IV × S2 [b]	–.03	[–.18, .12]				
aBAARS-IV × sex [a]			–.06	[–.15, .03]	–.06	[–.15, .04]
aBAARS-IV × age			–.06	[–.16, .04]	–.06	[–.16, .04]
aBAARS-IV × S1 [b]			.10	[–.06, .27]	.10	[–.06, .27]
aBAARS-IV × S2 [b]			–.02	[–.19, .14]	–.02	[–.19, .15]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

SUPPLEMENTAL TABLE 17: Multiple Linear Regressions for Orienting RT as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.01	[–.08, .11]	.01	[–.08, .10]	.01	[–.08, .10]
cBAARS-IV	**.05**	**[–.05, .15]**			.10	[–.01, .22]
aBAARS-IV			**–.07**	**[–.16, .03]**	**–.12***	**[–.23, –.01]**
Sex [a]	.04	[–.05, .13]	.03	[–.07, .12]	.04	[–.05, .14]
Age	.05	[–.04, .14]	.04	[–.05, .13]	.04	[–.05, .13]
cBAARS-IV × sex [a]	.06	[–.03, .15]				
cBAARS-IV × age	.00	[–.08, .09]				
cBAARS-IV × S1 [b]	–.08	[–.24, .08]				
cBAARS-IV × S2 [b]	–.06	[–.21, .09]				
aBAARS-IV × sex [a]			.02	[–.08, .11]	.02	[–.07, .11]
aBAARS-IV × age			–.05	[–.15, .05]	–.05	[–.15, .05]
aBAARS-IV × S1 [b]			–.06	[–.23, .10]	–.05	[–.22, .11]
aBAARS-IV × S2 [b]			–.08	[–.25, .08]	–.08	[–.24, .09]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. RT = reaction time; ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*$p < .05$. **$p < .01$. ***$p < .001$.

SUPPLEMENTAL TABLE 18: Multiple Linear Regressions for Orienting Errors as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	–.00	[–.10, .09]	–.02	[–.11, .07]	–.02	[–.11, .07]
cBAARS-IV	**.05**	**[–.04, .15]**			.08	[–.03, .19]
aBAARS-IV	.05	[–.05, .14]	**.03**	**[–.06, .12]**	**.04**	**[–.05, .14]**
Sex [a]	.03	[–.06, .12]	.03	[–.06, .12]	.03	[–.06, .12]
Age	.00	[–.09, .10]				
cBAARS-IV × sex [a]	.03	[–.06, .12]				
cBAARS-IV × age	–.01	[–.17, .15]				
cBAARS-IV × S1 [b]	.03	[–.12, .18]				
cBAARS-IV × S2 [b]			–.02	[–.11, .08]	–.06	[–.17, .05]
aBAARS-IV × sex [a]			.03	[–.06, .12]	.03	[–.06, .13]
aBAARS-IV × age			–.05	[–.15, .05]	–.05	[–.15, .05]
aBAARS-IV × S1 [b]			.09	[–.08, .25]	.09	[–.07, .26]
aBAARS-IV × S2 [b]			.18*	[.01, .34]	.18*	[.02, .35]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*$p < .05$. **$p < .01$. ***$p < .001$.

SUPPLEMENTAL TABLE 19: Multiple Linear Regressions for Congruency RT as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.01	[–.09, .10]	–.01	[–.10, .08]	–.01	[–.10, .08]
cBAARS-IV	**.11***	**[.01, .20]**			.04	[–.07, .15]
aBAARS-IV			**.14****	**[.04, .23]**	**.12***	**[.01, .22]**
Sex [a]	–.04	[–.13, .05]	–.05	[–.14, .04]	–.04	[–.14, .05]
Age	.01	[–.08, .10]	.01	[–.08, .11]	.01	[–.08, .11]
cBAARS-IV × sex [a]	.02	[–.07, .11]				
cBAARS-IV × age	.08	[–.01, .17]				
cBAARS-IV × S1 [b]	–.07	[–.23, .08]				
cBAARS-IV × S2 [b]	–.08	[–.23, .07]				
aBAARS-IV × sex [a]			–.01	[–.10, .08]	–.01	[–.10, .09]
aBAARS-IV × age			.10	[–.00, .20]	.10*	[.00, .20]
aBAARS-IV × S1 [b]			.06	[–.10, .23]	.07	[–.10, .23]
aBAARS-IV × S2 [b]			.11	[–.05, .28]	.12	[–.05, .28]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. RT = reaction time; ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*$p < .05$. **$p < .01$. ***$p < .001$.

SUPPLEMENTAL TABLE 20: Multiple Linear Regressions for Congruency Errors as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.01	[–.08, .10]	–.01	[–.10, .08]	–.01	[–.10, .08]
cBAARS-IV	**.10***	**[.00, .19]**			.07	[–.04, .18]
aBAARS-IV			**.07**	**[–.03, .16]**	**.03**	**[–.08, .14]**
Sex [a]	–.09*	[–.19, –.00]	–.11*	[–.20, –.02]	–.10*	[–.19, –.00]
Age	–.07	[–.17, .02]	–.08	[–.17, .01]	–.08	[–.17, .01]
cBAARS-IV × sex [a]	.01	[–.08, .11]				
cBAARS-IV × age	.05	[–.04, .13]				
cBAARS-IV × S1 [b]	–.07	[–.23, .08]				
cBAARS-IV × S2 [b]	–.12	[–.27, .03]				
aBAARS-IV × sex [a]			–.01	[–.10, .08]	–.01	[–.10, .09]
aBAARS-IV × age			.07	[–.03, .17]	.08	[–.02, .18]
aBAARS-IV × S1 [b]			.11	[–.05, .28]	.12	[–.05, .29]
aBAARS-IV × S2 [b]			.13	[–.04, .29]	.13	[–.03, .30]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*$p < .05$. **$p < .01$. ***$p < .001$.

SUPPLEMENTAL TABLE 21: Multiple Linear Regressions for Congruency Errors as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	–.00	[–.09, .09]	.01	[–.08, .10]	.01	[–.08, .10]
cBAARS-IV	**–.12***	**[–.21, –.02]**			–.07	[–.18, .04]
aBAARS-IV			**–.13***	**[–.22, –.03]**	**–.09**	**[–.20, .02]**
Sex [a]	.02	[–.07, .12]	.04	[–.05, .13]	.03	[–.07, .12]
Age	.08	[–.02, .17]	.08	[–.01, .17]	.08	[–.01, .17]
cBAARS-IV × sex [a]	–.01	[–.10, .08]				
cBAARS-IV × age	–.01	[–.09, .08]				
cBAARS-IV × S1 [b]	.10	[–.05, .26]				
cBAARS-IV × S2 [b]	.06	[–.09, .21]				
aBAARS-IV × sex [a]			–.06	[–.16, .03]	–.07	[–.16, .03]
aBAARS-IV × age			–.01	[–.11, .09]	–.02	[–.12, .08]
aBAARS-IV × S1 [b]			–.06	[–.22, .11]	–.07	[–.23, .10]
aBAARS-IV × S2 [b]			–.13	[–.29, .04]	–.13	[–.30, .03]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*$p < .05$. **$p < .01$. ***$p < .001$.

SUPPLEMENTAL TABLE 22: Multiple Linear Regressions for False Alarms as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.01	[–.09, .10]	–.01	[–.10, .09]	–.01	[–.10, .09]
cBAARS-IV	**–.06**	**[–.15, .04]**			–.06	[–.18, .05]
aBAARS-IV			**–.02**	**[–.11, .07]**	**.01**	**[–.10, .12]**
Sex [a]	.10*	[.00, .19]	.11*	[.02, .20]	.10*	[.01, .20]
Age	.06	[–.03, .15]	.07	[–.03, .16]	.07	[–.03, .16]
cBAARS-IV × sex [a]	.06	[–.03, .16]				
cBAARS-IV × age	–.04	[–.12, .05]				
cBAARS-IV × S1 [b]	.19*	[.04, .35]				
cBAARS-IV × S2 [b]	.21**	[.06, .36]				
aBAARS-IV × sex [a]			.00	[–.09, .10]	.00	[–.09, .10]
aBAARS-IV × age			.06	[–.04, .16]	.06	[–.04, .16]
aBAARS-IV × S1 [b]			.07	[–.10, .23]	.06	[–.11, .23]
aBAARS-IV × S2 [b]			.09	[–.07, .26]	.09	[–.08, .25]

Note. *N* = 459. The predictor of interest for each model, which names the model, is in bold. ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*$p < .05$. **$p < .01$. ***$p < .001$.

SUPPLEMENTAL TABLE 23: Multiple Linear Regressions for Hit Slope as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	–.01	[–.11, .08]	–.01	[–.10, .09]	–.01	[–.10, .09]
cBAARS-IV	**–.03**	**[–.12, .07]**			–.01	[–.12, .10]
aBAARS-IV			**–.03**	**[–.12, .06]**	**–.02**	**[–.13, .08]**
Sex [a]	.08	[–.01, .18]	.09	[–.01, .18]	.08	[–.01, .18]
Age	.07	[–.02, .17]	.08	[–.01, .17]	.08	[–.01, .17]
cBAARS-IV × sex [a]	–.03	[–.13, .06]				
cBAARS-IV × age	–.01	[–.10, .08]				
cBAARS-IV × S1 [b]	.03	[–.12, .19]				
cBAARS-IV × S2 [b]	.07	[–.08, .22]				
aBAARS-IV × sex [a]			–.07	[–.17, .02]	–.07	[–.17, .02]
aBAARS-IV × age			–.03	[–.13, .07]	–.03	[–.14, .07]
aBAARS-IV × S1 [b]			–.08	[–.25, .09]	–.08	[–.25, .09]
aBAARS-IV × S2 [b]			–.08	[–.24, .09]	–.08	[–.24, .09]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

SUPPLEMENTAL TABLE 24: Multiple Linear Regressions for False Alarm Slope as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	–.01	[–.10, .09]	.00	[–.09, .10]	.00	[–.09, .10]
cBAARS-IV	**.10***	**[.00, .19]**			.10	[–.02, .21]
aBAARS-IV			**.07**	**[–.03, .16]**	**.02**	**[–.09, .13]**
Sex [a]	.01	[–.08, .11]	.01	[–.09, .10]	.02	[–.07, .11]
Age	.04	[–.06, .13]	.04	[–.05, .13]	.04	[–.05, .13]
cBAARS-IV × sex [a]	–.01	[–.11, .08]				
cBAARS-IV × age	.06	[–.03, .15]				
cBAARS-IV × S1 [b]	.14	[–.02, .30]				
cBAARS-IV × S2 [b]	.15*	[.00, .30]				
aBAARS-IV × sex [a]			–.08	[–.17, .02]	–.08	[–.17, .02]
aBAARS-IV × age			.01	[–.10, .11]	.01	[–.09, .11]
aBAARS-IV × S1 [b]			–.00	[–.17, .16]	.01	[–.16, .17]
aBAARS-IV × S2 [b]			–.08	[–.24, .09]	–.07	[–.24, .10]

Note. *N* = 459. The predictor of interest for each model, which names the model, is in bold. ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*p < .05. **p < .01. ***p < .001.

SUPPLEMENTAL TABLE 25: Multiple Linear Regressions for Mean RT in Arousal Vigilance as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.00	[–.09, .09]	.00	[–.09, .09]	.00	[–.09, .09]
cBAARS-IV	**.19*****	**[.10, .29]**			.14*	[.03, .25]
aBAARS-IV			**.16*****	**[.07, .26]**	**.09**	**[–.02, .20]**
Sex [a]	.08	[–.01, .18]	.06	[–.03, .15]	.08	[–.01, .17]
Age	.02	[–.07, .11]	.02	[–.07, .11]	.02	[–.07, .11]
cBAARS-IV × sex [a]	.01	[–.08, .11]				
cBAARS-IV × age	–.04	[–.12, .05]				
cBAARS-IV × S1 [b]	–.08	[–.23, .08]				
cBAARS-IV × S2 [b]	–.05	[–.19, .10]				
aBAARS-IV × sex [a]			.04	[–.05, .13]	.04	[–.05, .13]
aBAARS-IV × age			–.06	[–.16, .04]	–.05	[–.15, .05]
aBAARS-IV × S1 [b]			–.11	[–.28, .05]	–.10	[–.26, .07]
aBAARS-IV × S2 [b]			–.06	[–.22, .11]	–.05	[–.21, .12]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. RT = reaction time; ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

SUPPLEMENTAL TABLE 26: Multiple Linear Regressions for the Standard Deviation of the RT in Arousal Vigilance as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.01	[–.08, .10]	–.01	[–.10, .09]	–.01	[–.10, .09]
cBAARS-IV	**.17*****	**[.07, .26]**			.09	[–.02, .20]
aBAARS-IV			**.18*****	**[.09, .28]**	**.14***	**[.03, .25]**
Sex [a]	.06	[–.03, .15]	.05	[–.04, .14]	.06	[–.03, .15]
Age	–.05	[–.14, .04]	–.05	[–.14, .04]	–.05	[–.14, .04]
cBAARS-IV × sex [a]	.05	[–.05, .14]				
cBAARS-IV × age	–.01	[–.09, .08]				
cBAARS-IV × S1 [b]	.07	[–.08, .23]				
cBAARS-IV × S2 [b]	.07	[–.08, .22]				
aBAARS-IV × sex [a]			.03	[–.06, .13]	.04	[–.06, .13]
aBAARS-IV × age			.00	[–.10, .10]	.01	[–.09, .11]
aBAARS-IV × S1 [b]			.03	[–.14, .19]	.03	[–.13, .20]
aBAARS-IV × S2 [b]			.07	[–.09, .24]	.08	[–.08, .25]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. RT = reaction time; ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

SUPPLEMENTAL TABLE 27: Multiple Linear Regressions for Lapses as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.00	[–.09, .09]	.00	[–.09, .10]	.00	[–.09, .10]
cBAARS-IV	**.18*****	**[.08, .27]**			.10	[–.01, .21]
aBAARS-IV			**.18*****	**[.09, .27]**	**.13***	**[.02, .24]**
Sex [a]	.09	[–.01, .18]	.07	[–.02, .16]	.09	[–.01, .18]
Age	–.03	[–.12, .06]	–.03	[–.12, .06]	–.03	[–.12, .06]
cBAARS-IV × sex [a]	.00	[–.09, .10]				
cBAARS-IV × age	–.04	[–.13, .05]				
cBAARS-IV × S1 [b]	–.08	[–.23, .08]				
cBAARS-IV × S2 [b]	–.06	[–.21, .09]				
aBAARS-IV × sex [a]			.05	[–.04, .14]	.05	[–.04, .14]
aBAARS-IV × age			–.07	[–.17, .03]	–.06	[–.16, .04]
aBAARS-IV × S1 [b]			–.12	[–.28, .05]	–.11	[–.27, .06]
aBAARS-IV × S2 [b]			–.06	[–.22, .10]	–.05	[–.22, .11]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

SUPPLEMENTAL TABLE 28: Multiple Linear Regressions for the Slope of Mean RT in Arousal Vigilance as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.02	[–.08, .11]	–.00	[–.10, .09]	–.00	[–.10, .09]
cBAARS-IV	**.08**	**[–.01, .18]**			.03	[–.08, .14]
aBAARS-IV			**.09**	**[–.00, .18]**	**.07**	**[–.04, .18]**
Sex [a]	–.01	[–.10, .09]	–.00	[–.10, .09]	.00	[–.09, .10]
Age	–.05	[–.14, .04]	–.05	[–.14, .04]	–.05	[–.14, .04]
cBAARS-IV × sex [a]	.04	[–.05, .13]				
cBAARS-IV × age	–.03	[–.12, .06]				
cBAARS-IV × S1 [b]	.19*	[.04, .34]				
cBAARS-IV × S2 [b]	.07	[–.08, .22]				
aBAARS-IV × sex [a]			.01	[–.09, .10]	.01	[–.08, .10]
aBAARS-IV × age			–.02	[–.12, .08]	–.01	[–.11, .09]
aBAARS-IV × S1 [b]			.13	[–.03, .30]	.14	[–.03, .31]
aBAARS-IV × S2 [b]			.09	[–.08, .26]	.09	[–.07, .26]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. RT = reaction time; ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*p < .05. **p < .01. ***p < .001.

SUPPLEMENTAL TABLE 29: Multiple Linear Regressions for the Slope of the Standard Deviation of the RT in Arousal Vigilance as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	–.00	[–.10, .09]	–.01	[–.10, .09]	–.01	[–.10, .09]
cBAARS-IV	**.03**	**[–.07, .12]**			–.00	[–.12, .11]
aBAARS-IV			**.06**	**[–.04, .15]**	**.06**	**[–.05, .17]**
Sex [a]	–.07	[–.16, .03]	–.07	[–.16, .03]	–.07	[–.16, .03]
Age	–.01	[–.10, .08]	–.01	[–.10, .09]	–.01	[–.10, .09]
cBAARS-IV × sex [a]	–.03	[–.13, .06]				
cBAARS-IV × age	–.00	[–.09, .08]				
cBAARS-IV × S1 [b]	.09	[–.07, .24]				
cBAARS-IV × S2 [b]	.01	[–.14, .17]				
aBAARS-IV × sex [a]			–.04	[–.13, .05]	–.04	[–.13, .05]
aBAARS-IV × age			.03	[–.07, .13]	.03	[–.07, .13]
aBAARS-IV × S1 [b]			.01	[–.16, .18]	.01	[–.16, .18]
aBAARS-IV × S2 [b]			.02	[–.15, .19]	.02	[–.15, .19]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. RT = reaction time; ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

SUPPLEMENTAL TABLE 30: Multiple Linear Regressions for the Slope of Lapses as a Function of ADHD Symptoms

Predictor	Model 1: Childhood		Model 2: Adulthood		Model 3: Late onset	
	β	CI	β	CI	B	CI
Intercept	.01	[–.08, .11]	–.01	[–.10, .09]	–.01	[–.10, .09]
cBAARS-IV	**–.01**	**[–.11, .08]**			–.04	[–.16, .07]
aBAARS-IV			**.02**	**[–.08, .11]**	**.04**	**[–.07, .15]**
Sex [a]	–.06	[–.16, .03]	–.05	[–.15, .04]	–.06	[–.15, .03]
Age	–.03	[–.12, .06]	–.03	[–.12, .06]	–.03	[–.12, .06]
cBAARS-IV × sex [a]	.03	[–.06, .13]				
cBAARS-IV × age	–.01	[–.10, .08]				
cBAARS-IV × S1 [b]	.18*	[.02, .33]				
cBAARS-IV × S2 [b]	.08	[–.07, .23]				
aBAARS-IV × sex [a]			–.01	[–.11, .08]	–.01	[–.11, .08]
aBAARS-IV × age			.04	[–.06, .14]	.04	[–.06, .14]
aBAARS-IV × S1 [b]			.17*	[.00, .34]	.17	[–.00, .33]
aBAARS-IV × S2 [b]			.13	[–.03, .30]	.13	[–.04, .30]

Note. *N* = 462. The predictor of interest for each model, which names the model, is in bold. ADHD = attention-deficit/hyperactivity disorder; CI = confidence interval; cBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Childhood Symptoms; aBAARS-IV = Barkley Adult ADHD Rating Scale-IV: Current Symptoms; S = Sample.

[a] 0 = man, 1 = woman. [b] 0 = out, 1 = in.

*p < .05. **p < .01. ***p < .001.

CHAPTER V:
POTENTIAL AROUSAL MEDIATORS IN ADHD INTERVENTIONS

Study 5

The Effects of Nonpharmacological Interventions for ADHD on Indices of Autonomic Functioning: Protocol for a Systematic Review and Meta-Analysis

This work has been registered as:

Coll-Martín, T., Evangelista, J., Hussain, Z., Cortese, S., Westwood, S., Bellato, A. (2022). The effects of non-pharmacological interventions for ADHD on indices of autonomic functioning: Systematic review and meta-analysis. *PROSPERO*. https://www.crd.york.ac.uk/prospero/display_record.php?RecordID=372965

This chapter also includes preliminary results of the final study, whose corresponding manuscript will be presented in the same order of authorship, along with Amparo Diaz-Román and Iman Idrees as second and third authors.

Abstract

Arousal dysregulation is a common feature of ADHD and has been proposed as one of the mechanisms underlying the disorder. This alteration has been observed in autonomic nervous system functioning, typically in the form of hypo-arousal. Although medication, especially stimulants, tends to “normalize” this dysregulation, the efficacy of other ADHD treatments remains unclear. In this paper, we present the protocol of a systematic review and meta-analysis to evaluate the effects of nonpharmacological interventions on autonomic functioning in people with ADHD (PROSPERO: CRD42022372965). For this review, we will include any kind of nonpharmacological intervention (e.g., psychosocial, neuropsychological, dietary) measuring indices of autonomic domains, namely cardiac, electrodermal, and pupil activity, among others. Two independent reviewers will carry out the literature search procedure, extraction, and coding of the data of interest. Preliminary results have so far identified 12 articles meeting the criteria. Although most of the studies were randomized controlled trials, their small sample size limits the statistical power. Moreover, the effects of the interventions on indices of autonomic arousal were rather mixed. Taken together, while further studies with larger samples are needed, it might be that nonpharmacological interventions do not compensate for ADHD-associated hypo-arousal to the same extent as medication. This could account for the limited effect on core symptoms by nonpharmacological interventions. Moreover, this would underscore the importance of developing nonpharmacological interventions aimed at targeting arousal dysregulation.

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental condition characterized by age-inappropriate, persistent, and impairing levels of inattention and/or hyperactivity–impulsivity (American Psychiatric Association [APA], 2013). Prevalent in about 5% of children and 2.5% of adults (Polanczyk et al., 2007; Simon et al., 2009), this disorder is a risk factor for several negative outcomes, including educational underachievement, difficulties with employment, and criminality (Faraone et al., 2015; Fletcher, 2014; Loe & Feldman, 2007). Against this backdrop, identifying the mechanisms underlying ADHD symptoms and mediating their response to interventions is crucial to enhancing the approach to the disorder (Castellanos & Tannock, 2002; Sonuga-Barke et al., 2023).

Despite the recognized heterogeneity in the neurocognitive pathways of ADHD (Fair et al., 2012; Luo et al., 2019), accounts focused on arousal dysregulation are highly influential in this literature (Geissler et al., 2014; Halperin & Schulz, 2006; Sergeant, 2005; Sikström & Söderlund, 2007). In this vein, meta-analyses of cognitive performance found that intrasubject intertrial variability, often interpreted as a marker of impaired arousal regulation (e.g., Sergeant, 2005), is higher in individuals with ADHD (Bella-Fernández et al., 2023; Kofler et al., 2013) or with higher ADHD symptoms (Coll-Martín, Sonuga-Barke, et al., 2023). While informative, these behavioural findings only provide an indirect or partial measure of arousal that can be biased by general cognitive and motor processes affecting task performance. Indeed, arousal involves the physiological mechanisms that characterize alertness, wakefulness, and reactivity to the environment (Lacey, 1967). This is governed by the interplay between the central and peripheral nervous systems.

The autonomic nervous system, a component of the peripheral nervous system with a fundamental role in arousal regulation, comprises two main branches that exert opposite

but coordinated forces: the sympathetic nervous system and the parasympathetic nervous system. The former, in charge of mobilizing resources for fight-or-flight responses, involves upregulating peripheral indices of autonomic arousal (e.g., increased heart rate and skin conductance level). In contrast, the parasympathetic nervous system, responsible for relaxation and preservation of energetic resources, leads to downregulation of arousal levels (e.g., deceleration and increased variability in heart rate).

Regarding ADHD, Bellato et al. (2020) conducted a systematic review of physiological indices of autonomic functioning. They found that individuals with ADHD exhibited altered autonomic levels, more often in the form of hypo-arousal than hyper-arousal. Consistently, it has been hypothesized that attention impairments in ADHD are direct manifestations of this low level of arousal, while hyperactivity-impulsivity behaviours are an autoregulatory attempt of the organism to create a stimulating environment in order to stabilize arousal (Geissler et al., 2014). Alternatively, inattention has also been viewed as a compensatory strategy to upregulate arousal (Sonuga-Barke, Wiersema, et al., 2010).

Taken together, autonomic arousal processes may constitute key targets or monitoring variables in interventions for ADHD. In this sense, a recent meta-analysis found that medication, specially stimulants, “normalized” the activity of the autonomic nervous system in people with ADHD (Idrees et al., 2023). Concretely, stimulants and nonstimulants increased heart rate and blood pressure, and greater electrodermal activity and pupil diameter were observed after stimulant medication. Indeed, this pattern is consistent with an upregulatory effect of medication on the hypo-arousal typically observed in ADHD (Bellato et al., 2020). Given that pharmacological interventions substantially improve ADHD symptoms (Cortese, 2020), changes in autonomic functioning may underlie this effect and the enhancement of neurocognitive outcomes (Coghill, Seth, Pedroso, et al., 2014).

In contrast to medication, meta-analyses suggest that the efficacy of nonpharmacological interventions on core ADHD symptoms is limited, although they can improve some co-occurring outcomes (Faraone et al., 2021; Sibley et al., 2023). In this sense, although behavioural interventions (e.g., parent training) have not shown substantial effects on ADHD symptoms, they improve behavioural problems (i.e., symptoms of oppositional defiant disorder and/or conduct disorder) and parenting (Daley et al., 2014; Dekkers et al., 2022). Furthermore, while computerized cognitive training appears to produce practice-like gains restricted to the trained cognitive domain, the efficacy on core symptoms is minimal, with only minor reductions in inattention (Westwood et al., 2023). Regarding neurotherapeutics, neurofeedback has a small efficacy on inattention (Riesco-Matías et al., 2021), while brain stimulation (i.e., repetitive transcranial magnetic stimulation and transcranial direct current stimulation) could improve some cognitive functions but has very limited evidence of reducing ADHD symptoms (Westwood et al., 2021). Moreover, meta-analyses on meditation and physical exercise have failed to find reliable evidence of efficacy on ADHD symptoms, although the latter treatment led to a reduction in anxiety and depression (Zang, 2019; Zhang et al., 2018). As for dietary interventions such as omega-3 fatty acids supplementation and restriction of artificial food colors, there is evidence of small-to-moderate improvements in ADHD symptoms (J. P. C. Chang et al., 2018; Nigg et al., 2012).

Examining the effect of nonpharmacological interventions for ADHD on indices of autonomic functioning may help to elucidate the potential of such interventions on arousal regulation and how this relates to changes in symptoms and other relevant outcomes. Some previous works have shed light on this research question through single empirical studies (e.g., Bayo-Tallón, 2020; Beauchaine et al., 2015; Ludyga et al., 2020). However, to the best of our knowledge, the present study constitutes the first systematic review with meta-analysis aimed at evaluating this literature. As far as improvements in ADHD

symptoms are related to changes in autonomic arousal, we expect that nonpharmacological interventions will have a rather limited efficacy on the upregulation and normalization of autonomic indices. Ultimately, comparing the autonomic effects between different nonpharmacological interventions may help to identify promising treatments worth further study owing to their theoretical plausibility.

Methods

Eligibility Criteria

Studies will need to meet the criteria outlined below to be included in this review.

Study Design

Empirical studies where indices of autonomic arousal were collected before and after the initiation of a nonpharmacological intervention. Previous systematic or narrative reviews will not be included; however, reference lists will be searched to identify suitable studies that meet inclusion criteria. Case studies will not be included.

Participants/Population

Children, adolescents, and adults, who meet *Diagnostic and Statistical Manual of Mental Disorders* (DSM) or *International Classification of Diseases* (ICD) diagnostic criteria for ADHD or exceed cut-off points on rating scales with validated measures of ADHD according to the European ADHD Guidelines Group.

Intervention

Any type of nonpharmacological intervention.

Comparators

No restrictions.

Main Outcome

Any measures of autonomic arousal, including (but not limited to) heart rate, heart rate variability, electrodermal activity, and pupil dilation. Behavioural measures of arousal (i.e., indices of task performance) will not be included.

Search Strategy

We will search electronic bibliographic databases (PubMed, Embase, PsycINFO, and Web of Science) and preprint servers (medRxiv, bioRxiv, and PsyArXiv) for studies suitable to be included in this review as well as reference lists of eligible studies and recent review articles. The search will include full journal articles accepted for publication. If these are not available, we will contact the corresponding author of abstracts (e.g., conference proceedings) deemed potentially eligible to request additional information on study eligibility and, if needed, data for the meta-analysis. No language, type of document, or time restrictions will be applied.

The search strategy will include terms associated with the following domains:

- Autonomic arousal.
- ADHD.

The full search strategy will be attached to the final manuscript as a separate file.

Data Extraction

Titles and abstracts of initially retrieved studies and from additional sources will be independently screened by two members of the research team to identify those that could meet inclusion criteria. Full texts of potentially eligible studies will be assessed by two researchers against the inclusion criteria. Any disagreement between the two authors will be assessed by the senior author and disagreements will be resolved by discussion.

A standardized form (Excel spreadsheet) will be used to extract data from the included studies, including publication details (year, institution), study design, sample demographics/clinical characteristics (age, sex, ethnic background, intellectual functioning, co-occurring conditions), details of intervention, information on measures of autonomic functioning and raw data (mean and SD for pre- and post-intervention period, or effect size, if available). Data not available from the manuscript will be requested from the corresponding authors.

Quality Assessment

Based on the type of studies included, the authors will identify the most suitable tools (e.g., Newcastle Ottawa Scale, revised Cochrane risk of bias tool for randomized trials [RoB 2]) to assess the risk of bias. This will be done independently by two authors. Unresolved classification of studies will be arbitrated by the senior author.

Data Synthesis

A narrative synthesis of the findings will be presented to describe the studies included in the review, for each measure of interest. If applicable, meta-analyses will be performed on the outcome measures (standardized mean difference or correlation coefficients) using random effect models, and meta-regressions or subgroup analyses will be performed to investigate the potential confounding effect of variables such as developmental stage. To assess the heterogeneity of effect sizes, we will use the Q statistics and I^2 index, which estimates the percentage of variation among effect sizes that can be attributed to true heterogeneity. Publication bias will be assessed and, when detected, trim and fill analyses will be performed. Analyses will be performed using R.

Analysis of Subgroups or Subsets

If applicable, we will conduct analyses on the primary outcomes in participants sub-grouped by age (e.g., children, adolescents, adults) or type of nonpharmacological

intervention. Moreover, we will investigate (narratively) whether changes in autonomic arousal associated with nonpharmacological interventions, are comparable to those associated with pharmacological interventions or placebo, if applicable.

Results (Preliminary)

Of the 7,180 non-duplicate records found, 7,095 were excluded during title and abstract screening, leaving 85 for full-text eligibility assessment. At the time of writing this thesis, 12 of the 85 articles have been included with the agreement of two independent researchers of the team. **Table 1** shows the characteristics of the studies based on the criteria of a team member (TC-M; therefore, the final version of the manuscript may include changes in this regard). The different studies covered age groups ranging from preschool to young adults. Interventions were highly diverse, encompassing psychosocial (behavioural therapy, meditation), neuropsychological (biofeedback, cognitive training), dietary (omega-3 supplementation), physiotherapeutic (manual therapy), and physical exercise. Although this favors the representativeness of the review, the heterogeneity due to the type of intervention and its implementation makes the application of a meta-analysis unadvisable.

Crucially, although most of the studies (8 of 12) were RCTs, which may favour the internal validity of their conclusions, their small sample size (*Mdn* = 24.45 per group) limited their sensitivity to detect true effects. Indeed, the corresponding statistical power of these studies to detect a medium effect size (i.e., $d_s = 0.5$) was less than 50%, assuming an alpha level of .05. This hinders the interpretation of null differential effects of intervention (i.e., Condition × Time), which were predominant in the studies of the present systematic review. Moreover, the effects of the interventions on indices of autonomic arousal were rather mixed. For example, while some studies found an increase in skin conductance (Beauchaine et al., 2015; Johnstone et al., 2010), an index of the

sympathetic nervous system leading to higher arousal, another study observed somewhat the opposite pattern (Gabriely et al., 2020). Furthermore, studies with multiple indices of cardiac function found that the intervention affected indices associated with high and low arousal levels in the same direction (Bell et al., 2018; Buchhorn et al., 2018).

Taken together, although further studies with larger samples are needed, it might be that nonpharmacological interventions do not have as direct an effect on compensating for ADHD-associated hypo-arousal as medication does. This could explain the lack of effects on core symptoms by nonpharmacological interventions. Also, this would highlight the importance of designing and studying nonpharmacological interventions aimed at targeting arousal dysregulation.

Table 1

Summary of studies included in the systematic review

Authors (year)	Study design	Age group	Sample	Intervention type	ANS domain (measure)	Main findings
Bayo-Tallón (2020)	RCT	Children	$N = 48$ ($n_1 = 24$, $n_2 = 24$)	Manual cranial therapy versus massage (both as an add-on to methylphenidate)	Respiratory rate, heart rate, blood pressure (SBP and DBP), and heart rate variability (RMSSD, LF, HF, LF/HF)	Manual cranial therapy versus massage: Greater decrease in DPB (short, medium, and long term), greater increase in RMSSD (short term), greater decrease in LF (medium term) greater increase in HF (medium term), and greater decrease in LF/HF (medium and long term). No other intervention effect was significantly different between both groups.
Bayo-Tallón et al. (2020)	RCT	Children	$N = 8$ ($n_{intervention} = 4$, $n_{control} = 4$)	Manual cranial therapy as an add-on to usual multimodal treatment versus usual multimodal treatment alone	Heart rate variability (RMSSD, LF, HF, LF/HF)	No intervention effects were significantly different between both groups for any measure.
Beauchaine et al. (2015) [a]	RCT	Preschoolers	$N = 140$ ($n_{intervention} = 49$, $n_{waitlist} = 50$, $n_{controls} = 41$)	Behavioural parent- and child-training: the Incredible Years	Electrodermal activity (ns-SCR)	No significant differences between the post-treatment intervention group and the pre-treatment waitlist group. A greater increase in post-intervention electrodermal activity (short-term and follow-up) in the intervention group compared to typically developing controls, who did not receive the intervention. There were no differences between the two groups in the pre-intervention measures.

Authors (year)	Study design	Age group	Sample	Intervention type	ANS domain (measure)	Main findings
Bell et al. (2018) [a]	One-group pre–post design [b]	Preschoolers	*N* = 99	Behavioural parent- and child-training: the Incredible Years	Heart rate variability (RSA) and cardiac function (PEP), both at rest and in response to incentives	There was a significant increment in RSA at rest and a significant decrement in RSA reactivity. Only the increment in resting RSA and changes in PEP reactivity were mediated by reductions in negative parenting.
Buchhorn et al. (2018)	One-group pre–post design	Children and adolescents	*N* = 18	Omega-3 supplementation	Heart rate (mean R-R intervals) and heart rate variability (RMSSD, HF)	The three indices significantly increased after the intervention
S. C. L. Cohen et al. (2018)	RCT	Preschoolers	*N* = 23 ($n_{intervention}$ = 12, $n_{waitlist}$ = 11)	Yoga	Heart rate variability (RMSSD, SDNN, HF, LF/HF)	After the intervention in the first group, no significant differences between groups controlling for baseline scores. There were no significant differences between pre- and post-intervention measures
Eisenberg et al. (2004)	One-group pre–post design	Children and adolescents	*N* = 19	Biofeedback	Heart rate variability (calculated as the beat-to-beat variation)	Not reported (request pending)
Gabriely et al. (2020)	RCT	Young adults	*N* = 71 ($n_{mindfulness}$ = 27, $n_{breathing}$ = 35, $n_{waitlist}$ = 9)	Mindfulness-based stress reduction versus device-guided breathing	Breathing rate, heart rate, electrodermal activity (SCL), and blood pressure (SBP, DPB)	After the intervention, there was a specific decrease in breathing rate for the device-guided breathing group and a specific increase in electrodermal activity for the control group (both as effects on interaction and as simple pre-post contrasts). There were no significant intervention effects for the other measures.

Authors (year)	Study design	Age group	Sample	Intervention type	ANS domain (measure)	Main findings
Johnstone et al. (2010)	RCT	Children and adolescents	N = 29 ($n_{intervention}$ = 15, $n_{control}$ = 14)	Computer-based cognitive training	Electrodermal activity (SCL during a task, measured as eight 30-s intervals)	The Interval × Condition × Time interaction did not reach significance. However, by looking at the plot, there is probably a significantly greater pre-post increment in SCL for the intervention than for the control group.
Robe & Dobrean (2022)	RCT	Children and adolescents	N = 70 ($n_{intervention}$ = 35, $n_{control}$ = 35)	Mindfulness (single session)	Heart rate variability (RMSSD, HF)	There was a significant increase in RMSSD values from pre- to post-treatment only in the intervention group. However, none of the two Condition × Time interactions were significant.
Vitiello et al. (2012)	RCT	Children	N = 288 ($n_{multimodal}$ = 144, $n_{medication}$ = 144) [c]	Multimodal intervention (i.e., combined medication and behavioural therapy) versus medication alone	Heart rate and blood pressure (SBP and DBP)	After the intervention, the Condition × Time interaction was not significant for any of the measures. Regarding the nine follow-up evaluations for each of the three measures, only one of them had a different heart rate observed between the two groups.
Yu et al. (2020)	RCS	Children and adolescents	N = 30	Acute aerobic exercise versus control (i.e., watch a video)	Heart rate variability (LF, HF, LF/HF)	There was a Condition × Time interaction for LF and HF, but not for LF/HF. After the intervention, LF was lower for the exercise group than for the control group. Participants in the exercise group, but not those in the control group, reduced their HF after the intervention.

Note. ANS = autonomic nervous system; RCT = randomized controlled trial; RCS = randomized crossover study; SBP = systolic blood pressure; DBP = diastolic blood pressure; RMSSD = root mean square of successive differences; LF = low frequency; HF = high frequency; ns-SCR = nonspecific skin conductance response; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period.

[a] Same data set, although Bell et al. (2018) did not include the control group. [b] Although the RCT included an immediate intervention group and a waiting list group that subsequently received the intervention, the analyses reported in the article did not make this distinction. [c] The whole study sample (*N* = 579) included two other groups: behavioural therapy alone, and usual community treatment. Since the majority of participants in the latter group received stimulant medication, both groups are not presented in this table, as their comparisons are not informative.

CHAPTER VI: GENERAL DISCUSSION

Summary of findings

The general aim of this dissertation was to understand the executive and arousal attentional alterations underlying ADHD symptoms, grounded on dimensional and neurodevelopmental perspectives from a broader biopsychosocial framework, and ultimately consider implications for translational interventions. Five studies, grouped in three chapters, were conducted to address distinct aspects of this general aim.

In **Study 1** (**Chapter III**), we sought to establish a neurocognitive behavioural task capable of (a) feasibly collecting large samples from different contexts, (b) measuring relevant indices of attentional functioning with sufficient reliability, and (c) conceptually and empirically differentiating between executive and arousal measures. Our study showed that the ANTI-Vea-UGR platform can make the assessment of attentional functioning largely accessible for running, collecting, and analyzing large samples of participants following the principles of open science and open source. In fact, using this online platform to administer the ANTI-Vea allowed the collection of data from 349 valid participants for this thesis. Moreover, evidence from more than a dozen studies that have employed this task shows that executive and arousal attentional processes are empirically dissociable both through experimental manipulations (e.g., Hemmerich et al., 2023; Sanchis et al., 2020) and within the association pattern of some attention-related traits (e.g., Cásedas et al., 2022). Finally, although the reliability of the indices of the online version of the ANTI-Vea did not differ substantially from either the lab version or subtask versions for the attentional networks (i.e., ANT or ANTI), indices of differential scores (mainly alerting and orienting networks) or vigilance decrement exhibited rather suboptimal reliability. The latter underscores the importance of using large samples to counteract the issues of statistical power associated with low reliability in correlational designs (Parsons et al., 2019).

In **Studies 2–4** (**Chapter IV**), we aimed to provide transparent and reliable evidence on the relationship between attentional processes and self-reported ADHD symptoms, distinguishing between (a) executive and arousal components as well as between (b) childhood (retrospectively assessed), adult, and late-onset symptoms. In **Study 2**, we observed a neurodevelopmental dissociation consistent with Halperin and Schulz's (2006) model. Specifically, symptoms in childhood were associated with arousal ANTI-Vea indices (i.e., alerting network and arousal vigilance), while symptoms in adulthood correlated with executive measures of vigilance decrement. However, neither the preregistered close replication with robustness checks via multiverse analyses (**Study 3**) nor a multi-sample study with over four times the original study's sample size (**Study 4**) supported the neurodevelopmental dissociation initially found. In fact, the final pattern of results suggested that both executive and arousal alterations were associated with ADHD symptoms in childhood, adulthood, and late onset (i.e., symptoms in adulthood after controlling for those in childhood). This highlights the fragility of conclusions drawn from relatively exploratory studies (i.e., **Study 2**), despite having a conventionally acceptable sample size in the area (i.e., N = 113 valid participants). At the same time, the importance of large sample replication studies for proper control of statistical errors is underscored, especially in a literature where reliability issues in various neurocognitive measures may lead to an attenuation of observed effect sizes.

In **Study 5** (**Chapter V**), our objective was twofold: (a) to examine whether current interventions for ADHD, translational or otherwise, enhance the regulation of arousal mechanisms, and (b) to identify promising arousal-based translational interventions for ADHD. Unlike the previous chapters, in this study the arousal measure of interest was recorded by psychophysiological indices of the functioning of the autonomic nervous system. This is partly because indices of autonomic functioning are considered by some to be a more direct measure of arousal than indices based on performance in cognitive

tasks (Bellato et al., 2020; Idrees et al., 2023). Furthermore, in intervention studies for people with ADHD, arousal measures are either lacking (e.g., simple reaction time tasks, such as arousal vigilance indices in the ANTI-Vea) or unreliable (e.g., the alerting network in the ANT or ANTI versions; Ishigami et al., 2016). Preliminary results have identified 12 studies analyzing the effects of nonpharmacological interventions for ADHD on autonomic arousal indices. However, given the low statistical power and high heterogeneity across study designs and intervention types, drawing robust conclusions on the current state of the art in this area was challenging. Nonetheless, there is a tentative suggestion that, at least, most nonpharmacological interventions may not effectively improve arousal regulation in individuals with ADHD.

In sum, the results from these five studies lead to the following conclusions. First, the ANTI-Vea seems to be valid for the empirical dissociation between executive and arousal attentional processes. While some of its indices may raise reliability concerns, these can be mitigated by increasing the sample size (**Study 1**). Second, even when using the ANTI-Vea task in relatively large samples of community adults, both executive and arousal alterations were indistinctly associated with increased ADHD symptoms, irrespective of age (childhood vs. adult) or course (child-onset vs. late-onset) of such symptoms (**Studies 2–4**). Third, it appears unlikely that most current nonpharmacological interventions can enhance the functioning of the arousal processes underlying ADHD symptoms (**Study 5**). In the next section, we will integrate these findings with previous literature and derive proposals that may contribute to the future of translational interventions for ADHD.

Findings in context

Neurocognitive nature of late-onset ADHD: Implications for conceptualization and translational interventions

One of the key findings of this thesis has been the suggested pathophysiological similarity between childhood-onset and late-onset ADHD symptoms. Specifically, both executive and arousal processes were related to ADHD symptoms irrespective of whether these symptoms had an early or late age of onset. Although using a different design from conventional, our findings are relevant to one of the hottest debates surrounding ADHD (Sonuga-Barke et al., 2023): Is late-onset ADHD a distinct condition from standard ADHD? The implications of this issue affect both the conceptualization of ADHD itself and the developmental considerations for the design of translational interventions. This section aims to contextualize and operationalize this ongoing debate, situate our contributions, and outline future directions.

The root of this issue lies in the conception of ADHD development. From the earliest descriptions, ADHD-like syndromes were considered conditions manifested early in life with a benign course that diminished with age (Crichton, 1798, as cited in Asherson & Agnew-Blais, 2019). Subsequent formulations continued to consider ADHD-like disorders as childhood syndromes that were outgrown during adolescence. It was not until a few decades ago that longitudinal studies revealed that ADHD-like conditions can persist into adulthood (Faraone, Biederman, & Mick, 2006; Wood et al., 1976), thus refuting the notion that it was a disorder restricted to childhood. Therefore, the ADHD trajectory, coupled with its high heritability and associated neurocognitive impairments,[24] led diagnostic manuals

[24] In fact, the neural relevance in ADHD-like syndromes was reflected in the terms "minimal brain damage" and "minimal brain dysfunction" that used to be employed to denote the disorder.

to deem ADHD as a child-onset neurodevelopmental disorder. This is the case of the current *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association [APA], 2013) and *International Statistical Classification of Diseases and Related Health Problems* (11th ed,; ICD-11; World Health Organization [WHO], 2019).

The notion of ADHD as a neurodevelopmental disorder may convey empirical consistency and diagnostic credibility. However, the definition of neurodevelopmental disorder is somewhat vague and not very explicit. For example, the ICD-11 defines them as "behavioural and cognitive disorders that arise during the developmental period" and, unlike schizophrenia or bipolar disorder, "whose core features are neurodevelopmental" (WHO, 2023, para. 1). In the case of DSM-5, neurodevelopmental disorders "onset in the developmental period" and "typically manifests early in development, often before the child enters grade school" (APA, 2013, p. 31). In neither case is it clear what is initiated (e.g., the full disorder? Some symptoms present but only to a subclinical level? Neurocognitive liability?) nor how far the developmental period extends, noting that prefrontal maturation can go beyond 30 years (Kolk & Rakic, 2022).

Although the delimitation of neurodevelopmental disorders is ambiguous, both manuals are clearer in applying such boundaries to ADHD. In particular, they set that several (DSM-5; APA, 2013) or significant (ICD-11; WHO, 2019) ADHD symptoms must be present prior to age 12.[25] Consequently, ADHD would be restricted to a childhood-onset neurodevelopmental disorder. This implies assuming that adolescents or adults with ADHD had been carrying the disorder since childhood.

[25] Before DSM-5, the upper limit of age of onset was set at 7 years. The decision to extend this boundary to 12 years was the result of retrospective studies that found no differences in patterns of psychiatric comorbidity, functional impairment, familial risk, neurocognitive deficits, and response to medication between early (age of onset ≤ 7 years) and later onset groups (Faraone, Biederman, Doyle, et al., 2006; Faraone, Biederman, Spencer, et al., 2006; Reinhardt et al., 2007).

Nevertheless, the neurodevelopmental nature of ADHD has been challenged by recent research from a range of countries (Agnew-Blais et al., 2016; Caye et al., 2016; Cooper et al., 2018; Moffitt et al., 2015). Using a population-based longitudinal design, these studies followed a cohort of individuals who received diagnostic evaluations in childhood and adulthood. Surprisingly, they found that a substantial proportion of adults with ADHD did not meet the diagnostic threshold—nor even several symptoms—in childhood. Although there is a dispute on exact rates of this so-called late-onset ADHD, it is estimated to constitute around 1%–2% of the population and roughly half of the all adults with ADHD (Asherson & Agnew-Blais, 2019). Of note, as any disorder, late-onset ADHD also presents clinical impairment that demands clinical attention and further research on its causes.

In broad terms, explanations of late-onset ADHD can be classified between denialist and affirmational accounts. Denialist views consider that late-onset ADHD is not a genuine phenomenon, but a product of methodological and/or statistical artifacts. For example, since the method commonly used to diagnose ADHD in childhood (i.e., parent-report) is different from that used in adulthood (i.e., self-report), it is possible that assessment in adulthood may focus on more internalizing aspects of the syndrome (Sonuga-Barke, 2017). Furthermore, differences in diagnostic accuracy between the two procedures, coupled with routine diagnostic errors, might lead to false positives in adulthood and/or false negatives in childhood (Agnew-Blais, 2017; Faraone & Biederman, 2016). Moreover, late-onset ADHD cases could actually be secondary attentional impairments that are better explained by another mental disorder (e.g., mood disorder, anxiety disorder, substance use disorder), which is an exclusion criterion according to DSM-5 (Taylor et al., 2022). Although these artifacts can probably explain part of the cases of late-onset ADHD, a thorough review of this literature concluded that at least a substantial portion of these cases are genuine (Asherson & Agnew-Blais, 2019).

Based on the idea that late-onset ADHD exists, affirmational perspectives are mainly divided between those who consider this disorder to be part of ADHD and those who hold it as a new and distinct clinical entity. Empirical arguments in this debate typically focus on comparing late-onset ADHD with childhood-onset ADHD across a wide range of domains, namely genes, neurocognitive impairments, comorbidities, treatment response, demographics, and other clinical features. Although it may be useful to consider these aspects, it is crucial to establish a framework that allows for the operationalization of the different affirmational proposals in order to formally compare them, focusing on key elements and implications for the conceptualization of ADHD in general. To this end, **Figure 1** provides an attempt to represent three different proposals grounded on the developmental causal modelling framework (Morton & Frith, 1995).

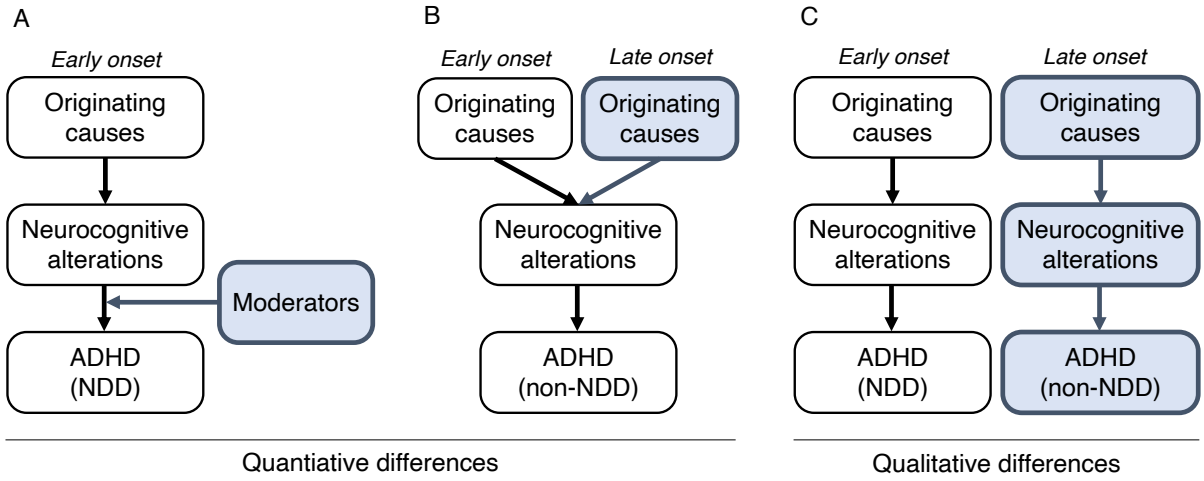


Figure 1. Three competing proposals to conceptualize the neurodevelopmental nature of ADHD as a function of the differences between its child- and late-onset forms. The blue-colored boxes represent elements of each model that are distinctive for late-onset ADHD. **Panels A–B**: The differences between child-onset ADHD and late-onset ADHD are quantitative; therefore, all forms of ADHD should be conceived either as an NDD (if **Panel A** model is supported) or as a non-NDD (if **Panel B** model is supported). **Panel C**: The differences between child-onset ADHD and late-onset ADHD are qualitative; therefore, child-onset ADHD should be conceived as an NDD, while late-onset ADHD should be conceived as a non-NDD. NDD = neurodevelopmental disorder; non-NDD = non-neurodevelopmental disorder.

The two first proposals (**Figure 1 A–B**) are quantitative difference models. That is, in these scenarios late-onset ADHD is no more than a variant of the same disorder that

appear at a different time. The difference between these two proposals is in the model factor that differs in late-onset ADHD. The first (**Figure 1A**) points to the moderating factors of the relationship between neurocognitive alterations and ADHD symptoms. According to this view, neurocognitive alterations are present and stable from early childhood, but symptoms may remain obscured until later when the external (e.g., supportive family) or internal (e.g., high IQ) scaffoldings are removed or insufficient to meet new environmental demands (e.g., moving out of home, starting university, paternity). This idea of masked ADHD in childhood, especially in subthreshold cases, has been held by some scholars (Faraone & Biederman, 2016; Kosaka et al., 2019). According to this position, it would be reasonable to consider ADHD, including its late onset, as a neurodevelopmental disorder, since neurocognitive lability would already be present in childhood.

The second quantitative account (**Figure 1B**) focuses on the etiological factors of ADHD across lifespan. From this perspective, the array of neurocognitive alterations underpinning ADHD can also be caused after childhood by de novo interactions between biological (e.g., brain damage, impaired maturation, substance abuse), psychological (e.g., stress, effort or delay aversion), and environmental (e.g., high demands, harsh reactions) variables. In this case, neurocognitive alterations would not be present in childhood. Since neurocognitive signs are the earliest manifestation and the most lenient operationalization of a neurodevelopmental disorder (Sonuga-Barke, 2017), a disorder that does not meet this criterion for a substantial proportion of its cases should not be considered neurodevelopmental. On the contrary, ADHD would better fit the category of general mental health disorder (Rohde, 2023).

Continuing with the second proposal, note that plausible nongenetic etiologies (e.g., extreme institutional deprivation, high digital exposure) are not exclusion criteria for childhood ADHD; therefore, they should not be for adolescent or adult ADHD either.

Furthermore, etiologies with a genetic component that begin to cause neurocognitive alterations after childhood—either because the genetic load is low and requires riskier environments, or because such genes have a later mechanism of expression (e.g., Z. Chang et al., 2013)—would not fit with the notion of early onset of central nervous system changes characteristic of neurodevelopmental disorders. While the phenomenon of masked ADHD in childhood may also occur in this second model, this would not justify the conceptualization of ADHD as a neurodevelopmental disorder.

Finally, the third proposal (**Figure 1C**) is a qualitative difference model. In this case it does make sense to conceptualize late-onset ADHD as a distinct disorder. While in **Figure 1B** both early and late originating causes result in a similar set of neurocognitive alterations (i.e., equifinality at the neurocognitive level), in this last scenario the etiological factors of late-onset ADHD cause a neurocognitive profile that, despite heterogeneity, is substantially different from that of child-onset ADHD. For example, following the Halperin and Schulz's (2006) neurodevelopmental model, child-onset ADHD would be related to arousal alterations, while late-onset ADHD would associate with executive impairments. Alternatively, it could be the case that late-onset ADHD has no obvious neurocognitive deficits (Moffitt et al., 2015). The idea of assigning such a determinant role to neurocognitive alterations in late-onset ADHD is due to two main reasons. First, this criterion is similar to that employed by another nosological approach to conceptualize potential cases of ADHD acquired through high digital exposure (Sonuga-Barke & Kostyrka-Allchorne, 2023): If the exposure produces brain changes, it can be considered ADHD; if the changes are only behavioral, it is a phenocopy (i.e., a non-ADHD syndrome mimicking ADHD). Second, a distinction based on neurocognitive alterations is highly useful for the design and implementation of translational interventions. Thus, in this case it is worth making a distinction between child-onset ADHD (a neurodevelopmental disorder) and adult-onset ADHD (a non-neurodevelopmental disorder).

Having operationalized the three proposed conceptualizations of ADHD, we are now better positioned to clearly analyze their evidence, including the contributions of this thesis. First, there is the distinction between quantitative and qualitative difference models. In order to know which of the two receives more support, the key is to test whether neurocognitive impairments are different for early-onset ADHD compared to late-onset ADHD. As we noted previously, the seminal work of Moffit et al. (2015) supported the qualitative differences model, in that individuals with late-onset ADHD did not show neurocognitive impairment, unlike adults with childhood-onset ADHD who did show neurocognitive impairment. However, subsequent research, following a similar longitudinal design, mainly supported the quantitative differences models (Cooper et al., 2018; Ilbegi et al., 2021; Riglin et al., 2022): either both groups differed from the control group, or the groups did not differ from each other.

In this context, the empirical series of this thesis (**Studies 2–4**) is intended to complement this body of evidence by employing a dimensional approach. In line with the bulk of the literature, the combination of our studies supported the quantitative difference model, suggesting that the distinction between the two forms of ADHD is not adequate. The main value of our contribution was the transparent control of statistical errors and the measurement of two theoretically and empirically dissociable neurocognitive processes. Regarding the former, not only does our experimental series have the largest sample size to date for analyzing this research question, but it also includes preregistered analyses, sophisticated power analyses controlling for measurement error, and multiverse analyses to examine the robustness of our conclusions. As for the latter, despite the importance of including non-executive measures relevant for ADHD, this had hardly been done in this literature, as they have chosen to include different executive measures. In this sense, to the best of our knowledge, our empirical series is the first to show that alterations in

arousal processes are related to late-onset ADHD symptoms, challenging the idea that arousal dysregulation was more typical of child-onset ADHD.

Taken together, evidence from both longitudinal studies using case-control designs and the present thesis, which employs a dimensional approach with retrospective and concurrent self-reports, converges in supporting quantitative differences between child- and late-onset ADHD. The idea that both forms of the disorder have similar neurocognitive alterations is also somewhat consistent with the fact that current translational interventions have no differential effects for children versus adults, assuming that the latter group has a substantial proportion of individuals with late-onset ADHD. At the brain level, it has been found that the volume of a region associated with ADHD symptomatology in adolescence also predicts the emergence of symptoms in adulthood (Albaugh et al., 2019). In any case, to consolidate support for this scenario of quantitative differences, more research is needed with neurocognitive measures at different levels (e.g., brain connectivity, autonomic nervous system, relationships with the endocrine system and the microbiome) and of multiple processes (e.g., delay aversion, mind-wandering).

Going further, assuming the quantitative differences model implies that the age-of-onset criterion for ADHD should be abolished (Faraone & Biederman, 2016; Sonuga-Barke, 2017). Although this entails that the disorder can manifest itself for the first time at any age, it does not necessarily mean that ADHD ceases to be a neurodevelopmental disorder. To shed light on this question, the first proposal (**Figure 1A**) should be tested against the second (**Figure 1B**). At present the evidence on this issue is not very clear. It can be argued that the lower polygenic risk score and higher female ratio in late-onset ADHD (Asherson & Agnew-Blais, 2019) could reflect a post-childhood acquired etiology for this variant of the disorder (favoring the model in **Figure 1B**). However, it would also be plausible that these etiological factors in turn impact the moderating factors that mask the onset of symptoms in childhood (favoring the model in **Figure 1A**). For example, due

to cultural factors, girls may put more effort into masking their ADHD symptoms to be socially perceived as typical girls, a situation that could become more untenable as adulthood arrives (Sonuga-Barke, 2023).

Following the proposed framework, the above question should be addressed by longitudinal studies with multiple neurocognitive measures. Specifically, the key would be to determine whether adults with late-onset ADHD had brain and cognitive alterations in childhood of similar magnitude to those who already had ADHD (favoring the model in **Figure 1A**; ADHD as a neurodevelopmental disorder) or not (favoring the model in **Figure 1B**; ADHD as a non-neurodevelopmental disorder). At the same time, the role of putative protective moderating factors (e.g., IQ, supportive family) in the expression of disorder symptoms could be analyzed. Currently, studies addressing this question are scarce, contradictory, and underpowered (Ilbegi et al., 2021; Moffit et al., 2015). Nor do they directly address the role of putative protective moderating factors (e.g., IQ, supportive family) in the expression of disorder symptoms.

Mechanisms of translational interventions for ADHD: From the executive to the arousal pathway

In the preceding section, we concluded that the neurocognitive alterations of child-onset and late-onset ADHD seems to be identical to each other. Therefore, translational interventions should be designed to address the same neurocognitive targets irrespective of the individual's age or the onset of the disorder. In this section we will discuss what these neurocognitive targets should be and how our studies can shed light on this question.

Previous theoretical and empirical literature, mostly built upon case–control designs, suggests that the heterogeneous pathophysiology of ADHD involves both executive and

non-executive domains (Luo et al., 2019). This conclusion is consistent with that of the empirical series of the present thesis (**Studies 2–4**), based on a dimensional perspective. Concretely, we found that executive and arousal processes seem to underlie ADHD symptoms across the population, irrespective of age of symptomatology onset. In fact, we observed that the associations of executive and arousal processes with ADHD symptoms were independent from each other, thus highlighting the dissociation between both neurocognitive processes.

The variety of mechanisms underlying ADHD contrasts with the type of neurocognitive targets typically targeted in translational interventions for this disorder. In this sense, it has been argued that these interventions appear to have been designed almost exclusively to train executive domains, while targeting arousal regulation or other non-executive processes is generally ignored (Sonuga-Barke & Cortese, 2018; Westwood et al., 2023). This is the case of current computerized cognitive training programs for ADHD, which aim to strengthen different executive processes (e.g., working memory, response inhibition, attentional control) through the practice of challenging tasks in which they are involved. This type of practice is also the main component of structured-play-based parent training, which is employed at the preschool age and is delivered in a non-computerized format (Coll-Martín et al., 2019). Like cognitive training, most neurotherapies appear constrained in their focus on executive domains. In this sense, the most common modalities of neurofeedback and transcranial direct current stimulation (tDCS) are largely based on the cerebral cortex, while their impact on subcortical structures associated with arousal (e.g., locus coeruleus) or other non-executive processes is more difficult or indirect. Of note, it has been proposed that both theta/beta neurofeedback bands play a crucial role in executive control processes (Bluschke et al., 2016). Moreover, a protocol of tDCS served to mitigate executive but not arousal vigilance (Hemmerich et al., 2023).

In addition to being inconsistent with the pathophysiological heterogeneity of ADHD, the excessive focus on directly targeting executive domains in the above-mentioned translational interventions contrasts with current cognitive training models for the general population (Cásedas & Lupiáñez, 2023; Gathercole et al., 2019; von Bastian et al., 2022). In fact, the influential capacity-efficiency model distinguishes two pathways by which training-induced improvements can be transferred to untrained tasks or everyday activities (von Bastian et al., 2022). The first pathway aims to expand the intrinsic *capacity* of trained cognitive functions and has been suggested to directly involve executive control processes, such as in mindfulness training (Cásedas & Lupiáñez, 2023). In contrast, the *efficiency* pathway, which aims to optimize performance within the limits of cognitive capacity, encompasses domains that are generally not primarily executive in nature but can enhance the functioning of executive control (e.g., arousal regulation, motivation, strategies, affective processing). Critically, evidence suggests that the transfer of training-induced improvements does not seem occur through increases in cognitive capacity, but through gains in efficiency (von Bastian et al., 2022).

Viewing ADHD from a dimensional perspective provides a rationale for incorporating insights from general cognitive training theories into the analysis and development of translational interventions for the disorder. Just like in ADHD, the most common formats of cognitive training programs (e.g., “computerized cognitive training”) for neurotypical individuals have failed to yield improvements in cognitive performance in tasks or daily life (Sala & Gobet, 2019; Simons et al., 2016; Stojanoski et al., 2021). This suggests that the translational crisis in ADHD is part of a broader crisis that affect the effectiveness of cognitive training in the general population. Since the traditional cognitive training format is based on the principles of neural plasticity and the metaphor of the “brain as a muscle” (Simons et al., 2016), this cognitive training modality consists of the mere practice of generally repetitive and decontextualized tasks that usually involve executive functions.

The fact that these types of computerized cognitive training—and similar ones—are exclusively focused on improving cognitive capacity, at the expense of efficiency, may explain their failure to generate neurocognitive transfer that, in turn, has an impact on ADHD symptoms.

Taken together, although neurocognitive alterations in ADHD involve both executive and non-executive processes, translational interventions tend to target only the former. At the same time, general theories of cognitive training suggest that the limited efficacy of translational interventions for ADHD may be due to their focus on improving executive capacities, which seems to be theoretically implausible. Unlike cognitive capacity, training-induced improvements in cognitive efficiency has received substantial support. Therefore, the development of translational interventions that favor cognitive efficiency mechanisms could generate transferable improvements to the neurocognitive processes altered in the disorder and, thus, have a genuine impact on ADHD symptoms. These types of translational interventions in ADHD would involve moving from directly targeting executive control processes more closely linked to neurocognitive capacity toward the search for non-primarily executive targets more related to the mechanisms of neurocognitive efficiency. Given the relevance of arousal alterations in the pathophysiology of ADHD, the regulation of this mechanism could play a crucial role for translational interventions that promote cognitive efficiency.

Figure 2 illustrates a refined model of translational intervention for ADHD, distinguishing the two pathways of transfer of training-induced improvements and the neurocognitive alterations most closely linked to each. On the one hand, there are intervention approaches designed to improve executive capacity, such as computer-based cognitive training or structured-play-based parent training, as well as neurotherapies that primarily impact executive processes (e.g., more common

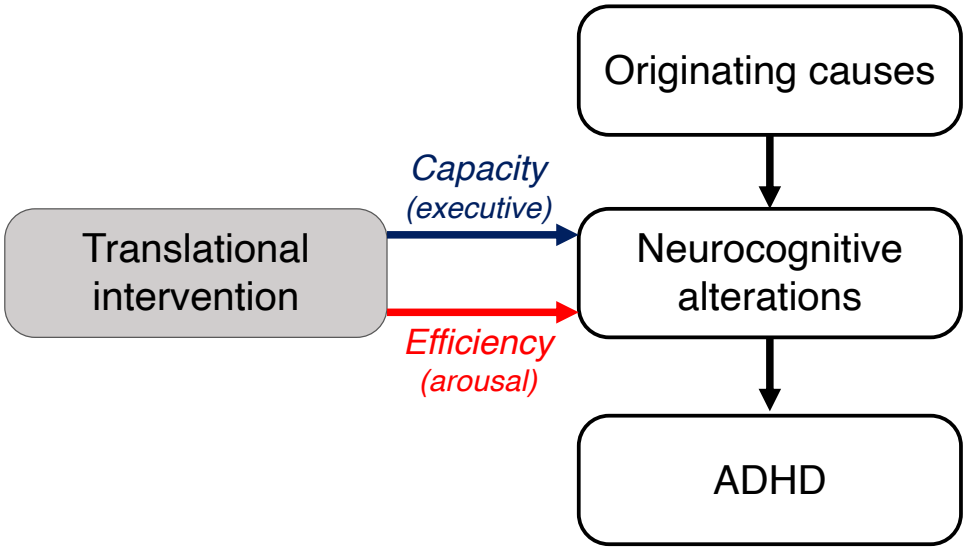


Figure 2. Two pathways through which translational interventions can impact the neurocognitive processes underlying ADHD. While there is little evidence that translational intervention improves cognitive ability, improvement in cognitive efficiency is a more promising avenue. This justifies the development of arousal-based translational interventions to address the core symptoms of the disorder.

neurofeedback and tDCS protocols). This first pathway seems to fail to improve the neurocognitive alterations underlying ADHD and thus the core symptoms of the disorder (see **Translational Interventions for ADHD** in **Introduction**). In contrast, intervention approaches that promote cognitive efficiency, presumably by regulating arousal mechanisms in ADHD, constitute a promising avenue for impacting symptoms of the disorder (see von Bastian et al., 2022, for the substantial evidence supporting training-induced improvements in cognitive efficiency).

Unlike executive-based translational interventions, *arousal-based translational interventions* for ADHD may have been less prominent due to the less intuitive nature of the efficiency (vs. capacity) pathway and less accessibility to study key hubs in arousal processes (e.g., locus coeruleus). However, the relevance of this target is evident in several aspects. For example, both stimulant and non-stimulant medications have in common the action on noradrenaline (Cortese, 2020), the main neurotransmitter of the alertness network (Petersen & M. I. Posner, 2012). Furthermore, arousal impairments were more consistent and of greater magnitude than executive impairments in the multi-

sample study of the present dissertation (**Study 4**). Finally, theories of arousal dysregulation tend to be integrated with motivational mechanisms, which represent a fundamental neurocognitive pathway in ADHD (Sonuga-Barke, Wiersema, et al., 2010; van der Meere et al., 2010). In fact, the role of motivation in arousal regulation makes clear the linkage of this process with cognitive efficiency, rather than cognitive capacity associated with resources (Botvinick & Braver, 2015). The biopsychosocial view of ADHD as a condition that is not necessarily dysfunctional is also in line with the motivational account.

In addition to the paucity of translational interventions for ADHD based on arousal regulation, **Study 5** failed to identify nonpharmacological interventions that, either intentionally or incidentally, could upregulate autonomic arousal. Therefore, at present, the design of translational interventions targeting arousal mechanisms must be based on mainly theoretical aspects. Given that effort is the main regulatory mechanism of arousal (Sergeant, 2005), one possible avenue would be to develop interventions that promote the individual's willingness to exert effort. In this sense, contemporary proposals point out that the effort tends to be aversive, but that in a context in which effort is consistently rewarded, people might learn that effort is valuable and become more willing to exert it in daily life (Inzlicht et al., 2018). In this process, known as learned industriousness, effort becomes a secondary reinforcer, as it is rewarding by itself. Of note, this mechanism of cognitive efficiency has been proposed as one of the pathways to explain the improvements in cognitive performance produced by mindfulness practice (Cásedas et al., 2020) and learning to play an instrument (Román-Caballero et al., 2022), two cognitive training modalities that allow effort to be associated with natural rewards from the personal and social context. As these cognitive training modalities have not been sufficiently studied in ADHD, future studies should explore their impact on ADHD symptoms and the role of effort and arousal processes as mediating mechanisms.

In relation to neurotherapies that target arousal mechanisms, the need for neurofeedback protocols based on sleep markers, one of the most frequent alterations in ADHD, has been pointed out (M. Lecendreux, personal communication, May 19, 2023). Likewise, it has been proposed that brain stimulation of regions closer to the brainstem and locus coeruleus, such as external trigeminal nerve stimulation, may have a genuine impact on ADHD symptoms (E. J. S. Sonuga-Barke, personal communication, May 20, 2023). Future research should explore these promising hypotheses.

APPENDIX

Cuando el TDAH comienza en la adultez

¿Un trastorno distinto?

The content of this work has been published as:

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https://www.cienciacognitiva.org/?p=2245

Cuando el TDAH comienza en la adultez ¿Un trastorno distinto?

Durante un largo tiempo se ha considerado el trastorno por déficit de atención e hiperactividad (TDAH) como un trastorno del desarrollo, lo que en última instancia supone aceptar que las personas adultas que lo padecen lo llevarían arrastrando desde la infancia. Sin embargo, estudios longitudinales han puesto de manifiesto no solo que el TDAH puede comenzar en la adultez, sino también que este inicio tardío es lo habitual en la población adulta con TDAH. Saber si el TDAH de inicio en la adultez es un trastorno distinto al TDAH convencional resulta primordial, puesto que entraña importantes consecuencias para la práctica clínica.

El trastorno por déficit de atención e hiperactividad (TDAH) se define habitualmente como una condición del neurodesarrollo caracterizada por un patrón persistente de inatención, hiperactividad e impulsividad que interfiere con el funcionamiento social, académico o laboral (American Psychiatric Association [APA], 2013). Aunque durante décadas se pensaba que el TDAH afectaba únicamente a niños en edad escolar, progresivamente se ha ido tomando conciencia de que este trastorno puede estar presente en la edad adulta. De hecho, la actual versión del Manual Diagnóstico y Estadístico de Trastornos Mentales, el DSM-5, ha sido la primera en incorporar criterios diagnósticos específicos para el TDAH en la adultez, que afecta a un 2.5% de esta población, frente al 5% de casos en la infancia.

Una presuposición sostenida por manuales diagnósticos, personal clínico y el público general ha sido que el TDAH adulto no es más que una continuación de un TDAH infantil que se ha hecho persistente. El propio DSM-5 incluye al TDAH dentro de los trastornos del neurodesarrollo y afirma que “el TDAH empieza en la infancia” (APA, 2013,

p. 61). En consecuencia, establece que para diagnosticar este trastorno es imprescindible que los síntomas hayan comenzado antes de los 12 años.

Sin embargo, este supuesto fundamental del TDAH adulto comenzó a ponerse en entredicho a partir de una investigación pionera de Moffitt y colaboradores (2015). Este estudio realizó un seguimiento a 1009 individuos desde su nacimiento hasta los 38 años. Sorprendentemente, encontraron que apenas había solapamiento entre el grupo de participantes que habían sido diagnosticados en la infancia y los que posteriormente recibieron el mismo diagnóstico en la adultez (véase Figura 1).

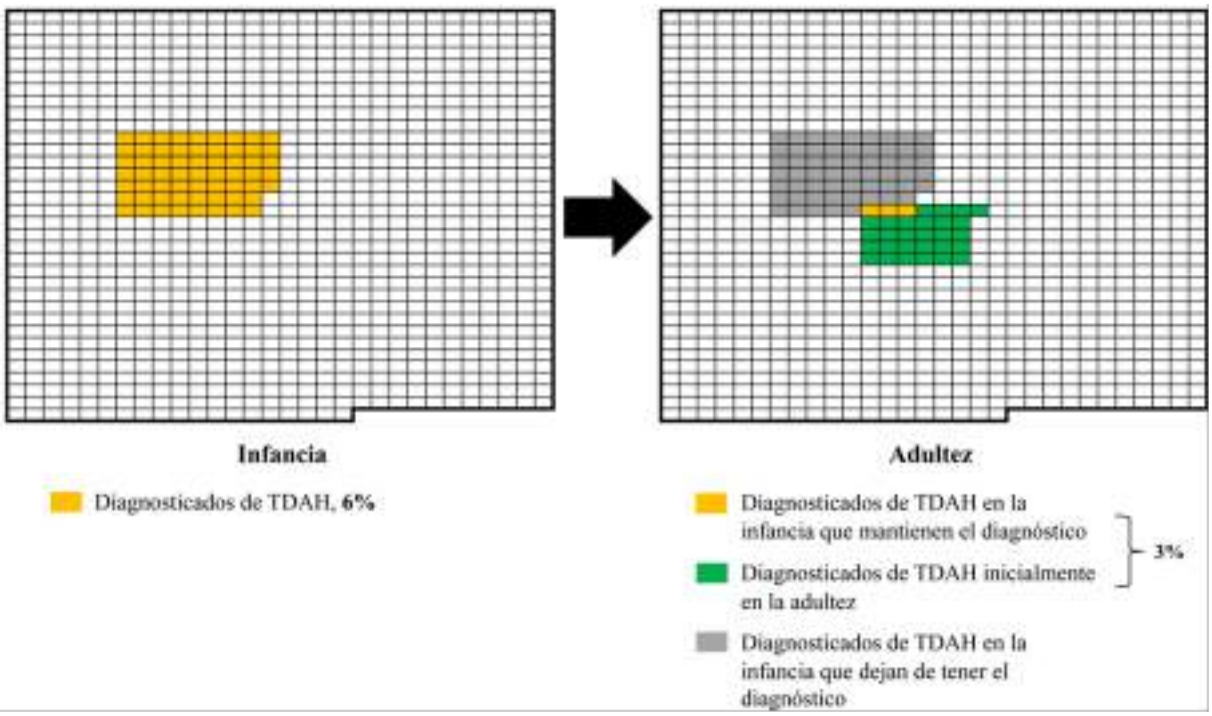

Figura 1. Resultados del seguimiento de una cohorte representativa compuesta por 1009 individuos de Nueva Zelanda desde su nacimiento hasta los 38 años (Moffitt et al., 2015). *Cada uno de ellos está representado por un elemento de la cuadrícula. Una primera evaluación diagnóstica de TDAH tuvo lugar cuando estos participantes estaban en su infancia (panel izquierdo). Posteriormente, cuando los participantes tenían 38 años se volvió a realizar un diagnóstico de TDAH, pero excluyendo el criterio de la edad de inicio de síntomas (panel derecho). Como era de esperar, los resultados mostraron un 6% de diagnósticos de TDAH en la infancia y un 3% en la adultez, porcentajes cercanos a los índices de prevalencia establecidos. La sorpresa llegó cuando observaron que ambos grupos de personas con TDAH estaban compuestos por individuos prácticamente distintos. Concretamente, solo un 5% de quienes fueron diagnosticados de TDAH en la infancia mantuvieron el diagnóstico en la adultez, mientras que el 90% de personas adultas con TDAH no habían recibido el diagnóstico cuando eran niños/as.*

Así, aunque la existencia de un TDAH de inicio en la adultez es ya un fenómeno ampliamente aceptado en la comunidad científica, la controversia gira en torno a la siguiente cuestión: ¿qué relación tiene este trastorno con el TDAH de inicio en la infancia? Una posibilidad es que las diferencias entre ambas formas del trastorno sean meramente cuantitativas. Es decir, que las personas con TDAH tardío tengan una condición menos severa o ciertos factores de protección (p. ej., apoyo familiar, menos demandas, habilidades cognitivas compensadoras) que demoren la aparición de los síntomas clínicos hasta que los factores de riesgo aumenten o los de protección disminuyan. La otra posibilidad es que el TDAH tardío sea un trastorno de naturaleza cualitativamente distinta del TDAH convencional. A favor de esta idea está que el grupo de TDAH tardío, a diferencia del anterior, muestra una menor heredabilidad, una distribución por sexos más equitativa y un nivel de síntomas en la infancia claramente alejado del umbral diagnóstico (Agnew-Blais et al., 2016; Moffitt et al., 2015).

Una estrategia especialmente útil para profundizar en la naturaleza de esta distinción consiste en comparar el perfil neuropsicológico de ambos tipos de TDAH. Un estudio reciente de nuestro grupo de investigación (Coll-Martín et al., 2021) puede arrojar luz en este asunto. En él pedimos a 120 estudiantes universitarios que evaluaran el grado en que recordaban haber experimentado síntomas TDAH en su infancia y el grado en que los experimentaban en la actualidad. Posteriormente, empleamos una tarea atencional que medía dos componentes de vigilancia: la mera reactividad al ambiente (vigilancia de activación) y la capacidad para detectar estímulos relevantes infrecuentes (vigilancia ejecutiva). Ambos componentes suelen decaer durante el tiempo de ejecución de la tarea (Hemmerich et al., 2020).

Como se muestra en Figura 2, nuestros resultados arrojaron una doble disociación. Mayores niveles de sintomatología TDAH en la infancia se asociaron con un peor funcionamiento de la vigilancia de activación, pero no de la vigilancia ejecutiva. En

cambio, la severidad de los síntomas en la adultez estaba relacionada con un rendimiento más pobre en la vigilancia ejecutiva, pero no en la de activación.

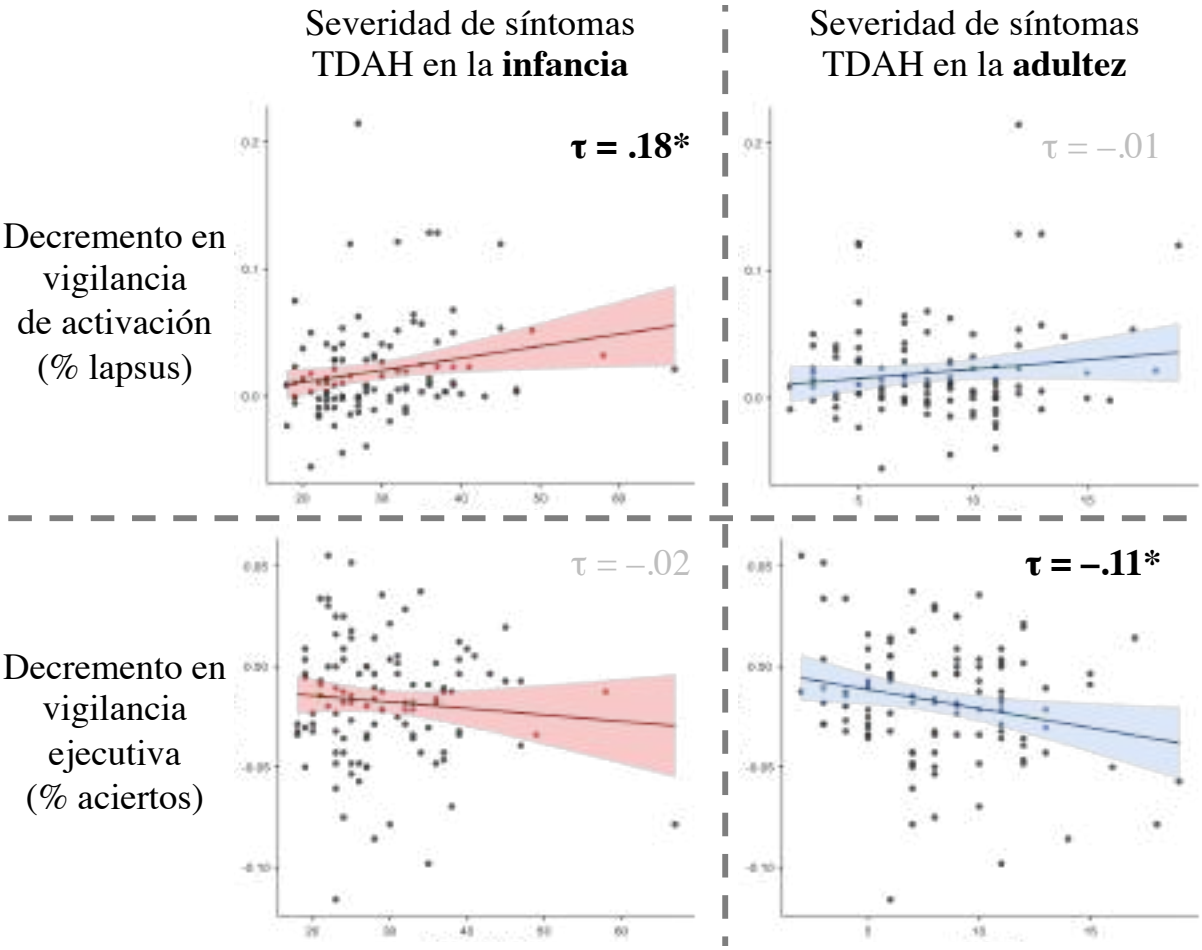

Figura 2. Diagramas de dispersión mostrando la correlación entre los síntomas TDAH (en la infancia y en la adultez) y los componentes de la vigilancia (de activación y ejecutiva). *La severidad de los síntomas TDAH en la infancia y en la adultez (ejes de abscisa) se midió con las escalas BAARS-IV y ASRS-5, respectivamente. La vigilancia de activación se calcula como el incremento en el porcentaje de lapsus (respuestas excesivamente lentas) que se produce en la tarea a medida que pasa el tiempo. La vigilancia ejecutiva se mide como el decremento paulatino en el porcentaje de veces que el participante detecta correctamente el desplazamiento infrecuente del estímulo objetivo. En negrita y con asterisco se muestran los coeficientes de correlación que resultaron significativos. Se puede observar que, en tanto que el decremento en la vigilancia de activación se relaciona con los síntomas en la infancia, el decremento en la vigilancia ejecutiva lo hace con los síntomas en la adultez.*

Aunque estos resultados deben considerarse preliminares y se ven limitados por el hecho de basarse en autoinformes retrospectivos para medir síntomas en la infancia, estos hallazgos apoyarían la concepción del TDAH adulto como una entidad cualitativamente distinta a la de su homólogo en la infancia, con una etiología propia que

merece ser explorada en futuros estudios. De hecho, algunas teorías influyentes sobre el desarrollo del TDAH, como el modelo de Halperin y Schulz (2006), podrían encontrar un mayor acomodo al distinguir la aparición tardía del trastorno.

En definitiva, el reconocimiento del TDAH de inicio en la adultez ha propiciado una nueva línea de investigación para desentrañar su naturaleza y, en consecuencia, ajustar su abordaje práctico. Si las diferencias con el TDAH convencional no van mucho más allá de la edad de inicio, entonces tendría sentido eliminar este criterio diagnóstico para así poder abarcar su debut en la adultez. En cambio, si se encuentran evidencias de que el TDAH tardío es un síndrome cualitativamente distinto del anterior, sería más razonable recogerlo como una nueva categoría diagnóstica fuera de los trastornos del neurodesarrollo.

REFERENCES

References

Adólfsdóttir, S., Sørensen, L., & Lundervold, A. J. (2008). The attention network test: A characteristic pattern of deficits in children with ADHD. *Behavioral and Brain Functions*, *4*, Article 9. https://doi.org/10.1186/1744-9081-4-9

Aduen, P. A., Kofler, M. J., Bradshaw, C., Sarver, D. E., & Cox, D. J. (2020). The role of top-down attentional control and ADHD symptoms in predicting future motor vehicle crash risk. *Neuropsychology*, *34*(8), 894–905. https://doi.org/10.1037/neu0000707

Advokat, C., Martino, L., Hill, B. D., & Gouvier, W. (2007). Continuous Performance Test (CPT) of college students with ADHD, psychiatric disorders, cognitive deficits, or no diagnosis. *Journal of Attention Disorders*, *10*(3), 253–256. https://doi.org/10.1177/1087054706292106

Agnew-Blais, J. (2017, September 21–23). *When should attentional impairment in adulthood be classified as ADHD?* [Conference session]. UKAAN Conference, London, UK.

Agnew-Blais, J. C., Polanczyk, G. V., Danese, A., Wertz, J., Moffitt, T. E., & Arseneault, L. (2016). Evaluation of the persistence, remission, and emergence of attention-deficit/hyperactivity disorder in young adulthood. *JAMA Psychiatry*, *73*(7), 713–720. https://doi.org/10.1001/jamapsychiatry.2016.0465

Agnew-Blais, J. C., Wertz, J., Arseneault, L., Belsky, D. W., Danese, A., Pingault, J.-B., Polanczyk, G. V., Sugden, K., Williams, B., & Moffitt, T. E. (2022). Mother's and children's ADHD genetic risk, household chaos and children's ADHD symptoms: A gene–environment correlation study. *Journal of Child Psychology and Psychiatry*, *63*(10), 1153–1163. https://doi.org/10.1111/jcpp.13659

Albaugh, M. D., Ivanova, M., Chaarani, B., Orr, C., Allgaier, N., Althoff, R. R., D' Alberto, N., Hudson, K., Mackey, S., Spechler, P. A., Banaschewski, T., Brühl, R., Bokde, A.

L. W., Bromberg, U., Büchel, C., Cattrell, A., Conrod, P. J., Desrivières, S., Flor, H., … Potter, A. S. (2019). Ventromedial prefrontal volume in adolescence predicts hyperactive/inattentive symptoms in adulthood. *Cerebral Cortex*, *29*(5), 1866–1874. https://doi.org/10.1093/cercor/bhy066

Albrecht, B., Brandeis, D., Uebel, H., Heinrich, H., Mueller, U. C., Hasselhorn, M., Steinhausen, H.-C., Rothenberger, A., & Banaschewski, T. (2008). Action monitoring in boys with attention-deficit/hyperactivity disorder, their nonaffected siblings, and normal control subjects: Evidence for an endophenotype. *Biological Psychiatry*, *64*(7), 615–625. https://doi.org/10.1016/j.biopsych.2007.12.016

Alister, M., Vickers-Jones, R., Sewell, D. K., & Ballard, T. (2021). How do we choose our giants? Perceptions of replicability in psychological science. *Advances in Methods and Practices in Psychological Science*, *4*(2). https://doi.org/10.1177/25152459211018199

Al-Shargie, F., Tariq, U., Mir, H., Alawar, H., Babiloni, F., & Al-Nashash, H. (2019). Vigilance decrement and enhancement techniques: A review. *Brain Sciences*, *9*(8), Article 8. https://doi.org/10.3390/brainsci9080178

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.).

American Psychiatric Association. (1995). *Manual diagnóstico y estadístico de los trastornos mentales* [Diagnostic and statistical manual of mental disorders] (J. J. López-Ibor Aliño, Dir.; M. Valdés Miyar, Coord.; 4th ed.). Masson. (Original work published 1994).

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). https://doi.org/10.1176/appi. books.9780890425596

Appelbaum, M., Cooper, H., Kline, R. B., Mayo-Wilson, E., Nezu, A. M., & Rao, S. M. (2018). Journal article reporting standards for quantitative research in psychology: The APA

Publications and Communications Board task force report. *American Psychologist*, *73*(1), 3–25. https://doi.org/10.1037/amp0000191

Aragonès, E., Lluís Piñol, J., Ramos-Quiroga, J. A., López-Cortacans, G., Caballero, A., & Bosch, R. (2010). Prevalence in adults of attention deficit hyperactivity disorder using the medical records of primary care. *Revista Española de Salud Pública*, *84*(4), 417–422. https://www.mscbs.gob.es/biblioPublic/publicaciones/recursos_propios/resp/revista_cdrom/vol84/vol84_4/RS844C_415ingles.pdf

Arnett, A. B., Pennington, B. F., Willcutt, E. G., DeFries, J. C., & Olson, R. K. (2015). Sex differences in ADHD symptom severity. *Journal of Child Psychology and Psychiatry*, *56*(6), 632–639. https://doi.org/10.1111/jcpp.12337

Arora, S., Lawrence, M. A., & Klein, R. M. (2020). The Attention Network Test Database: ADHD and cross-cultural applications. *Frontiers in Psychology*, *11*, Article 388. https://doi.org/10.3389/fpsyg.2020.00388

Asherson, P., & Agnew-Blais, J. (2019). Annual research review: Does late-onset attention-deficit/hyperactivity disorder exist? *Journal of Child Psychology and Psychiatry*, *60*(4), 333–352. https://doi.org/10.1111/jcpp.13020

Astle, D. E., & Fletcher-Watson, S. (2020). Beyond the core-deficit hypothesis in developmental disorders. *Current Directions in Psychological Science*, *29*(5), 431–437. https://doi.org/10.1177/0963721420925518

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*(1), 65–94. https://doi.org/10.4324/9781315198941

Barkley, R. A. (2011). *Barkley Adult ADHD Rating Scale-IV (BAARS-IV)*. Guilford Press. https://www.guilford.com/books/Barkley-Adult-ADHD-Rating-Scale-IV-BAARS-

IV/Russell-Barkley/9781609182038

Barkley, R. A., & Fischer, M. (2019). Hyperactive child syndrome and estimated life expectancy at young adult follow-up: The role of ADHD persistence and other potential predictors. *Journal of Attention Disorders*, *23*(9), 907–923. https://doi.org/10.1177/1087054718816164

Barkley, R. A., & Murphy, K. R. (2011). The nature of executive function (EF) deficits in daily life activities in adults with ADHD and their relationship to performance on EF tests. *Journal of Psychopathology and Behavioral Assessment*, *33*(2), 137–158. https://doi.org/10.1007/s10862-011-9217-x

Barr, D. J., Levy, R., Scheepers, C., & Tily, H. J. (2013). Random effects structure for confirmatory hypothesis testing: Keep it maximal. *Journal of Memory and Language*, *68*(3), 255–278. https://doi.org/10.1016/j.jml.2012.11.001

Basner, M., Mollicone, D., & Dinges, D. F. (2011). Validity and sensitivity of a brief psychomotor vigilance test (PVT-B) to total and partial sleep deprivation. *Acta Astronautica*, *69*(11), 949–959. https://doi.org/10.1016/j.actaastro.2011.07.015

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*, *67*, 1–48. https://doi.org/10.18637/jss.v067.i01

Bayo-Tallón, V. (2020). *Efectos de la Terapia Manual sobre la sintomatología del TDAH infantil* [Doctoral dissertation, Autonomous University of Barcelona]. Tesis Doctorals en Xarxa. https://www.tdx.cat/handle/10803/671040#page=1

Bayo-Tallón, V., Esquirol-Caussa, J., Pàmias-Massana, M., Planells-Keller, K., Cascos-Rodríguez, A., & Palao-Vidal, D. J. (2020). Effectiveness of a manual therapy program as adjuvant treatment for school-age children with attention-deficit/hyperactivity disorder: A randomized pilot study. *SAGE Open*, *10*(4).

https://doi.org/10.1177/2158244020983023

Beauchaine, T. P. (2003). Taxometrics and developmental psychopathology. *Development and Psychopathology*, *15*(3), 501–527. https://doi.org/10.1017/s0954579403000270

Beauchaine, T. P., Neuhaus, E., Gatzke-Kopp, L. M., Reid, M. J., Chipman, J., Brekke, A., Olliges, A., Shoemaker, S., & Webster-Stratton, C. (2015). Electrodermal responding predicts responses to, and may be altered by, preschool intervention for ADHD. *Journal of Consulting and Clinical Psychology*, *83*(2), 293–303. https://doi.org/10.1037/a0038405

Becker, S. P., Willcutt, E. G., Leopold, D. R., Fredrick, J. W., Smith, Z. R., Jacobson, L. A., Burns, G. L., Mayes, S. D., Waschbusch, D. A., Froehlich, T. E., McBurnett, K., Servera, M., & Barkley, R. A. (2023). Report of a work group on sluggish cognitive tempo: Key research directions and a consensus change in terminology to cognitive disengagement syndrome. *Journal of the American Academy of Child & Adolescent Psychiatry*, *62*(6), 629–645. https://doi.org/10.1016/j.jaac.2022.07.821

Bell, Z., Shader, T., Webster-Stratton, C., Reid, M. J., & Beauchaine, T. P. (2018). Improvements in negative parenting mediate changes in children's autonomic responding following a preschool intervention for ADHD. *Clinical Psychological Science*, *6*(1), 134–144. https://doi.org/10.1177/2167702617727559

Bella-Fernández, M., Martin-Moratinos, M., Li, C., Wang, P., & Blasco-Fontecilla, H. (2023). Differences in ex-gaussian parameters from response time distributions between individuals with and without attention deficit/hyperactivity disorder: A meta-analysis. *Neuropsychology Review*. https://doi.org/10.1007/s11065-023-09587-2

Bellato, A., Arora, I., Hollis, C., & Groom, M. J. (2020). Is autonomic nervous system function atypical in attention deficit hyperactivity disorder (ADHD)? A systematic review of the evidence. *Neuroscience & Biobehavioral Reviews*, *108*, 182–206.

https://doi.org/10.1016/j.neubiorev.2019.11.001

Benedict, F. T., Vivier, P. M., & Gjelsvik, A. (2015). Mental health and bullying in the United States among children aged 6 to 17 years. *Journal of Interpersonal Violence*, *30*(5), 782–795. https://doi.org/10.1177/0886260514536279

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society. Series B (Methodological)*, *57*(1), 289–300. http://www.jstor.org/stable/10.2307/2346101

Berger, A., & Posner, M. I. (2000). Pathologies of brain attentional networks. *Neuroscience and Biobehavioral Reviews*, *24*(1), 3–5. https://doi.org/10.1016/S0149-7634(99)00046-9

Bernanke, J., Luna, A., Chang, L., Bruno, E., Dworkin, J., & Posner, J. (2022). Structural brain measures among children with and without ADHD in the Adolescent Brain and Cognitive Development Study cohort: A cross-sectional US population-based study. *The Lancet Psychiatry*, *9*(3), 222–231. https://doi.org/10.1016/S2215-0366(21)00505-8

Birchwood, J., & Daley, D. (2012). Brief report: The impact of attention deficit hyperactivity disorder (ADHD) symptoms on academic performance in an adolescent community sample. *Journal of Adolescence*, *35*(1), 225–231. https://doi.org/10.1016/j.adolescence.2010.08.011

Bluschke, A., Roessner, V., & Beste, C. (2016). Editorial perspective: How to optimise frequency band neurofeedback for ADHD. *Journal of Child Psychology and Psychiatry*, 57(4), 457–461. https://doi.org/10.1111/jcpp.12521

Booth, J. E., Carlson, C. L., & Tucker, D. M. (2007). Performance on a neurocognitive measure of alerting differentiates ADHD combined and inattentive subtypes: A preliminary report. *Archives of Clinical Neuropsychology*, *22*(4), 423–432.

https://doi.org/10.1016/j.acn.2007.01.017

Borsboom, D., Cramer, A. O. J., & Kalis, A. (2019). Brain disorders? Not really: Why network structures block reductionism in psychopathology research. *Behavioral and Brain Sciences*, *42*, Article e2. https://doi.org/10.1017/S0140525X17002266

Bosnjak, M., Fiebach, C. J., Mellor, D., Mueller, S., O'Connor, D. B., Oswald, F. L., & Sokol, R. I. (2022). A template for preregistration of quantitative research in psychology: Report of the joint psychological societies preregistration task force. *The American Psychologist*, *77*(4), 602–615. https://doi.org/10.1037/amp0000879

Botvinick, M., & Braver, T. (2015). Motivation and cognitive control: From behavior to neural mechanism. *Annual Review of Psychology*, *66*, 83–113. https://doi.org/10.1146/annurev-psych-010814-015044

Breslau, J., Miller, E., Joanie Chung, W.-J., & Schweitzer, J. B. (2011). Childhood and adolescent onset psychiatric disorders, substance use, and failure to graduate high school on time. *Journal of Psychiatric Research*, *45*(3), 295–301. https://doi.org/10.1016/j.jpsychires.2010.06.014

Brodeur, D. A., & Pond, M. (2001). The development of selective attention in children with attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*, *29*(3), 229–239. https://doi.org/10.1023/A:1010381731658

Buchhorn, R., Koenig, J., Jarczok, M. N., Eichholz, H., Willaschek, C., Thayer, J. F., & Kaess, M. (2018). A case series on the potential effect of omega-3-fatty acid supplementation on 24-h heart rate variability and its circadian variation in children with attention deficit (hyperactivity) disorder. *ADHD Attention Deficit and Hyperactivity Disorders*, *10*(2), 135–139. https://doi.org/10.1007/s12402-017-0240-y

Bueno, V. F., Kozasa, E. H., da Silva, M. A., Alves, T. M., Louzã, M. R., & Pompéia, S. (2015). Mindfulness Meditation Improves Mood, Quality of Life, and Attention in Adults with

Attention Deficit Hyperactivity Disorder. *BioMed Research International*, *2015*, Article 962857. https://doi.org/10.1155/2015/962857

Burt, S. A. (2009). Rethinking environmental contributions to child and adolescent psychopathology: A meta-analysis of shared environmental influences. *Psychological Bulletin*, *135*(4), 608–637. https://doi.org/10.1037/a0015702

Bush, G. (2010). Attention-deficit/hyperactivity disorder and attention networks. *Neuropsychopharmacology*, *35*(1), 278–300. https://doi.org/10.1038/npp.2009.120

Callejas, A., Lupiàñez, J., Funes, M. J., & Tudela, P. (2005). Modulations among the alerting, orienting and executive control networks. *Experimental Brain Research*, *167*(1), 27–37. https://doi.org/10.1007/s00221-005-2365-z

Callejas, A., Lupiáñez, J., & Tudela, P. (2004). The three attentional networks: On their independence and interactions. *Brain and Cognition*, *54*(3), 225–227. https://doi.org/10.1016/j.bandc.2004.02.012

Casagrande, M., Martella, D., Ruggiero, M. C., MacCari, L., Paloscia, C., Rosa, C., & Pasini, A. (2012). Assessing attentional systems in children with attention deficit hyperactivity disorder. *Archives of Clinical Neuropsychology*, *27*(1), 30–44. https://doi.org/10.1093/arclin/acr085

Cásedas, L., Pirruccio, V., Vadillo, M. A., & Lupiáñez, J. (2020). Does mindfulness meditation training enhance executive control? A systematic review and meta-analysis of randomized controlled trials in adults. *Mindfulness*, *11*(2), 411–424. https://doi.org/10.1007/s12671-019-01279-4

Cásedas, L., Cebolla, A., & Lupiáñez, J. (2022). Individual differences in dispositional mindfulness predict attentional networks and vigilance performance. *Mindfulness*, *13*(4), 967–981. https://doi.org/10.1007/s12671-022-01850-6

Cásedas, L., & Lupiáñez, J. (2023). *Overview of the CEM Framework* [Unpublished

manuscript]. Department of Experimental Psychology, University of Granada.

Castellanos, F. X., Sonuga-Barke, E. J. S., Milham, M. P., & Tannock, R. (2006). Characterizing cognition in ADHD: Beyond executive dysfunction. *Trends in Cognitive Sciences*, *10*(3), 117–123. https://doi.org/10.1016/J.TICS.2006.01.011

Castellanos, F. X., & Tannock, R. (2002). Neuroscience of attention-deficit/hyperactivity disorder: The search for endophenotypes. *Nature Reviews Neuroscience*, *3*(8), 617–628. https://doi.org/10.1038/nrn896

Catalá-López, F., Hutton, B., Page, M. J., Driver, J. A., Ridao, M., Alonso-Arroyo, A., Valencia, A., Macías Saint-Gerons, D., & Tabarés-Seisdedos, R. (2022). Mortality in persons with autism spectrum disorder or attention-deficit/hyperactivity disorder: A systematic review and meta-analysis. *JAMA Pediatrics*, *176*(4), Article e216401. https://doi.org/10.1001/JAMAPEDIATRICS.2021.6401

Caye, A., Rocha, T. B.-M., Anselmi, L., Murray, J., Menezes, A. M. B., Barros, F. C., Gonçalves, H., Wehrmeister, F., Jensen, C. M., Steinhausen, H.-C., Swanson, J. M., Kieling, C., & Rohde, L. A. (2016). Attention-deficit/hyperactivity disorder trajectories from childhood to young adulthood: Evidence from a birth cohort supporting a late-onset syndrome. *JAMA Psychiatry*, *73*(7), 705–712. https://doi.org/10.1001/jamapsychiatry.2016.0383

Caye, A., Sibley, M. H., Swanson, J. M., & Rohde, L. A. (2017). Late-onset ADHD: Understanding the evidence and building theoretical frameworks. *Current Psychiatry Reports*, *19*(12), Article 106. https://doi.org/10.1007/s11920-017-0858-7

Chan, E., Mattingley, J. B., Huang-Pollock, C., English, T., Hester, R., Vance, A., & Bellgrove, M. A. (2009). Abnormal spatial asymmetry of selective attention in ADHD. *Journal of Child Psychology and Psychiatry*, *50*(9), 1064–1072. https://doi.org/10.1111/j.1469-7610.2009.02096.x

Chang, J. P. C., Su, K. P., Mondelli, V., & Pariante, C. M. (2018). Omega-3 polyunsaturated fatty acids in youths with attention deficit hyperactivity disorder: A systematic review and meta-analysis of clinical trials and biological studies. *Neuropsychopharmacology*, *43*(3), 534–545. https://doi.org/10.1038/npp.2017.160

Chang, Z., Lichtenstein, P., Asherson, P. J., & Larsson, H. (2013). Developmental twin study of attention problems: High heritabilities throughout development. *JAMA Psychiatry*, *70*(3), 311–318. https://doi.org/10.1001/jamapsychiatry.2013.287

Chang, Z., Lichtenstein, P., D'Onofrio, B. M., Sjölander, A., & Larsson, H. (2014). Serious transport accidents in adults with attention-deficit/hyperactivity disorder and the effect of medication: A population-based study. *JAMA Psychiatry*, *71*(3), 319–325. https://doi.org/10.1001/jamapsychiatry.2013.4174

Charach, A., & Fernandez, R. (2013). Enhancing ADHD medication adherence: Challenges and opportunities. *Current Psychiatry Reports*, *15*(7), Article 371. https://doi.org/10.1007/s11920-013-0371-6

Cheesman, R., Eilertsen, E. M., Ayorech, Z., Borgen, N. T., Andreassen, O. A., Larsson, H., Zachrisson, H., Torvik, F. A., & Ystrom, E. (2021). How interactions between ADHD and schools affect educational achievement: A family-based genetically sensitive study. *Journal of Child Psychology and Psychiatry*, *63*(10), 1174–1185. https://doi.org/10.1111/jcpp.13656

Chen, Q., Hartman, C. A., Haavik, J., Harro, J., Klungsøyr, K., Hegvik, T.-A., Wanders, R., Ottosen, C., Dalsgaard, S., & Faraone, S. V. (2018). Common psychiatric and metabolic comorbidity of adult attention-deficit/hyperactivity disorder: A population-based cross-sectional study. *PLOS ONE*, *13*(9), Article e0204516. https://doi.org/10.1371/journal.pone.0204516

Chhibber, A., Watanabe, A. H., Chaisai, C., Veettil, S. K., & Chaiyakunapruk, N. (2021).

Global economic burden of attention-deficit/hyperactivity disorder: A systematic review. *PharmacoEconomics*, *39*(4), 399–420. https://doi.org/10.1007/s40273-020-00998-0

Claypoole, V. L., Neigel, A. R., Fraulini, N. W., Hancock, G. M., & Szalma, J. L. (2018). Can vigilance tasks be administered online? A replication and discussion. *Journal of Experimental Psychology: Human Perception and Performance*, *44*(9), 1348–1355. https://doi.org/10.1037/xhp0000538

Coghill, D. R., Hayward, D., Rhodes, S. M., Grimmer, C., & Matthews, K. (2014). A longitudinal examination of neuropsychological and clinical functioning in boys with attention deficit hyperactivity disorder (ADHD): Improvements in executive functioning do not explain clinical improvement. *Psychological Medicine*, *44*(5), 1087–1099. https://doi.org/10.1017/S0033291713001761

Coghill, D. R., Seth, S., & Matthews, K. (2014). A comprehensive assessment of memory, delay aversion, timing, inhibition, decision making and variability in attention deficit hyperactivity disorder: Advancing beyond the three-pathway models. *Psychological Medicine*, *44*(9), 1989–2001. https://doi.org/10.1017/S0033291713002547

Coghill, D., & Sonuga-Barke, E. J. S. (2012). Annual research review: Categories versus dimensions in the classification and conceptualisation of child and adolescent mental disorders – implications of recent empirical study. *Journal of Child Psychology and Psychiatry*, *53*(5), 469–489. https://doi.org/10.1111/j.1469-7610.2011.02511.x

Cohen, A. L., & Shapiro, S. K. (2007). Exploring the performance differences on the flicker task and the Conners' Continuous Performance Test in adults with ADHD. *Journal of Attention Disorders*, *11*(1), 49–63. https://doi.org/10.1177/1087054706292162

Cohen, S. C. L., Harvey, D. J., Shields, R. H., Shields, G. S., Rashedi, R. N., Tancredi, D. J., Angkustsiri, K., Hansen, R. L., & Schweitzer, J. B. (2018). Effects of yoga on attention,

impulsivity, and hyperactivity in preschool-aged children with attention-deficit hyperactivity disorder symptoms. *Journal of Developmental & Behavioral Pediatrics*, *39*(3), 200–209. https://doi.org/10.1097/DBP.0000000000000552

Coll-Martín, T., Carretero-Dios, H., & Lupiáñez, J. (2021). Attentional networks, vigilance, and distraction as a function of attention-deficit/hyperactivity disorder symptoms in an adult community sample. *British Journal of Psychology*, *112*(4), 1–27. https://doi.org/10.1111/bjop.12513

Coll-Martín, T., Carretero-Dios, H., & Lupiáñez, J. (2023). *Attention-deficit/hyperactivity disorder symptoms as function of arousal and executive vigilance: Testing the Halperin and Schulz's neurodevelopmental model in an adult community sample*. PsyArXiv. https://doi.org/10.31234/osf.io/jzk7b

Coll-Martín, T., Carretero-Dios, H., & Lupiáñez, J. (2019, November 14–16). *Puede el entrenamiento en autorregulación mediante juego estructurado reducir los síntomas de TDAH en preescolares? Una revisión sistemática y meta-análisis* [Can self-regulation training through structured play reduce ADHD symptoms in preschoolers? A systematic review and meta-analysis] [Conference presentation abstract]. Fifth International Congress of Clinical and Health Psychology on Children and Adolescents, Oviedo, Spain. http://www.aitanacongress.com/2019/wp-content/uploads/2019/10/Abstracts_2019.pdf

Coll-Martín, T., Carretero-Dios, H., & Lupiáñez, J. (2022, September 13). Cuando el TDAH comienza en la adultez: ¿Un trastorno distinto? [When ADHD begins in adulthood: A distinct disorder?]. *Ciencia Cognitiva*, *16*(3), 61–64. https://www.cienciacognitiva.org/?p=2245

Coll-Martín, T., Evangelista, J., Hussain, Z., Cortese, S., Westwood, S., & Bellato, A. (2022). *The effects of non-pharmacological interventions for ADHD on indices of autonomic*

functioning: Systematic review and meta-analysis. PROSPERO. https://www.crd.york.ac.uk/prospero/display_record.php?RecordID=372965

Coll-Martín, T., Román-Caballero, R., Martínez-Caballero, M. del R., Martín-Sánchez, P. del C., Trujillo, L., Cásedas, L., Castellanos, M. C., Hemmerich, K., Manini, G., Aguirre, M. J., Botta, F., Marotta, A., Martín-Arévalo, E., Gabriel, L. F. G., & Lupiáñez, J. (2023). The ANTI-Vea-UGR platform: A free online resource to measure attentional networks (alertness, orienting, and executive control) functioning and executive/arousal vigilance. Journal of Intelligence, 11, Article 181. https://doi.org/10.3390/jintelligence11090181

Coll-Martín, T., Sonuga-Barke, E. J. S., Carretero-Dios, H., & Lupiáñez, J. (2023). *Are attentional processes differentially related to symptoms of ADHD in childhood, adulthood, and late onset? Evidence from a multi-sample of community adults* [Manuscript in preparation]. Department of Behavioral Sciences Methodology, University of Granada.

Combs, M. A., Canu, W. H., Broman-Fulks, J. J., Rocheleau, C. A., & Nieman, D. C. (2015). Perceived stress and ADHD symptoms in adults. *Journal of Attention Disorders*, *19*(5), 425–434. https://doi.org/10.1177/1087054712459558

Conners, C. K. (2000). *Conners' Continuous Performance Test II: Computer program for windows, technical guide, and software manual* (Multi-Health Systems Staff, Ed.). MultiHealth Systems Inc. https://www.scienceopen.com/document?vid=abe27624-215d-4220-af39-5e13ab893030

Cooper, M., Hammerton, G., Collishaw, S., Langley, K., Thapar, A., Dalsgaard, S., Stergiakouli, E., Tilling, K., Davey Smith, G., Maughan, B., O'Donovan, M., Thapar, A., & Riglin, L. (2018). Investigating late-onset ADHD: A population cohort investigation. *Journal of Child Psychology and Psychiatry*, *59*(10), 1105–1113.

https://doi.org/10.1111/JCPP.12911

Corbetta, M. (1998). Frontoparietal cortical networks for directing attention and the eye to visual locations: Identical, independent, or overlapping neural systems? *Proceedings of the National Academy of Sciences*, *95*(3), 831–838. https://doi.org/10.1073/pnas.95.3.831

Cortese, S. (2020). Pharmacologic treatment of attention deficit-hyperactivity disorder. *The New England Journal of Medicine*, *383*(11), 1050–1056. https://doi.org/10.1056/NEJMra1917069

Cortese, S., Adamo, N., Del Giovane, C., Mohr-Jensen, C., Hayes, A. J., Carucci, S., Atkinson, L. Z., Tessari, L., Banaschewski, T., Coghill, D., Hollis, C., Simonoff, E., Zuddas, A., Barbui, C., Purgato, M., Steinhausen, H.-C., Shokraneh, F., Xia, J., & Cipriani, A. (2018). Comparative efficacy and tolerability of medications for attention-deficit hyperactivity disorder in children, adolescents, and adults: A systematic review and network meta-analysis. *The Lancet. Psychiatry*, *5*(9), 727–738. https://doi.org/10.1016/S2215-0366(18)30269-4

Cortese, S., Solmi, M., Michelini, G., Bellato, A., Blanner, C., Canozzi, A., Eudave, L., Farhat, L. C., Højlund, M., Köhler-Forsberg, O., Leffa, D. T., Rohde, C., de Pablo, G. S., Vita, G., Wesselhoeft, R., Martin, J., Baumeister, S., Bozhilova, N. S., Carlisi, C. O., … Correll, C. U. (2023). Candidate diagnostic biomarkers for neurodevelopmental disorders in children and adolescents: A systematic review. *World Psychiatry*, *22*(1), 129–149. https://doi.org/10.1002/wps.21037

Craig, C. M., & Klein, M. I. (2019). The Abbreviated Vigilance Task and its attentional contributors. *Human Factors*, *61*(3), 426–439. https://doi.org/10.1177/0018720818822350

Cussen, A., Sciberras, E., Ukoumunne, O. C., & Efron, D. (2012). Relationship between

symptoms of attention-deficit/hyperactivity disorder and family functioning: A community-based study. *European Journal of Pediatrics*, *171*(2), 271–280. https://doi.org/10.1007/s00431-011-1524-4

Daley, D., van der Oord, S., Ferrin, M., Danckaerts, M., Doepfner, M., Cortese, S., & Sonuga-Barke, E. J. S. (2014). Behavioral interventions in attention-deficit/hyperactivity disorder: A meta-analysis of randomized controlled trials across multiple outcome domains. *Journal of the American Academy of Child & Adolescent Psychiatry*, *53*(8), 835-847.e5. https://doi.org/10.1016/J.JAAC.2014.05.013

Dang, J., King, K. M., & Inzlicht, M. (2020). Why Are Self-Report and Behavioral Measures Weakly Correlated? *Trends in Cognitive Sciences*, *24*(4), 267–269. https://doi.org/10.1016/j.tics.2020.01.007

de Souza Almeida, R., Faria, A., Jr., & Klein, R. M. (2021). On the origins and evolution of the Attention Network Tests. *Neuroscience and Biobehavioral Reviews*, *126*, 560–572. https://doi.org/10.1016/j.neubiorev.2021.02.028

De Zeeuw, P., Weusten, J., Van Dijk, S., Van Belle, J., & Durston, S. (2012). Deficits in cognitive control, timing and reward sensitivity appear to be dissociable in ADHD. *PLOS ONE*, *7*(12), Article e51416. https://doi.org/10.1371/journal.pone.0051416

DeBruine, L. (2021). *faux: Simulation for factorial designs* (1.1.0.) [Computer software]. Comprehensive R Archive Network. https://cran.r-project.org/web/packages/faux/index.html

Dekkers, T. J., Hornstra, R., van der Oord, S., Luman, M., Hoekstra, P. J., Groenman, A. P., & van den Hoofdakker, B. J. (2022). Meta-analysis: Which components of parent training work for children with attention-deficit/hyperactivity disorder? *Journal of the American Academy of Child and Adolescent Psychiatry*, *61*(4), 478–494. https://doi.org/10.1016/j.jaac.2021.06.015

Demontis, D., Walters, R. K., Martin, J., Mattheisen, M., Als, T. D., Agerbo, E., Baldursson, G., Belliveau, R., Bybjerg-Grauholm, J., Bækvad-Hansen, M., Cerrato, F., Chambert, K., Churchhouse, C., Dumont, A., Eriksson, N., Gandal, M., Goldstein, J. I., Grasby, K. L., Grove, J., … Neale, B. M. (2019). Discovery of the first genome-wide significant risk loci for attention deficit/hyperactivity disorder. *Nature Genetics*, *51*, 63–75. https://doi.org/10.1038/s41588-018-0269-7

Dinges, D. F., & Powell, J. W. (1985). Microcomputer analyses of performance on a portable, simple visual RT task during sustained operations. *Behavior Research Methods, Instruments, & Computers*, *17*(6), 652–655. https://doi.org/10.3758/BF03200977

Dinkler, L., Lundström, S., Gajwani, R., Lichtenstein, P., Gillberg, C., & Minnis, H. (2017). Maltreatment-associated neurodevelopmental disorders: A co-twin control analysis. *Journal of Child Psychology and Psychiatry*, *58*(6), 691–701. https://doi.org/10.1111/jcpp.12682

Doran, S. M., Dongen, H. P. A. V., & Dinges, D. F. (2001). Sustained attention performance during sleep deprivation: Evidence of state instability. *Archives Italiennes de Biologie*, *139*(3), 253–267. https://doi.org/10.4449/aib.v139i3.503

Dosenbach, N. U. F., Fair, D. A., Cohen, A. L., Schlaggar, B. L., & Petersen, S. E. (2008). A dual-networks architecture of top-down control. *Trends in Cognitive Sciences*, *12*(3), 99–105. https://doi.org/10.1016/j.tics.2008.01.001

Du Rietz, E., Cheung, C. H. M., McLoughlin, G., Brandeis, D., Banaschewski, T., Asherson, P., & Kuntsi, J. (2016). Self-report of ADHD shows limited agreement with objective markers of persistence and remittance. *Journal of Psychiatric Research*, *82*, 91–99. https://doi.org/10.1016/j.jpsychires.2016.07.020

Durston, S., Van Belle, J., & De Zeeuw, P. (2011). Differentiating frontostriatal and fronto-cerebellar circuits in attention-deficit/hyperactivity disorder. *Biological Psychiatry*,

69(12), 1178–1184. https://doi.org/10.1016/j.biopsych.2010.07.037

Eakin, L., Minde, K., Hechtman, L., Ochs, E., Krane, E., Bouffard, R., Greenfield, B., & Looper, K. (2004). The marital and family functioning of adults with ADHD and their spouses. *Journal of Attention Disorders*, *8*(1), 1–10. https://doi.org/10.1177/108705470400800101

Eisenberg, J., Ben-Daniel, N., Mei-Tal, G., & Wertman, E. (2004). An autonomic nervous system biofeedback modality for the treatment of attention deficit hyperactivity disorder—An open pilot study. *The Israel Journal of Psychiatry and Related Sciences*, *41*(1), 45–53. https://www.researchgate.net/publication/8545087_An_autonomic_nervous_system_biofeedback_modality_for_the_treatment_of_attention_deficit_hyperactivity_disorder_-_An_open_pilot_study

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, *196*(4286), 129–136. https://doi.org/10.1126/science.847460

Epstein, J. N., Conners, C. K., Sitarenios, G., & Erhardt, D. (1998). Continuous performance test results of adults with attention deficit hyperactivity disorder. *The Clinical Neuropsychologist*, *12*(2), 155–168. https://doi.org/10.1076/clin.12.2.155.2000

Epstein, J. N., Johnson, D. E., Varia, I. M., & Conners, C. K. (2001). Neuropsychological assessment of response inhibition in adults with ADHD. *Journal of Clinical and Experimental Neuropsychology*, *23*(3), 362–371. https://doi.org/10.1076/jcen.23.3.362.1186

Epstein, J. N., Langberg, J. M., Rosen, P. J., Graham, A., Narad, M. E., Antonini, T. N., Brinkman, W. B., Froehlich, T., Simon, J. O., & Altaye, M. (2011). Evidence for higher reaction time variability for children with ADHD on a range of cognitive tasks including reward and event rate manipulations. *Neuropsychology*, *25*(4), 427–441.

https://doi.org/10.1037/a0022155

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics*, *16*(1), 143–149. https://doi.org/10.3758/BF03203267

Ernst, M. D., & Hutson, A. D. (2003). Utilizing a quantile function approach to obtain exact bootstrap solutions. *Statistical Science*, *18*(2), 231–240. https://www.jstor.org/stable/3182853

Esterman, M., & Rothlein, D. (2019). Models of sustained attention. *Current Opinion in Psychology*, *29*, 174–180. https://doi.org/10.1016/j.copsyc.2019.03.005

Evans, S. W., Owens, J. S., Wymbs, B. T., & Ray, A. R. (2018). Evidence-based psychosocial treatments for children and adolescents with attention deficit/hyperactivity disorder. *Journal of Clinical Child & Adolescent Psychology*, *47*(2), 157–198. https://doi.org/10.1080/15374416.2017.1390757

Faheem, M., Akram, W., Akram, H., Khan, M. A., Siddiqui, F. A., & Majeed, I. (2022). Gender-based differences in prevalence and effects of ADHD in adults: A systematic review. *Asian Journal of Psychiatry*, *75*, 103205. https://doi.org/10.1016/j.ajp.2022.103205

Fair, D. A., Bathula, D., Nikolas, M. A., & Nigg, J. T. (2012). Distinct neuropsychological subgroups in typically developing youth inform heterogeneity in children with ADHD. *Proceedings of the National Academy of Sciences*, *109*(17), 6769–6774. https://doi.org/10.1073/pnas.1115365109

Fan, J., McCandliss, B. D., Sommer, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience*, *14*(3), 340–347. https://doi.org/10.1162/089892902317361886

Faraone, S. V., Asherson, P., Banaschewski, T., Biederman, J., Buitelaar, J. K., Ramos-

Quiroga, J. A., Rohde, L. A., Sonuga-Barke, E. J. S., Tannock, R., & Franke, B. (2015). Attention-deficit/hyperactivity disorder. *Nature Reviews Disease Primers*, *1*, Article 15020. https://doi.org/10.1038/nrdp.2015.20

Faraone, S. V., Banaschewski, T., Coghill, D., Zheng, Y., Biederman, J., Bellgrove, M. A., Newcorn, J. H., Gignac, M., Al Saud, N. M., Manor, I., Rohde, L. A., Yang, L., Cortese, S., Almagor, D., Stein, M. A., Albatti, T. H., Aljoudi, H. F., Alqahtani, M. M. J., Asherson, P., … Wang, Y. (2021). The World Federation of ADHD International Consensus Statement: 208 Evidence-based conclusions about the disorder. Neuroscience and Biobehavioral Reviews, 128, 789–818. https://doi.org/10.1016/j.neubiorev.2021.01.022

Faraone, S. V., & Biederman, J. (2016). Can attention-deficit/hyperactivity disorder onset occur in adulthood? *JAMA Psychiatry*, *73*(7), 655–656. https://doi.org/10.1001/jamapsychiatry.2016.0400

Faraone, S. V., Biederman, J., Doyle, A., Murray, K., Petty, C., Adamson, J. J., & Seidman, L. (2006). Neuropsychological studies of late onset and subthreshold diagnoses of adult attention-deficit/hyperactivity disorder. *Biological Psychiatry*, *60*(10), 1081–1087. https://doi.org/10.1016/j.biopsych.2006.03.060

Faraone, S. V., Biederman, J., & Mick, E. (2006). The age-dependent decline of attention deficit hyperactivity disorder: A meta-analysis of follow-up studies. *Psychological Medicine*, *36*(2), 159–165. https://doi.org/10.1017/S003329170500471X

Faraone, S. V., Biederman, J., Spencer, T., Mick, E., Murray, K., Petty, C., Adamson, J. J., & Monuteaux, M. C. (2006). Diagnosing adult attention deficit hyperactivity disorder: Are late onset and subthreshold diagnoses valid? *The American Journal of Psychiatry*, *163*(10), 1720–1859. https://doi.org/10.1176/ajp.2006.163.10.1720

Faraone, S. V., & Larsson, H. (2019). Genetics of attention deficit hyperactivity disorder.

Molecular Psychiatry, *24*(4), 562–575. https://doi.org/10.1038/s41380-018-0070-0

Fayyad, J., Sampson, N. A., Hwang, I., Adamowski, T., Aguilar-Gaxiola, S., Al-Hamzawi, A., Andrade, L. H. S. G., Borges, G., de Girolamo, G., Florescu, S., Gureje, O., Haro, J. M., Hu, C., Karam, E. G., Lee, S., Navarro-Mateu, F., O'Neill, S., Pennell, B.-E., Piazza, M., … World Health Organization World Mental Health Survey Collaborators. (2017). The descriptive epidemiology of DSM-IV Adult ADHD in the World Health Organization World Mental Health Surveys. *ADHD Attention Deficit and Hyperactivity Disorders*, *9*(1), 47–65. https://doi.org/10.1007/s12402-016-0208-3

Feltmate, B. B. T., Hurst, A. J., & Klein, R. M. (2020). Effects of fatigue on attention and vigilance as measured with a modified attention network test. *Experimental Brain Research*, *238*(11), 2507–2519. https://doi.org/10.1007/s00221-020-05902-y

Fernandez-Duque, D., & Posner, M. I. (2001). Brain imaging of attentional networks in normal and pathological states. *Journal of Clinical and Experimental Neuropsychology*, *23*(1), 74–93. https://doi.org/10.1076/jcen.23.1.74.1217

Fleming, M., Fitton, C. A., Steiner, M. F. C., McLay, J. S., Clark, D., King, A., Mackay, D. F., & Pell, J. P. (2017). Educational and health outcomes of children treated for attention-deficit/hyperactivity disorder. *JAMA Pediatrics*, *171*(7), Article e170691. https://doi.org/10.1001/jamapediatrics.2017.0691

Fletcher, J. M. (2014). The effects of childhood ADHD on adult labor market outcomes. *Health Economics*, *23*(2), 159–181. https://doi.org/10.1002/hec.2907

Forster, S. (2013). Distraction and mind-wandering under load. *Frontiers in Psychology*, *4*, Article 283. https://doi.org/10.3389/fpsyg.2013.00283

Forster, S., & Lavie, N. (2008). Failures to ignore entirely irrelevant distractors: The role of load. *Journal of Experimental Psychology: Applied*, *14*(1), 73–83. https://doi.org/10.1037/1076-898X.14.1.73

Forster, S., & Lavie, N. (2016). Establishing the attention-distractibility trait. *Psychological Science*, *27*(2), 203–212. https://doi.org/10.1177/0956797615617761

Forster, S., Robertson, D. J., Jennings, A., Asherson, P., & Lavie, N. (2014). Plugging the attention deficit: Perceptual load counters Increased distraction in ADHD. *Neuropsychology*, *28*(1), 91–97. https://doi.org/10.1037/neu0000020

Fortenbaugh, F. C., DeGutis, J., Germine, L., Wilmer, J. B., Grosso, M., Russo, K., & Esterman, M. (2015). Sustained attention across the life span in a sample of 10,000: Dissociating ability and strategy. *Psychological Science*, *26*(9), 1497–1510. https://doi.org/10.1177/0956797615594896

Franke, B. (2023). Editorial: It is time to modernize the concept of ADHD! *Journal of Child Psychology and Psychiatry*, *64*(6), 845–847. https://doi.org/10.1111/jcpp.13812

Franke, B., Michelini, G., Asherson, P., Banaschewski, T., Bilbow, A., Buitelaar, J. K., Cormand, B., Faraone, S. V., Ginsberg, Y., Haavik, J., Kuntsi, J., Larsson, H., Lesch, K.-P., Ramos-Quiroga, J. A., Réthelyi, J. M., Ribases, M., & Reif, A. (2018). Live fast, die young? A review on the developmental trajectories of ADHD across the lifespan. *European Neuropsychopharmacology*, *28*(10), 1059–1088. https://doi.org/10.1016/j.euroneuro.2018.08.001

Frazier, T. W., Youngstrom, E. A., & Naugle, R. I. (2007). The latent structure of attention-deficit/hyperactivity disorder in a clinic-referred sample. *Neuropsychology*, *21*(1), 45–64. https://doi.org/10.1037/0894-4105.21.1.45

Gabriely, R., Tarrasch, R., Velicki, M., & Ovadia-Blechman, Z. (2020). The influence of mindfulness meditation on inattention and physiological markers of stress on students with learning disabilities and/or attention deficit hyperactivity disorder. *Research in Developmental Disabilities*, *100*, Article 103630. https://doi.org/10.1016/j.ridd.2020.103630

Galéra, C., Bouvard, M.-P., Lagarde, E., Michel, G., Touchette, E., Fombonne, E., & Melchior, M. (2012). Childhood attention problems and socioeconomic status in adulthood: 18-year follow-up. *The British Journal of Psychiatry*, *201*(1), 20–25. https://doi.org/10.1192/bjp.bp.111.102491

Gathercole, S. E., Dunning, D. L., Holmes, J., & Norris, D. (2019). Working memory training involves learning new skills. *Journal of Memory and Language*, *105*, 19–42. https://doi.org/10.1016/j.jml.2018.10.003

Geissler, J., Romanos, M., Hegerl, U., & Hensch, T. (2014). Hyperactivity and sensation seeking as autoregulatory attempts to stabilize brain arousal in ADHD and mania? *ADHD Attention Deficit and Hyperactivity Disorders*, *6*(3), 159–173. https://doi.org/10.1007/s12402-014-0144-z

Germine, L., Nakayama, K., Duchaine, B. C., Chabris, C. F., Chatterjee, G., & Wilmer, J. B. (2012). Is the Web as good as the lab? Comparable performance from Web and lab in cognitive/perceptual experiments. *Psychonomic Bulletin & Review*, *19*(5), 847–857. https://doi.org/10.3758/s13423-012-0296-9

Gidziela, A., Ahmadzadeh, Y. I., Michelini, G., Allegrini, A. G., Agnew-Blais, J., Lau, L. Y., Duret, M., Procopio, F., Daly, E., Ronald, A., Rimfeld, K., & Malanchini, M. (2023). A meta-analysis of genetic effects associated with neurodevelopmental disorders and co-occurring conditions. *Nature Human Behaviour*, *7*(4), Article 4. https://doi.org/10.1038/s41562-023-01530-y

Gignac, G. E., & Szodorai, E. T. (2016). Effect size guidelines for individual differences researchers. *Personality and Individual Differences*, *102*, 74–78. https://doi.org/10.1016/j.paid.2016.06.069

Gilpin, A. R. (1993). Table for conversion of Kendall'S tau to Spearman'S rho within the context of measures of magnitude of effect for meta-analysis. *Educational and*

Psychological Measurement, *53*(1), 87–92. https://doi.org/10.1177/0013164493053001007

Gjone, H., Stevenson, J., & Sundet, J. M. (1996). Genetic influence on parent-reported attention-related problems in a Norwegian general population twin sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, *35*(5), 588–598. https://doi.org/10.1097/00004583-199605000-00013

Gmehlin, D., Fuermaier, A. B. M., Walther, S., Tucha, L., Koerts, J., Lange, K. W., Tucha, O., Weisbrod, M., & Aschenbrenner, S. (2016). Attentional lapses of adults with attention deficit hyperactivity disorder in tasks of sustained attention. *Archives of Clinical Neuropsychology*, *31*(4), 343–357. https://doi.org/10.1093/arclin/acw016

Greenberg, L. M., & Waldman, I. D. (1993). Developmental normative data on the test of variables of attention (T.O.V.A.). *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *34*(6), 1019–1030. https://doi.org/10.1111/j.1469-7610.1993.tb01105.x

Greene, R. W., Beszterczey, S. K., Katzenstein, T., Park, K., & Goring, J. (2002). Are students with ADHD more stressful to teach? Patterns of teacher stress in an elementary school sample. *Journal of Emotional and Behavioral Disorders*, *10*(2), 79–89. https://doi.org/10.1177/10634266020100020201

Gudjonsson, G. H., Sigurdsson, J. F., Smari, J., & Young, S. (2009). The relationship between satisfaction with life, ADHD symptoms, and associated problems among university students. *Journal of Attention Disorders*, *12*(6), 507–515. https://doi.org/10.1177/1087054708323018

Guimarães-da-Silva, P. O., Silva, K. L., Grevet, E. H., Salgado, C. A. I., Karam, R. G., Victor, M. M., Vitola, E. S., Mota, N. R., Fischer, A. G., Picon, F. A., Bertuzzi, G. P., Polina, E. R., Rohde, L. A., Belmonte-de-Abreu, P., & Bau, C. H. D. (2012). Does age of onset

of impairment impact on neuropsychological and personality features of adult ADHD? *Journal of Psychiatric Research*, *46*(10), 1307–1311. https://doi.org/10.1016/j.jpsychires.2012.06.010

Haan, E., Westmoreland, K. E., Schellhas, L., Sallis, H. M., Taylor, G., Zuccolo, L., & Munafò, M. R. (2022). Prenatal smoking, alcohol and caffeine exposure and offspring externalizing disorders: A systematic review and meta-analysis. *Addiction*, *117*(10), 2602–2613. https://doi.org/10.1111/add.15858

Hadfield, J. D., Wilson, A. J., Garant, D., Sheldon, B. C., & Kruuk, L. E. (2010). The misuse of BLUP in ecology and evolution. *The American Naturalist*, *175*(1), 116–125. https://doi.org/10.1086/648604

Halperin, J. M., Bédard, A. C. V., & Curchack-Lichtin, J. T. (2012). Preventive interventions for ADHD: A neurodevelopmental perspective. *Neurotherapeutics*, *9*(3), 531–541. https://doi.org/10.1007/s13311-012-0123-z

Halperin, J. M., & Schulz, K. P. (2006). Revisiting the role of the prefrontal cortex in the pathophysiology of attention-deficit/hyperactivity disorder. *Psychological Bulletin*, *132*(4), 560–581. https://doi.org/10.1037/0033-2909.132.4.560

Halperin, J. M., Trampush, J. W., Miller, C. J., Marks, D. J., & Newcorn, J. H. (2008). Neuropsychological outcome in adolescents/young adults with childhood ADHD: Profiles of persisters, remitters and controls. *Journal of Child Psychology and Psychiatry*, *49*(9), 958–966. https://doi.org/10.1111/j.1469-7610.2008.01926.x

Harrison, X. A., Donaldson, L., Correa-Cano, M. E., Evans, J., Fisher, D. N., Goodwin, C. E. D., Robinson, B. S., Hodgson, D. J., & Inger, R. (2018). A brief introduction to mixed effects modelling and multi-model inference in ecology. *PeerJ*, *6*, Article e4794. https://doi.org/10.7717/peerj.4794

Haslam, N., McGrath, M. J., Viechtbauer, W., & Kuppens, P. (2020). Dimensions over

categories: A meta-analysis of taxometric research. *Psychological Medicine*, *50*(9), 1418–1432. https://doi.org/10.1017/S003329172000183X

Haslam, N., Williams, B., Prior, M., Haslam, R., Graetz, B., & Sawyer, M. (2006). The latent structure of attention-deficit/hyperactivity disorder: A taxometric analysis. *Australian and New Zealand Journal of Psychiatry*, *40*(8), 639–647. https://doi.org/10.1080/j.1440-1614.2006.01863.x

Hasler, R., Perroud, N., Meziane, H. B., Herrmann, F., Prada, P., Giannakopoulos, P., & Deiber, M.-P. (2016). Attention-related EEG markers in adult ADHD. *Neuropsychologia*, *87*, 120–133. https://doi.org/10.1016/j.neuropsychologia.2016.05.008

Hedge, C., Powell, G., & Sumner, P. (2018). The reliability paradox: Why robust cognitive tasks do not produce reliable individual differences. *Behavior Research Methods*, *50*(3), 1166–1186. https://doi.org/10.3758/s13428-017-0935-1

Hemmerich, K., Luna, F. G., Lupiáñez, J., & Martín-Arévalo, E. (2020, October 28). Estimulación eléctrica transcraneal: Funcionamiento y usos en investigación [Transcranial electrical stimulation: Operation and uses in research]. *Ciencia Cognitiva*, *14*(3), 72–75. https://www.cienciacognitiva.org/?p=2007

Hemmerich, K., Lupiáñez, J., Luna, F. G., & Martín-Arévalo, E. (2023). The mitigation of the executive vigilance decrement via HD-tDCS over the right posterior parietal cortex and its association with neural oscillations. *Cerebral Cortex*, *33*(11), 6761–6771. https://doi.org/10.1093/cercor/bhac540

Hilger, K., & Fiebach, C. J. (2019). ADHD symptoms are associated with the modular structure of intrinsic brain networks in a representative sample of healthy adults. *Network Neuroscience*, *3*(2), 567–588. https://doi.org/10.1162/netn_a_00083

Hilger, K., Sassenhagen, J., Kühnhausen, J., Reuter, M., Schwarz, U., Gawrilow, C., &

Fiebach, C. J. (2020). Neurophysiological markers of ADHD symptoms in typically-developing children. *Scientific Reports*, *10*(1), Article 22460. https://doi.org/10.1038/s41598-020-80562-0

Hinshaw, S. P. (2018). Attention deficit hyperactivity disorder (ADHD): Controversy, developmental mechanisms, and multiple levels of analysis. *Annual Review of Clinical Psychology*, *14*(1), 291–316. https://doi.org/10.1146/annurev-clinpsy-050817-084917

Hommel, B., Chapman, C. S., Cisek, P., Neyedli, H. F., Song, J.-H., & Welsh, T. N. (2019). No one knows what attention is. *Attention, Perception, & Psychophysics*, *81*(7), 2288–2303. https://doi.org/10.3758/s13414-019-01846-w

Huang-Pollock, C. L., Karalunas, S. L., Tam, H., & Moore, A. N. (2012). Evaluating vigilance deficits in ADHD: A meta-analysis of CPT performance. *Journal of Abnormal Psychology*, *121*(2), 360–371. https://doi.org/10.1037/a0027205

Huang-Pollock, C. L., & Nigg, J. T. (2003). Searching for the attention deficit in attention deficit hyperactivity disorder: The case of visuospatial orienting. *Clinical Psychology Review*, *23*(6), 801–830. https://doi.org/10.1016/S0272-7358(03)00073-4

Huang-Pollock, C. L., Nigg, J. T., & Carr, T. H. (2005). Deficient attention is hard to find: Applying the perceptual load model of selective attention to attention deficit hyperactivity disorder subtypes. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *46*(11), 1211–1218. https://doi.org/10.1111/j.1469-7610.2005.00410.x

Huang-Pollock, C., Ratcliff, R., McKoon, G., Roule, A., Warner, T., Feldman, J., & Wise, S. (2020). A diffusion model analysis of sustained attention in children with attention deficit hyperactivity disorder. *Neuropsychology*, *34*(6), 641–653. https://doi.org/10.1037/neu0000636

Huertas, F., Ballester, R., Gines, H. J., Hamidi, A. K., Moratal, C., & Lupiáñez, J. (2019).

Relative age effect in the sport environment. Role of physical fitness and cognitive function in youth soccer players. *International Journal of Environmental Research and Public Health*, *16*(16), Article 2837. https://doi.org/10.3390/ijerph16162837

Idrees, I., Bellato, A., Cortese, S., & Groom, M. J. (2023). The effects of stimulant and non-stimulant medications on the autonomic nervous system (ANS) functioning in people with ADHD: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, *144*, Article 104968. https://doi.org/10.1016/j.neubiorev.2022.104968

Ilbegi, S., Buitelaar, J. K., Hoekstra, P. J., Hartman, C. A., Franke, B., Faraone, S. V., Oosterlaan, J., Luman, M., van Lieshout, M., & Rommelse, N. N. J. (2021). Neurocognitive markers of late-onset ADHD: A 6-year longitudinal study. *Journal of Child Psychology and Psychiatry*, *62*(2), 244–252. https://doi.org/10.1111/jcpp.13272

Inzlicht, M., Shenhav, A., & Olivola, C. Y. (2018). The effort paradox: Effort is both costly and valued. *Trends in Cognitive Sciences*, *22*(4), 337–349. https://doi.org/10.1016/j.tics.2018.01.007

Ishigami, Y., Eskes, G. A., Tyndall, A. V., Longman, R. S., Drogos, L. L., & Poulin, M. J. (2016). The Attention Network Test-Interaction (ANT-I): Reliability and validity in healthy older adults. *Experimental Brain Research*, *234*(3), 815–827. https://doi.org/10.1007/s00221-015-4493-4

Ishigami, Y., & Klein, R. M. (2010). Repeated measurement of the components of attention using two versions of the Attention Network Test (ANT): Stability, isolability, robustness, and reliability. *Journal of Neuroscience Methods*, *190*(1), 117–128. https://doi.org/10.1016/j.jneumeth.2010.04.019

JASP Team. (2020). *JASP* (Version 0.13) [Computer software]. https://jasp-stats.org/

Johnson, D. E., Epstein, J. N., Waid, L. R., Latham, P. K., Voronin, K. E., & Anton, R. F. (2001). Neuropsychological performance deficits in adults with attention

deficit/hyperactivity disorder. *Archives of Clinical Neuropsychology*, *16*(6), 587–604. https://doi.org/10.1016/S0887-6177(00)00070-6

Johnston, S.-K., Hennessey, N. W., & Leitão, S. (2019). Determinants of assessing efficiency within auditory attention networks. *The Journal of General Psychology*, *146*(2), 134–169. https://doi.org/10.1080/00221309.2018.1541861

Johnstone, S. J., Roodenrys, S., Phillips, E., Watt, A. J., & Mantz, S. (2010). A pilot study of combined working memory and inhibition training for children with AD/HD. *ADHD Attention Deficit and Hyperactivity Disorders*, *2*(1), 31–42. https://doi.org/10.1007/s12402-009-0017-z

Jonsson, U., Alaie, I., Löfgren Wilteus, A., Zander, E., Marschik, P. B., Coghill, D., & Bölte, S. (2017). Annual Research Review: Quality of life and childhood mental and behavioural disorders - a critical review of the research. *Journal of Child Psychology and Psychiatry*, *58*(4), 439–469. https://doi.org/10.1111/jcpp.12645

Kennedy, M., Kreppner, J., Knights, N., Kumsta, R., Maughan, B., Golm, D., Rutter, M., Schlotz, W., & Sonuga-Barke, E. J. S. (2016). Early severe institutional deprivation is associated with a persistent variant of adult attention-deficit/hyperactivity disorder: Clinical presentation, developmental continuities and life circumstances in the English and Romanian Adoptees study. *Journal of Child Psychology and Psychiatry*, *57*(10), 1113–1125. https://doi.org/10.1111/jcpp.12576

Knouse, L. E., Teller, J., & Brooks, M. A. (2017). Meta-analysis of cognitive–behavioral treatments for adult ADHD. *Journal of Consulting and Clinical Psychology*, *85*(7), 737–750. https://doi.org/10.1037/ccp0000216

Kofler, M. J., Rapport, M. D., Sarver, D. E., Raiker, J. S., Orban, S. A., Friedman, L. M., & Kolomeyer, E. G. (2013). Reaction time variability in ADHD: A meta-analytic review of 319 studies. *Clinical Psychology Review*, *33*(6), 795–811.

https://doi.org/10.1016/j.cpr.2013.06.001

Kolk, S. M., & Rakic, P. (2022). Development of prefrontal cortex. *Neuropsychopharmacology*, *47*(1), Article 1. https://doi.org/10.1038/s41386-021-01137-9

Korbmacher, M., Azevedo, F., Pennington, C. R., Hartmann, H., Pownall, M., Schmidt, K., Elsherif, M., Breznau, N., Robertson, O., Kalandadze, T., Yu, S., Baker, B. J., O'Mahony, A., Olsnes, J. Ø.-S., Shaw, J. J., Gjoneska, B., Yamada, Y., Röer, J. P., Murphy, J., … Evans, T. (2023). The replication crisis has led to positive structural, procedural, and community changes. *Communications Psychology*, *1*, Article 3. https://doi.org/10.1038/s44271-023-00003-2

Kosaka, H., Fujioka, T., & Jung, M. (2019). Symptoms in individuals with adult-onset ADHD are masked during childhood. *European Archives of Psychiatry and Clinical Neuroscience*, *269*(6), 753–755. https://doi.org/10.1007/s00406-018-0893-3

Lacey, J.I., 1967. Somatic response patterning and stress: Some revisions for activation theory. In M. M. Appley & R. Trumbull (Eds.), *Psychological stress* (pp. 14–36). Appleton-Century-Crofts.

Lakens, D. (2017). Equivalence tests: A practical primer for *t* tests, correlations, and meta-analyses. *Social Psychological and Personality Science*, *8*(4), 355–362. https://doi.org/10.1177/1948550617697177

Lakens, D. (2022). Sample size justification. *Collabra: Psychology*, *8*(1), Article 33267. https://doi.org/10.1525/collabra.33267

Lamond, N., Jay, S. M., Dorrian, J., Ferguson, S. A., Roach, G. D., & Dawson, D. (2008). The sensitivity of a palm-based psychomotor vigilance task to severe sleep loss. *Behavior Research Methods*, *40*(1), 347–352. https://doi.org/10.3758/brm.40.1.347

Lampe, K., Konrad, K., Kroener, S., Fast, K., Kunert, H. J., & Herpertz, S. C. (2007).

Neuropsychological and behavioural disinhibition in adult ADHD compared to borderline personality disorder. *Psychological Medicine*, *37*(12), 1717–1729. https://doi.org/10.1017/S0033291707000517

Langner, R., & Eickhoff, S. B. (2013). Sustaining attention to simple tasks: A meta-analytic review of the neural mechanisms of vigilant attention. *Psychological Bulletin*, *139*(4), 870–900. https://doi.org/10.1037/a0030694

Lee, S., Hill, T. R., Johnson, B., Testa, R., Priya, V., Spencer-smith, M., & Coghill, D. (2022). *Can neurocognitive outcomes assist measurement-based care for children with attention-deficit/hyperactivity disorder? A systematic review and meta-analyses of the relationships among the changes in neurocognitive functions and clinical outcomes of attention-deficit/hyperactivity disorder in pharmacological and cognitive training interventions*. *Journal of Child and Adolescent Psychopharmacology*, *32*(5), 250–277. https://doi.org/10.1089/cap.2022.0028

Leenders, A. E. M., Damatac, C. G., Soheili-Nezhad, S., Chauvin, R. J. M., Mennes, M. J. J., Zwiers, M. P., van Rooij, D., Akkermans, S. E. A., Naaijen, J., Franke, B., Buitelaar, J. K., Beckmann, C. F., & Sprooten, E. (2021). Associations between attention-deficit hyperactivity disorder (ADHD) symptom remission and white matter microstructure: A longitudinal analysis. *JCPP Advances*, *1*(3), Article e12040. https://doi.org/10.1002/jcv2.12040

Lensing, M. B., Zeiner, P., Sandvik, L., & Opjordsmoen, S. (2015). Quality of life in adults aged 50+ with ADHD. *Journal of Attention Disorders*, *19*(5), 405–413. https://doi.org/10.1177/1087054713480035

Lim, J., & Dinges, D. F. (2008). Sleep deprivation and vigilant attention. *Annals of the New York Academy of Sciences*, *1129*(1), 305–322. https://doi.org/10.1196/annals.1417.002

Loe, I. M., & Feldman, H. M. (2007). Academic and educational outcomes of children with

ADHD. *Journal of Pediatric Psychology*, *32*(6), 643–654. https://doi.org/10.1093/jpepsy/jsl054

Loh, S., Lamond, N., Dorrian, J., Roach, G., & Dawson, D. (2004). The validity of psychomotor vigilance tasks of less than 10-minute duration. *Behavior Research Methods, Instruments, & Computers*, *36*(2), 339–346. https://doi.org/10.3758/bf03195580

Lüdecke, D. (2022). *sjPlot: Data visualization for statistics in social science* (2.8.12) [Computer software]. Comprehensive R Archive Network. https://cran.r-project.org/web/packages/sjPlot/index.html

Lüdecke, D., Ben-Shachar, M. S., Patil, I., Waggoner, P., & Makowski, D. (2021). performance: An R package for assessment, comparison and testing of statistical models. *Journal of Open Source Software*, *6*(60), Article 3139. https://doi.org/10.21105/joss.03139

Ludyga, S., Gerber, M., Mücke, M., Brand, S., Weber, P., Brotzmann, M., & Pühse, U. (2020). The acute effects of aerobic exercise on cognitive flexibility and task-related heart rate variability in children with ADHD and healthy controls. *Journal of Attention Disorders*, *24*(5), 693–703. https://doi.org/10.1177/1087054718757647

Luna, F. G. (2019). *Redes atencionales y vigilancia ejecutiva y de activación* [Doctoral dissertation, University of Granada]. Digibug. https://digibug.ugr.es/handle/10481/56866

Luna, F. G., Barttfeld, P., Martín-Arévalo, E., & Lupiáñez, J. (2021). The ANTI-Vea task: Analyzing the executive and arousal vigilance decrements while measuring the three attentional networks. *Psicológica*, *42*, 1–26. https://doi.org/10.2478/psicolj-2021-0001

Luna, F. G., Marino, J., Roca, J., & Lupiáñez, J. (2018). Executive and arousal vigilance decrement in the context of the attentional networks: The ANTI-Vea task. *Journal of*

Neuroscience Methods, *306*, 77–87. https://doi.org/10.1016/J.JNEUMETH.2018.05.011

Luna, F. G., Roca, J., Martín-Arévalo, E., & Lupiáñez, J. (2021). Measuring attention and vigilance in the laboratory vs. online: The split-half reliability of the ANTI-Vea. *Behavior Research Methods*, *53*(3), 1124–1147. https://doi.org/10.3758/s13428-020-01483-4

Luna, F. G., Román-Caballero, R., Barttfeld, P., Lupiáñez, J., & Martín-Arévalo, E. (2020). A High-Definition tDCS and EEG study on attention and vigilance: Brain stimulation mitigates the executive but not the arousal vigilance decrement. *Neuropsychologia*, *142*, 107447. https://doi.org/10.1016/j.neuropsychologia.2020.107447

Luna, F. G., Telga, M., Vadillo, M. A., & Lupiáñez, J. (2020). Concurrent working memory load may increase or reduce cognitive interference depending on the attentional set. *Journal of Experimental Psychology: Human Perception and Performance*, *46*(7), 667–680. https://doi.org/10.1037/xhp0000740

Luna, F. G., Tortajada, M., Martín-Arévalo, E., Botta, F., & Lupiáñez, J. (2022). A vigilance decrement comes along with an executive control decrement: Testing the resource-control theory. *Psychonomic Bulletin & Review*, *29*(5), 1831–1843. https://doi.org/10.3758/s13423-022-02089-x

Lundervold, A. J., Adolfsdottir, S., Halleland, H., Halmøy, A., Plessen, K., & Haavik, J. (2011). Attention Network Test in adults with ADHD - the impact of affective fluctuations. *Behavioral and Brain Functions*, *7*(1), 27. https://doi.org/10.1186/1744-9081-7-27

Lundervold, A. J., Vartiainen, H., Jensen, D., & Haavik, J. (2021). Test-retest reliability of the 25-item version of Wender Utah Rating Scale. Impact of current ADHD severity on retrospectively assessed childhood symptoms. *Journal of Attention Disorders*, *25*(7), 1001–1009. https://doi.org/10.1177/1087054719879501

Luo, Y., Weibman, D., Halperin, J. M., & Li, X. (2019). A review of heterogeneity in attention deficit/hyperactivity disorder (ADHD). *Frontiers in Human Neuroscience*, *13*, Article 42. https://doi.org/10.3389/fnhum.2019.00042

Lupiáñez, J., Castellanos, M. C., Luna, F. G., Martín-Arévalo, E., Román-Caballero, R., Hemmerich, K., Coll-Martín, T., Marotta, A., Martella, D., & Botta, F. (2022, May 5). *Modulations of sleep deprivation and motivation on Executive / Arousal vigilance and executive control: An on-line sleep deprivation study with the ANTI-Vea task* [Conference presentation abstract]. International APPE-SEPEX Meeting, Faro, Portugal. https://appesepexmeeting.appe.pt/abstracts/

Mackworth, J. F. (1964). Performance decrement in vigilance, threshold, and high-speed perceptual motor tasks. *Canadian Journal of Psychology / Revue Canadienne de Psychologie*, *18*(3), 209–223. https://doi.org/10.1037/h0083302

Mackworth, N. H. (1948). The breakdown of vigilance during prolonged visual search. The *Quarterly Journal of Experimental Psychology*, *1*, 6–21. https://doi.org/10.1080/17470214808416738

MacLeod, J. W., Lawrence, M. A., McConnell, M. M., Eskes, G. A., Klein, R. M., & Shore, D. I. (2010). Appraising the ANT: Psychometric and theoretical considerations of the Attention Network Test. *Neuropsychology*, *24*(5), 637–651. https://doi.org/10.1037/a0019803

Makowski, D., Ben-Shachar, M. S., Patil, I., & Lüdecke, D. (2020). Methods and Algorithms for Correlation Analysis in R. *Journal of Open Source Software*, *5*(51), Article 2306. https://doi.org/10.21105/joss.02306

Marchetta, N. D. J., Hurks, P. P. M., De Sonneville, L. M. J., Krabbendam, L., & Jolles, J. (2008). Sustained and focused attention deficits in adult ADHD. *Journal of Attention Disorders*, *11*(6), 664–676. https://doi.org/10.1177/1087054707305108

Martella, D., Aldunate, N., Fuentes, L. J., & Sánchez-Pérez, N. (2020). Arousal and executive alterations in attention deficit hyperactivity disorder (ADHD). *Frontiers in Psychology*, *11*, Article 1991. https://doi.org/10.3389/fpsyg.2020.01991

Mary, A., Slama, H., Mousty, P., Massat, I., Capiau, T., Drabs, V., & Peigneux, P. (2016). Executive and attentional contributions to theory of mind deficit in attention deficit/hyperactivity disorder (ADHD). *Child Neuropsychology*, *22*(3), 345–365. https://doi.org/10.1080/09297049.2015.1012491

Mason, D. J., Humphreys, G. W., & Kent, L. (2004). Visual search, singleton capture, and the control of attentional set in ADHD. *Cognitive Neuropsychology*, *21*(6), 661–687. https://doi.org/10.1080/02643290342000267

McAuley, T., Crosbie, J., Charach, A., & Schachar, R. (2014). The persistence of cognitive deficits in remitted and unremitted ADHD: A case for the state-independence of response inhibition. *Journal of Child Psychology and Psychiatry*, *55*(3), 292–300. https://doi.org/10.1111/jcpp.12160

McDonald, J. H. (2014). *Handbook of biological statistics*. Sparky House Publishing. http://www.biostathandbook.com/HandbookBioStatThird.pdf

McIntire, L. K., McKinley, R. A., Nelson, J. M., & Goodyear, C. (2017). Transcranial direct current stimulation versus caffeine as a fatigue countermeasure. *Brain Stimulation*, *10*(6), 1070–1078. https://doi.org/10.1016/j.brs.2017.08.005

Meier, M. E. (2021). Testing the attention-distractibility trait. *Memory & Cognition*, *49*(7), 1490–1504. https://doi.org/10.3758/s13421-021-01162-5

Metin, B., Roeyers, H., Wiersema, J. R., Van Der Meere, J., & Sonuga-Barke, E. (2012). A meta-analytic study of event rate effects on go/no-go performance in attention-deficit/hyperactivity disorder. *Biological Psychiatry*, *72*(12), 990–996. https://doi.org/10.1016/j.biopsych.2012.08.023

Moffitt, T. E., Houts, R., Asherson, P., Belsky, D. W., Corcoran, D. L., Hammerle, M., Harrington, H., Hogan, S., Meier, M. H., Polanczyk, G. V., Poulton, R., Ramrakha, S., Sugden, K., Williams, B., Rohde, L. A., & Caspi, A. (2015). Is adult ADHD a childhood-onset neurodevelopmental disorder? Evidence from a four-decade longitudinal cohort study. *American Journal of Psychiatry*, *172*(10), 967–977. https://doi.org/10.1176/appi.ajp.2015.14101266

Mohr-Jensen, C., Müller Bisgaard, C., Boldsen, S. K., & Steinhausen, H.-C. (2019). Attention-Deficit/Hyperactivity Disorder in Childhood and Adolescence and the Risk of Crime in Young Adulthood in a Danish Nationwide Study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *58*(4), 443–452. https://doi.org/10.1016/j.jaac.2018.11.016

Morton, J., & Frith, U. (1995). Causal modeling: A structural approach to developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Vol. 1. Theory and methods* (pp. 357–390). John Wiley & Sons.

Moses, M., Tiego, J., Demontis, D., Bragi Walters, G., Stefansson, H., Stefansson, K., Børglum, A. D., Arnatkeviciute, A., & Bellgrove, M. A. (2022). Working memory and reaction time variability mediate the relationship between polygenic risk and ADHD traits in a general population sample. *Molecular Psychiatry*, *27*(12), Article 12. https://doi.org/10.1038/s41380-022-01775-5

Mullane, J. C., Corkum, P. V., Klein, R. M., McLaughlin, E. N., & Lawrence, M. A. (2011). Alerting, orienting, and executive attention in children with ADHD. *Journal of Attention Disorders*, *15*(4), 310–320. https://doi.org/10.1177/1087054710366384

Narison, R., de Montalembert, M., Bayliss, A., & Conty, L. (2021). Measuring gaze and arrow cuing effects with a short test adapted to brain damaged patients with unilateral spatial neglect: A preliminary study. *Frontiers in Psychology*, *12*, Article 690197.

https://www.frontiersin.org/articles/10.3389/fpsyg.2021.690197

National Institute for Health and Care Excellence. (2019). *Attention deficit hyperactivity disorder: Diagnosis and management*. http://www.ncbi.nlm.nih.gov/books/NBK493361/

Nazar, B. P., Bernardes, C., Peachey, G., Sergeant, J., Mattos, P., & Treasure, J. (2016). The risk of eating disorders comorbid with attention-deficit/hyperactivity disorder: A systematic review and meta-analysis. *International Journal of Eating Disorders*, *49*(12), 1045–1057. https://doi.org/10.1002/eat.22643

Nigg, J. T., Lewis, K., Edinger, T., & Falk, M. (2012). Meta-analysis of attention-deficit/hyperactivity disorder or attention-deficit/hyperactivity disorder symptoms, restriction diet, and synthetic food color additives. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*(1), 86–97. https://doi.org/10.1016/j.jaac.2011.10.015

Nigg, J. T., Sibley, M. H., Thapar, A., & Karalunas, S. L. (2020). Development of ADHD: Etiology, heterogeneity, and early life course. *Annual Review of Developmental Psychology*, *2*(1), 559–583. https://doi.org/10.1146/annurev-devpsych-060320-093413

Nigg, J. T., Willcutt, E. G., Doyle, A. E., & Sonuga-Barke, E. J. S. (2005). Causal heterogeneity in attention-deficit/hyperactivity disorder: Do we need neuropsychologically impaired subtypes? *Biological Psychiatry*, *57*(11), 1224–1230. https://doi.org/10.1016/j.biopsych.2004.08.025

Nikolas, M. A., Marshall, P., & Hoelzle, J. B. (2019). The role of neurocognitive tests in the assessment of adult attention-deficit/hyperactivity disorder. *Psychological Assessment*, *31*(5), 685–698. https://doi.org/10.1037/pas0000688

Norman, L. J., Sudre, G., Price, J., Shastri, G. G., & Shaw, P. (2023). Evidence from "big data" for the default-mode hypothesis of ADHD: A mega-analysis of multiple large samples.

Neuropsychopharmacology, *48*, 281–289. https://doi.org/10.1038/s41386-022-01408-z

Oberlin, B. G., Alford, J. L., & Marrocco, R. T. (2005). Normal attention orienting but abnormal stimulus alerting and conflict effect in combined subtype of ADHD. *Behavioural Brain Research*, *165*(1), 1–11. https://doi.org/10.1016/j.bbr.2005.06.041

Oken, B. S., Salinsky, M. C., & Elsas, S. M. (2006). Vigilance, alertness, or sustained attention: Physiological basis and measurement. *Clinical Neurophysiology*, *117*(9), 1885–1901. https://doi.org/10.1016/j.clinph.2006.01.017

Open Science Collaboration. (2015). Estimating the reproducibility of psychological science. *Science*, *349*(6251), 1–8. https://doi.org/10.1126/science.aac4716

Owens, M. M., Allgaier, N., Hahn, S., Yuan, D., Albaugh, M., Adise, S., Chaarani, B., Ortigara, J., Juliano, A., Potter, A., & Garavan, H. (2021). Multimethod investigation of the neurobiological basis of ADHD symptomatology in children aged 9-10: Baseline data from the ABCD study. *Translational Psychiatry*, *11*(1), Article 64. https://doi.org/10.1038/s41398-020-01192-8

Parsons, S., Kruijt, A.-W., & Fox, E. (2019). Psychological science needs a standard practice of reporting the reliability of cognitive-behavioral measurements. *Advances in Methods and Practices in Psychological Science*, *2*(4), 378–395. https://doi.org/10.1177/2515245919879695

Peasgood, T., Bhardwaj, A., Biggs, K., Brazier, J. E., Coghill, D., Cooper, C. L., Daley, D., De Silva, C., Harpin, V., Hodgkins, P., Nadkarni, A., Setyawan, J., & Sonuga-Barke, E. J. S. (2016). The impact of ADHD on the health and well-being of ADHD children and their siblings. *European Child & Adolescent Psychiatry*, *25*(11), 1217–1231. https://doi.org/10.1007/s00787-016-0841-6

Peasgood, T., Bhardwaj, A., Brazier, J. E., Biggs, K., Coghill, D., Daley, D., Cooper, C. L., De

Silva, C., Harpin, V., Hodgkins, P., Nadkarni, A., Setyawan, J., & Sonuga-Barke, E. J. S. (2021). What is the health and well-being burden for parents living with a child with ADHD in the united kingdom? *Journal of Attention Disorders*, *25*(14), 1962–1976. https://doi.org/10.1177/1087054720925899

Perugini, M., Gallucci, M., & Costantini, G. (2014). Safeguard power as a protection against imprecise power estimates. *Perspectives on Psychological Science*, *9*(3), 319–332. https://doi.org/10.1177/1745691614528519

Petersen, S. E., & Posner, M. I. (2012). The attention system of the human brain: 20 years after. *Annual Review of Neuroscience*, *35*, 73–89. https://doi.org/10.1146/annurev-neuro-062111-150525

Pingault, J.-B., Tremblay, R. E., Vitaro, F., Carbonneau, R., Genolini, C., Falissard, B., & Côté, S. M. (2011). Childhood trajectories of inattention and hyperactivity and prediction of educational attainment in early adulthood: A 16-year longitudinal population-based study. *American Journal of Psychiatry*, *168*(11), 1164–1170. https://doi.org/10.1176/appi.ajp.2011.10121732

Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: A systematic review and metaregression analysis. *American Journal of Psychiatry*, *164*(6), 942–948. https://doi.org/10.1176/ajp.2007.164.6.942

Polanczyk, G. V., Casella, C., & Jaffee, S. R. (2019). Commentary: ADHD lifetime trajectories and the relevance of the developmental perspective to Psychiatry: Reflections on Asherson and Agnew-Blais, (2019). *Journal of Child Psychology and Psychiatry*, *60*(4), 353–355. https://doi.org/10.1111/jcpp.13050

Polanczyk, G. V., Willcutt, E. G., Salum, G. A., Kieling, C., & Rohde, L. A. (2014). ADHD prevalence estimates across three decades: An updated systematic review and meta-

regression analysis. *International Journal of Epidemiology*, *43*(2), 434–442. https://doi.org/10.1093/ije/dyt261

Posner, J., Polanczyk, G. V., & Sonuga-Barke, E. (2020). Attention-deficit hyperactivity disorder. *The Lancet*, *395*(10222), 450–462. https://doi.org/10.1016/S0140-6736(19)33004-1

Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology*, *32*(1), 3–25. https://doi.org/10.1080/00335558008248231

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, *13*, 25–42. https://doi.org/10.1146/annurev.ne.13.030190.000325

Psychology Software Tools, Inc. (2012). *E-Prime* (2.0) [Computer software]. https://pstnet.com/products/e-prime-legacy-versions/

Quintero, J., Morales, I., Vera, R., Zuluaga, P., & Fernández, A. (2019). The impact of adult ADHD in the quality of life profile. *Journal of Attention Disorders*, *23*(9), 1007–1016. https://doi.org/10.1177/1087054717733046

R Core Team. (2022). *R: A language and environment for statistical computing* (Version 4.2.1) [Computer software]. R Foundation for Statistical Computing. https://www.R-project.org/

Redacción Médica. (n.d.). *Un cuestionario de 6 preguntas detecta el TDAH en adultos*. Redacción Médica. https://www.redaccionmedica.com/secciones/psiquiatria/un-cuestionario-de-6-preguntas-detecta-el-tdah-en-adultos-1647

Reinhardt, M. C., Benetti, L., Victor, M. M., Grevet, E. H., Belmonte-de-Abreu, P., Faraone, S. V., & Rohde, L. A. (2007). Is age-at-onset criterion relevant for the response to methylphenidate in attention-deficit/hyperactivity disorder? *The Journal of Clinical Psychiatry*, *68*, 1109–1116. https://doi.org/10.4088/JCP.v68n0720

Riccio, C. A., & Reynolds, C. R. (2001). Continuous performance tests are sensitive to ADHD in adults but lack specificity. *Annals of the New York Academy of Sciences*, *931*(1), 113–139. https://doi.org/10.1111/j.1749-6632.2001.tb05776.x

Riesco-Matías, P., Yela-Bernabé, J. R., Crego, A., & Sánchez-Zaballos, E. (2021). What do meta-analyses have to say about the efficacy of neurofeedback applied to children with ADHD? Review of previous meta-analyses and a new meta-analysis. *Journal of Attention Disorders*, *25*(4), 473–485. https://doi.org/10.1177/1087054718821731

Riglin, L., Wootton, R. E., Livingston, L. A., Agnew-Blais, J., Arseneault, L., Blakey, R., Agha, S. S., Langley, K., Collishaw, S., O'Donovan, M. C., Smith, G. D., Stergiakouli, E., Tilling, K., & Thapar, A. (2022). "Late-onset" ADHD symptoms in young adulthood: Is this ADHD? *Journal of Attention Disorders*, *26*(10), 1271–1282. https://doi.org/10.1177/10870547211066486

Rimestad, M. L., Lambek, R., Zacher Christiansen, H., & Hougaard, E. (2016). Short- and long-term effects of parent training for preschool children with or at risk of ADHD: A systematic review and meta-analysis. *Journal of Attention Disorders*, *23*(5), 423–434. https://doi.org/10.1177/1087054716648775

Robe, A., & Dobrean, A. (2022). The effectiveness of a single session of mindfulness-based cognitive training on cardiac vagal control and core symptoms in children and adolescents with attention-deficit/hyperactivity disorder (ADHD): A preliminary randomized controlled trial. *European Child & Adolescent Psychiatry*. Advance online publication. https://doi.org/10.1007/s00787-022-02005-7

Roberts, K. L., Summerfield, A. Q., & Hall, D. A. (2006). Presentation modality influences behavioral measures of alerting, orienting, and executive control. *Journal of the International Neuropsychological Society*, *12*(4), 485–492. https://doi.org/10.1017/S1355617706060620

Robertson, I. H., Manly, T., Andrade, J., Baddeley, B. T., & Yiend, J. (1997). "Oops!": Performance correlates of everyday attentional failures in traumatic brain injured and normal subjects. *Neuropsychologia*, *35*(6), 747–758. https://doi.org/10.1016/s0028-3932(97)00015-8

Robinson, G. K. (1991). That BLUP is a good thing: The estimation of random effects. *Statistical Science*, *6*(1), 15–32. https://doi.org/10.1214/ss/1177011926

Roca, J., Castro, C., López-Ramón, M. F., & Lupiáñez, J. (2011). Measuring vigilance while assessing the functioning of the three attentional networks: The ANTI-Vigilance task. *Journal of Neuroscience Methods*, *198*(2), 312–324. https://doi.org/10.1016/j.jneumeth.2011.04.014

Rohde, L. (2023, May 18–21) *What is new on ADHD trajectories from childhood to adulthood?* [Conference session]. 9th World Congress on ADHD, Amsterdan, Netherlands.

Rohrer, J. M. (2018). Thinking clearly about correlations and causation: Graphical causal models for observational data. *Advances in Methods and Practices in Psychological Science*, *1*(1), 27–42. https://doi.org/10.1177/2515245917745629

Román-Caballero, R., Martín-Arévalo, E., & Lupiáñez, J. (2021). Attentional networks functioning and vigilance in expert musicians and non-musicians. *Psychological Research*, *85*(3), 1121–1135. https://doi.org/10.1007/s00426-020-01323-2

Román-Caballero, R., Martín-Arévalo, E., & Lupiáñez, J. (2023). Changes in response criterion and lapse rate as general mechanisms of vigilance decrement: Commentary on McCarley and Yamani (2021). *Psychological Science*, *34*(1), 132–136. https://doi.org/10.1177/09567976221121342

Román-Caballero, R., Vadillo, M. A., Trainor, L. J., & Lupiáñez, J. (2022). Please don't stop the music: A meta-analysis of the cognitive and academic benefits of instrumental musical training in childhood and adolescence. *Educational Research Review*, *35*,

100436. https://doi.org/10.1016/j.edurev.2022.100436

Rommel, A. S., Rijsdijk, F., Greven, C. U., Asherson, P., & Kuntsi, J. (2015). a longitudinal twin study of the direction of effects between ADHD symptoms and IQ. *PLOS ONE*, *10*(4), Article e0124357. https://doi.org/10.1371/JOURNAL.PONE.0124357

Rosvold, H. E., Mirsky, A. F., Sarason, I., Bransome Jr., E. D., & Beck, L. H. (1956). A continuous performance test of brain damage. *Journal of Consulting Psychology*, *20*, 343–350. https://doi.org/10.1037/h0043220

Rubia, K. (2022). Neurotherapeutics for ADHD: Do they work? *PsyCh Journal*, *11*(3), 419–427. https://doi.org/10.1002/pchj.544

Rueda, M. R., Fan, J., McCandliss, B. D., Halparin, J. D., Gruber, D. B., Lercari, L. P., & Posner, M. I. (2004). Development of attentional networks in childhood. *Neuropsychologia*, *42*(8), 1029–1040. https://doi.org/10.1016/j.neuropsychologia.2003.12.012

Ruiz-Goikoetxea, M., Cortese, S., Aznarez-Sanado, M., Magallón, S., Alvarez Zallo, N., Luis, E. O., de Castro-Manglano, P., Soutullo, C., & Arrondo, G. (2018). Risk of unintentional injuries in children and adolescents with ADHD and the impact of ADHD medications: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, *84*, 63–71. https://doi.org/10.1016/j.neubiorev.2017.11.007

Rydell, M., Lundström, S., Gillberg, C., Lichtenstein, P., & Larsson, H. (2018). Has the attention deficit hyperactivity disorder phenotype become more common in children between 2004 and 2014? Trends over 10 years from a Swedish general population sample. *Journal of Child Psychology and Psychiatry*, *59*(8), 863–871. https://doi.org/10.1111/jcpp.12882

Sadeh, A., Dan, O., & Bar-Haim, Y. (2011). Online assessment of sustained attention following sleep restriction. *Sleep Medicine*, *12*(3), 257–261.

https://doi.org/10.1016/j.sleep.2010.02.001

Sala, G., & Gobet, F. (2019). Cognitive training does not enhance general cognition. *Trends in Cognitive Sciences*, *23*(1), 9–20. https://doi.org/10.1016/j.tics.2018.10.004

Salomone, S., Fleming, G. R., Bramham, J., O'Connell, R. G., & Robertson, I. H. (2020). Neuropsychological deficits in adult ADHD: Evidence for differential attentional impairments, deficient executive functions, and high self-reported functional impairments. *Journal of Attention Disorders*, *24*(10), 1413–1424. https://doi.org/10.1177/1087054715623045

Sanchez-Garcia, M., Fernandez-Calderon, F., Carmona-Marquez, J., Chico-Garcia, M., Velez-Moreno, A., & Perez-Gomez, L. (2015). Psychometric properties and adaptation of the ASRS in a Spanish sample of patients with substance use disorders: Application of two IRT Rasch models. *Psychological Assessment*, *27*(2), 524–533. https://doi.org/10.1037/pas0000064

Sanchis, C., Blasco, E., Luna, F. G., & Lupiáñez, J. (2020). Effects of caffeine intake and exercise intensity on executive and arousal vigilance. *Scientific Reports*, *10*, Article 8393. https://doi.org/10.1038/s41598-020-65197-5

Sanders, A. F. (1983). Towards a model of stress and human performance. *Acta Psychologica*, *53*(1), 61–97. https://doi.org/10.1016/0001-6918(83)90016-1

Sanders, A. F. (1998). Elements of human performance: Reaction processes and attention in human skill. Lawrence Erlbaum Associates Publishers.

Schippers, L. M., Horstman, L. I., van de Velde, H., Pereira, R. R., Zinkstok, J., Mostert, J. C., Greven, C. U., & Hoogman, M. (2022). A qualitative and quantitative study of self-reported positive characteristics of individuals with ADHD. *Frontiers in Psychiatry*, *13*, Article 922788. https://doi.org/10.3389/fpsyt.2022.922788

Schoechlin, C., & Engel, R. R. (2005). Neuropsychological performance in adult attention-

deficit hyperactivity disorder: Meta-analysis of empirical data. *Archives of Clinical Neuropsychology*, *20*(6), 727–744. https://doi.org/10.1016/j.acn.2005.04.005

Schuhfried. (2013). Vienna Test Neuro. Schuhfried. https://delarosaresearch.com/uploads/Schu_catalog_VTS_3.00_2006.10_en.pdf

See, J. E., Howe, S. R., Warm, J. S., & Dember, W. N. (1995). Meta-analysis of the sensitivity decrement in vigilance. *Psychological Bulletin*, *117*(2), 230–249. https://doi.org/10.1037/0033-2909.117.2.230

Septier, M., Stordeur, C., Zhang, J., Delorme, R., & Cortese, S. (2019). Association between suicidal spectrum behaviors and attention-deficit/hyperactivity disorder: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, *103*, 109–118. https://doi.org/10.1016/j.neubiorev.2019.05.022

Sergeant, J. (2000). The cognitive-energetic model: An empirical approach to attention-deficit hyperactivity disorder. *Neuroscience & Biobehavioral Reviews*, *24*(1), 7–12. https://doi.org/10.1016/S0149-7634(99)00060-3

Sergeant, J. A. (2005). Modeling attention-deficit/hyperactivity disorder: A critical appraisal of the cognitive-energetic model. *Biological Psychiatry*, *57*(11), 1248–1255. https://doi.org/10.1016/j.biopsych.2004.09.010

Servera, M., & Cardo, E. (2006). Children Sustained Attention Task (CSAT): Normative, reliability, and validity data. *International Journal of Clinical and Health Psychology*, *6*(3), 697–707. https://www.redalyc.org/pdf/337/33760311.pdf

Shahar, E., & Shahar, D. J. (2012). Causal diagrams and change variables. *Journal of Evaluation in Clinical Practice*, *18*(1), 143–148. https://doi.org/10.1111/j.1365-2753.2010.01540.x

Shaw, M., Hodgkins, P., Caci, H., Young, S., Kahle, J., Woods, A. G., & Arnold, L. E. (2012). A systematic review and analysis of long-term outcomes in attention deficit

hyperactivity disorder: Effects of treatment and non-treatment. *BMC Medicine*, *10*, Article 99. https://doi.org/10.1186/1741-7015-10-99

Sibley, M. H., Arnold, L. E., Swanson, J. M., Hechtman, L. T., Kennedy, T. M., Owens, E., Molina, B. S. G., Jensen, P. S., Hinshaw, S. P., Roy, A., Chronis-Tuscano, A., Newcorn, J. H., Rohde, L. A., & Group, M. C. G. (2022). Variable patterns of remission from adhd in the multimodal treatment study of ADHD. *179*(2), 142–151. https://doi.org/10.1176/APPI.AJP.2021.21010032

Sibley, M. H., Bruton, A. M., Zhao, X., Johnstone, J. M., Mitchell, J., Hatsu, I., Arnold, L. E., Basu, H. H., Levy, L., Vyas, P., Macphee, F., Gonzalez, E. S., Kelley, M., Jusko, M. L., Bolden, C. R., Zulauf-McCurdy, C., Manzano, M., & Torres, G. (2023). Non-pharmacological interventions for attention-deficit hyperactivity disorder in children and adolescents. *The Lancet Child & Adolescent Health*, *7*(6), 415–428. https://doi.org/10.1016/S2352-4642(22)00381-9

Sibley, M. H., Pelham, W. E., Gnagy, E. M., Waxmonsky, J. G., Waschbusch, D. A., Derefinko, K. J., Garefino, A. C., Molina, B. S. G., Wymbs, B. T., Babinski, D. E., & Kuriyan, A. B. (2012). When diagnosing ADHD in young adults emphasize informant reports, DSM items, and impairment. *Journal of Consulting and Clinical Psychology*, *80*(6), 1052–1061. https://doi.org/10.1037/a0029098

Sikström, S., & Söderlund, G. (2007). Stimulus-dependent dopamine release in attention-deficit/hyperactivity disorder. *Psychological Review*, *114*(4), 1047–1075. https://doi.org/10.1037/0033-295X.114.4.1047

Simon, V., Czobor, P., Bálint, S., Mészáros, A., & Bitter, I. (2009). Prevalence and correlates of adult attention-deficit hyperactivity disorder: Meta-analysis. *The British Journal of Psychiatry*, *194*(3), 204–211. https://doi.org/10.1192/bjp.bp.107.048827

Simons, D. J., Boot, W. R., Charness, N., Gathercole, S. E., Chabris, C. F., Hambrick, D. Z.,

& Stine-Morrow, E. A. L. (2016). Do “brain-training” programs work? *Psychological Science in the Public Interest*, *17*(3), 103–186. https://doi.org/10.1177/1529100616661983

Simonsohn, U., Simmons, J. P., & Nelson, L. D. (2020). Specification curve analysis. *Nature Human Behaviour*, *4*(11), 1208–1214. https://doi.org/10.1038/s41562-020-0912-z

Sjöwall, D., Roth, L., Lindqvist, S., & Thorell, L. B. (2013). Multiple deficits in ADHD: Executive dysfunction, delay aversion, reaction time variability, and emotional deficits. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *54*(6), 619–627. https://doi.org/10.1111/jcpp.12006

Solanto, M. V., Etefia, K., & Marks, D. J. (2004). The utility of self-report measures and the continuous performance test in the diagnosis of ADHD in adults. *CNS Spectrums*, *9*(9), 649–659. https://doi.org/10.1017/S1092852900001929

Soler-Gutiérrez, A.-M., Pérez-González, J.-C., & Mayas, J. (2023). Evidence of emotion dysregulation as a core symptom of adult ADHD: A systematic review. *PLOS ONE*, *18*(1), Article e0280131. https://doi.org/10.1371/journal.pone.0280131

Song, M., Dieckmann, N. F., & Nigg, J. T. (2019). Addressing discrepancies between ADHD prevalence and case identification estimates among US children utilizing NSCH 2007-2012. *Journal of Attention Disorders*, *23*(14), 1691–1702. https://doi.org/10.1177/1087054718799930

Sonuga-Barke, E., Bitsakou, P., & Thompson, M. (2010). Beyond the dual pathway model: Evidence for the dissociation of timing, inhibitory, and delay-related impairments in attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *49*(4), 345–355. https://doi.org/10.1016/j.jaac.2009.12.018

Sonuga-Barke, E., & Cortese, S. (2018). Cognitive training approaches for ADHD: Can they be made more effective? In T. Banaschewski, Coghill, & A. Zuddas (Eds.), *Oxford*

textbook of attention deficit hyperactivity disorder (pp. 358–365). Oxford University Press. https://doi.org/10.1093/med/9780198739258.003.0038

Sonuga-Barke, E. J. S. (2003). The dual pathway model of AD/HD: An elaboration of neuro-developmental characteristics. *Neuroscience & Biobehavioral Reviews*, *27*(7), 593–604. https://doi.org/10.1016/j.neubiorev.2003.08.005

Sonuga-Barke, E. J. S. (2005). Causal models of attention-deficit/hyperactivity disorder: From common simple deficits to multiple developmental pathways. *Biological Psychiatry*, *57*(11), 1231–1238. https://doi.org/10.1016/j.biopsych.2004.09.008

Sonuga-Barke, E. J. S. (2013). Attention-deficit/hyperactivity disorder: Toward a developmental synthesis. In P. D. Zelazo (Ed.), *The oxford handbook of developmental psychology: Vol. 2. Self and other* (p. 549–578). Oxford University Press. https://doi.org/10.1093/oxfordhb/9780199958474.013.0022

Sonuga-Barke, E. J. S. (2017, October 2–5). *When should attentional impairment in adulthood be classified as ADHD?* [Conference session]. EUNETHYDIS 25th Network Meeting, Cologne, Germany.

Sonuga-Barke, E. J. S. (2019, September 24). *What causes ADHD? Can science improve treatment?* [Conference session]. ACAMH ADHD Masterclass, London, UK.

Sonuga-Barke, E. J. S. (2020). Editorial: 'People get ready': Are mental disorder diagnostics ripe for a Kuhnian revolution? *Journal of Child Psychology and Psychiatry*, *61*(1), 1–3. https://doi.org/10.1111/jcpp.13181

Sonuga-Barke, E. J. S. (Guest). (2023, August 7). *What we're all getting wrong about ADHD* [Audio podcast episode]. Instant genius. https://www.ivoox.com/what-we-8217-re-all-getting-wrong-about-adhd-audios-mp3_rf_113791475_1.html

Sonuga-Barke, E. J. S., Becker, S. P., Bölte, S., Castellanos, F. X., Franke, B., Newcorn, J. H., Nigg, J. T., Rohde, L. A., & Simonoff, E. (2023). Annual research review: Perspectives

on progress in ADHD science – from characterization to cause. *Journal of Child Psychology and Psychiatry*, *64*(4), 506–532. https://doi.org/10.1111/jcpp.13696

Sonuga-Barke, E. J. S., Beckett, C., Kreppner, J., Castle, J., Colvert, E., Stevens, S., Hawkins, A., & Rutter, M. (2008). Is sub-nutrition necessary for a poor outcome following early institutional deprivation? *Developmental Medicine and Child Neurology*, *50*(9), 664–671. https://doi.org/10.1111/j.1469-8749.2008.03065.x

Sonuga-Barke, E. J. S., & Fearon, R. M. P. (2019). Commentary: 'Ready or not here I come': Developmental immaturity as a driver of impairment and referral in young-for-school-grade ADHD children. A reformulation inspired by Whitely et al. (2019). *Journal of Child Psychology and Psychiatry*, *60*(4), 392–394. https://doi.org/10.1111/jcpp.13039

Sonuga-Barke, E. J. S., & Halperin, J. M. (2010). Developmental phenotypes and causal pathways in attention deficit/hyperactivity disorder: Potential targets for early intervention? *Journal of Child Psychology and Psychiatry*, *51*(4), 368–389. https://doi.org/10.1111/j.1469-7610.2009.02195.x

Sonuga-Barke, E. J. S., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and delay aversion—I. The effect of delay on choice. *Journal of Child Psychology and Psychiatry*, *33*(2), 387–398. https://doi.org/10.1111/j.1469-7610.1992.tb00874.x

Sonuga-Barke, E. J. S., Wiersema, J. R., van der Meere, J. J., & Roeyers, H. (2010). Context-dependent dynamic processes in attention deficit/hyperactivity disorder: Differentiating common and unique effects of state regulation deficits and delay aversion. *Neuropsychology Review*, *20*(1), 86–102. https://doi.org/10.1007/s11065-009-9115-0

Sonuga-Barke, E., & Kostyrka-Allchorne, K. (2023). Editorial Perspective: Attention-deficit/hyperactivity disorder viewed as neuro-divergence in the digital world. Journal of Child Psychology and Psychiatry, and Allied Disciplines, 64(6), 972–974. https://doi.org/10.1111/jcpp.13710

Sonuga-Barke, E., & Thapar, A. (2021). The neurodiversity concept: Is it helpful for clinicians and scientists? *The Lancet Psychiatry*, *8*(7), 559–561. https://doi.org/10.1016/S2215-0366(21)00167-X

Spearman, C. (1904). The proof and measurement of association between two things. *American Journal of Psychology*, *15*(1), 72–101. https://doi.org/10.2307/1412159

Steegen, S., Tuerlinckx, F., Gelman, A., & Vanpaemel, W. (2016). Increasing transparency through a multiverse analysis. *Perspectives on Psychological Science*, *11*(5), 702–712. https://doi.org/10.1177/1745691616658637

Stern, A., Agnew-Blais, J., Danese, A., Fisher, H. L., Matthews, T., Polanczyk, G. V., Wertz, J., & Arseneault, L. (2020). Associations between ADHD and emotional problems from childhood to young adulthood: A longitudinal genetically-sensitive study. *Journal of Child Psychology and Psychiatry*, *61*(11), 1234–1242. https://doi.org/10.1111/jcpp.13217

Stojanoski, B., Wild, C. J., Battista, M. E., Nichols, E. S., & Owen, A. M. (2021). Brain training habits are not associated with generalized benefits to cognition: An online study of over 1000 "brain trainers". *Journal of Experimental Psychology*: *General*, *150*(4), 729–738. https://doi.org/10.1037/xge0000773

Sturm, W., de Simone, A., Krause, B. J., Specht, K., Hesselmann, V., Radermacher, I., Herzog, H., Tellmann, L., Müller-Gärtner, H.-W., & Willmes, K. (1999). Functional anatomy of intrinsic alertness: Evidence for a fronto-parietal-thalamic-brainstem network in the right hemisphere. *Neuropsychologia*, *37*(7), 797–805. https://doi.org/10.1016/S0028-3932(98)00141-9

Swanson, J., Gupta, S., Lam, A., Shoulson, I., Lerner, M., Modi, N., Lindemulder, E., & Wigal, S. (2003). Development of a new once-a-day formulation of methylphenidate for the treatment of attention-deficit/hyperactivity disorder: Proof-of-concept and proof-of-

product studies. *Archives of General Psychiatry*, *60*(2), 204–211. https://doi.org/10.1001/archpsyc.60.2.204

Taylor, L. E., Kaplan-Kahn, E. A., Lighthall, R. A., & Antshel, K. M. (2022). Adult-onset ADHD: A critical analysis and alternative explanations. *Child Psychiatry and Human Development*, *53*(4), 635–653. https://doi.org/10.1007/s10578-021-01159-w

Thomson, D. R., Besner, D., & Smilek, D. (2016). A critical examination of the evidence for sensitivity loss in modern vigilance tasks. *Psychological Review*, *123*(1), 70–83. https://doi.org/10.1037/rev0000021

Tiwari, T., Singh, A. L., & Singh, I. L. (2009). Task demand and workload: Effects on vigilance performance and stress. *Journal of the Indian Academy of Applied Psychology*, *35*(2), 265–275.

Tucha, L., Fuermaier, A. B. M., Koerts, J., Buggenthin, R., Aschenbrenner, S., Weisbrod, M., Thome, J., Lange, K. W., & Tucha, O. (2017). Sustained attention in adult ADHD: Time-on-task effects of various measures of attention. *Journal of Neural Transmission*, *124*(Suppl 1), S39–S53. https://doi.org/10.1007/s00702-015-1426-0

Tucha, L., Tucha, O., Laufkötter, R., Walitza, S., Klein, H. E., & Lange, K. W. (2008). Neuropsychological assessment of attention in adults with different subtypes of attention-deficit/hyperactivity disorder. *Journal of Neural Transmission*, *115*(2), 269–278. https://doi.org/10.1007/s00702-007-0836-z

Tucha, L., Tucha, O., Walitza, S., Sontag, T. A., Laufkötter, R., Linder, M., & Lange, K. W. (2009). Vigilance and sustained attention in children and adults with ADHD. *Journal of Attention Disorders*, *12*(5), 410–421. https://doi.org/10.1177/1087054708315065

Tucha, O., Walitza, S., Mecklinger, L., Sontag, T.-A., Kübber, S., Linder, M., & Lange, K. W. (2006). Attentional functioning in children with ADHD – predominantly hyperactive-impulsive type and children with ADHD – combined type. *Journal of Neural*

Transmission, *113*(12), 1943–1953. https://doi.org/10.1007/s00702-006-0496-4

Ustun, B., Adler, L. A., Rudin, C., Faraone, S. V., Spencer, T. J., Berglund, P., Gruber, M. J., & Kessler, R. C. (2017). The world health organization adult attention-deficit/hyperactivity disorder self-report screening scale for DSM-5. *JAMA Psychiatry*, *74*(5), 520–526. https://doi.org/10.1001/jamapsychiatry.2017.0298

van der Meere, J. (2005). State Regulation and Attention Deficit Hyperactivity Disorder. In D. Gozal & D. L. Molfese (Eds.), *Attention deficit hyperactivity disorder: From genes to patients* (pp. 413–433). Humana Press. https://doi.org/10.1385/1-59259-891-9:413

van der Meere, J. J., Börger, N. A., & Wiersema, J. R. (2010). ADHD: State regulation and motivation. *Current Medical Literature. Psychiatry*, *21*(1), 14–20.

van Lieshout, M., Luman, M., Buitelaar, J., Rommelse, N. N. J., & Oosterlaan, J. (2013). Does neurocognitive functioning predict future or persistence of ADHD? A systematic review. *Clinical Psychology Review*, *33*(4), 539–560. https://doi.org/10.1016/j.cpr.2013.02.003

van Zomeren, A. H., & Brouwer, W. H. (1994). *Clinical neuropsychology of attention*. Oxford University Press.

Vazire, S., Schiavone, S. R., & Bottesini, J. G. (2022). Credibility beyond replicability: Improving the four validities in psychological science. *Current Directions in Psychological Science*, *31*(2), 162–168. https://doi.org/10.1177/09637214211067779

Vázquez-Marrufo, M., García-Valdecasas Colell, M., Galvao-Carmona, A., Sarrias-Arrabal, E., & Tirapu-Ustárroz, J. (2019). El Attention Network Test en el estudio de los déficits cognitivos de pacientes con trastorno por déficit de atención [The Attention Network Test in the study of cognitive impairment of ADHD patients]. *Revista De Neurología*, *69*(10), 423–432. https://doi.org/10.33588/rn.6910.2019202

Vitiello, B., Elliott, G. R., Swanson, J. M., Arnold, L. E., Hechtman, L., Abikoff, H., Molina,

B. S. G., Wells, K., Wigal, T., Jensen, P. S., Greenhill, L. L., Kaltman, J. R., Severe, J. B., Odbert, C., Hur, K., & Gibbons, R. (2012). Blood pressure and heart rate over 10 years in the multimodal treatment study of children with ADHD. *American Journal of Psychiatry*, *169*(2), 167–177. https://doi.org/10.1176/appi.ajp.2011.10111705

Volkow, N. D., & Swanson, J. M. (2003). Variables that affect the clinical use and abuse of methylphenidate in the treatment of ADHD. *The American Journal of Psychiatry*, *160*(11), 1909–1918. https://doi.org/10.1176/appi.ajp.160.11.1909

von Bastian, C. C., Belleville, S., Udale, R. C., Reinhartz, A., Essounni, M., & Strobach, T. (2022). Mechanisms underlying training-induced cognitive change. *Nature Reviews Psychology*, *1*, 30–41. https://doi.org/10.1038/s44159-021-00001-3

von Wirth, E., Mandler, J., Breuer, D., & Döpfner, M. (2021). The accuracy of retrospective recall of childhood ADHD: Results from a longitudinal study. *Journal of Psychopathology and Behavioral Assessment*, *43*(2), 413–426. https://doi.org/10.1007/s10862-020-09852-1

Westwood, S. J., Radua, J., & Rubia, K. (2021). Noninvasive brain stimulation in children and adults with attention-deficit/hyperactivity disorder: A systematic review and meta-analysis. *Journal of Psychiatry & Neuroscience*, *46*(1), E14–E33. https://doi.org/10.1503/jpn.190179

Westwood, S., Parlatini, V., Rubia, K., Cortese, S., Sonuga-Barke, E. S. J., Thapar, A., & European ADHD Guidelines Group (EAGG). (2023). Computerized cognitive training in attention-deficit/hyperactivity disorder (ADHD): A meta-analysis of randomized controlled trials with blinded and objective outcomes. *Molecular Psychiatry*, *28*, 1402–1414. https://doi.org/10.1038/s41380-023-02000-7

Weyandt, L. L., Oster, D. R., Gudmundsdottir, B. G., DuPaul, G. J., & Anastopoulos, A. D. (2017). Neuropsychological functioning in college students with and without ADHD.

Neuropsychology, *31*(2), 160–172. https://doi.org/10.1037/neu0000326

Wickham, H. (2011). The split-apply-combine strategy for data analysis. *Journal of Statistical Software*, *40*, 1–29. https://doi.org/10.18637/jss.v040.i01

Wickham, H., Averick, M., Bryan, J., Chang, W., McGowan, L., François, R., Grolemund, G., Hayes, A., Henry, L., Hester, J., Kuhn, M., Pedersen, T., Miller, E., Bache, S., Müller, K., Ooms, J., Robinson, D., Seidel, D., Spinu, V., … Yutani, H. (2019). Welcome to the tidyverse. *Journal of Open Source Software*, *4*(43), Article 1686. https://doi.org/10.21105/joss.01686

Wilding, J. (2005). Is attention impaired in ADHD? *British Journal of Developmental Psychology*, *23*(4), 487–505. https://doi.org/10.1348/026151005X48972

Willcutt, E. G. (2012). The prevalence of DSM-IV attention-deficit/hyperactivity disorder: A meta-analytic review. *Neurotherapeutics*, *9*(3), 490–499. https://doi.org/10.1007/s13311-012-0135-8

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. *Biological Psychiatry*, *57*(11), 1336–1346. https://doi.org/10.1016/J.BIOPSYCH.2005.02.006

Wolraich, M. L., Hagan, J. F., Jr., Allan, C., Chan, E., Davison, D., Earls, M., Evans, S. W., Flinn, S. K., Froehlich, T., Frost, J., Holbrook, J. R., Lehmann, C. U., Lessin, H. R., Okechukwu, K., Pierce, K. L., Winner, J. D., Zurhellen, W., & Subcommittee on Children and Adolescents with Attention-Deficit/Hyperactive Disorder. (2019). Clinical practice guideline for the diagnosis, evaluation, and treatment of attention-deficit/hyperactivity disorder in children and adolescents. *Pediatrics*, *144*(4), Article e20192528. https://doi.org/10.1542/peds.2019-2528

World Health Organization. (2019). *International statistical classification of diseases and*

related health problems (11th ed.). https://icd.who.int/

World Health Organization. (2023). Neurodevelopmental disorders. In *International statistical classification of diseases and related health problems* (11th ed.). https://icd.who.int/browse11/l-m/en#/http%3A%2F%2Fid.who.int%2Ficd%2Fentity%2F1516623224

Wood, D. R., Reimherr, F. W., Wender, P. H., & Johnson, G. E. (1976). Diagnosis and treatment of minimal brain dysfunction in adults: A preliminary report. *Archives of General Psychiatry*, *33*(12), 1453–1460. https://doi.org/10.1001/archpsyc.1976.01770120057005

Xu, G., Strathearn, L., Liu, B., Yang, B., & Bao, W. (2018). Twenty-year trends in diagnosed attention-deficit/hyperactivity disorder among us children and adolescents, 1997-2016. *JAMA Network Open*, *1*(4), Article e181471. https://doi.org/10.1001/jamanetworkopen.2018.1471

Young, Z., Moghaddam, N., & Tickle, A. (2020). The efficacy of cognitive behavioral therapy for adults with ADHD: A systematic review and meta-analysis of randomized controlled trials. *Journal of Attention Disorders*, *24*(6), 875–888. https://doi.org/10.1177/1087054716664413

Youyou, W., Yang, Y., & Uzzi, B. (2023). A discipline-wide investigation of the replicability of Psychology papers over the past two decades. *Proceedings of the National Academy of Sciences*, *120*(6), Article e2208863120. https://doi.org/10.1073/pnas.2208863120

Yu, C.-L., Hsieh, S.-S., Chueh, T.-Y., Huang, C.-J., Hillman, C. H., & Hung, T.-M. (2020). The effects of acute aerobic exercise on inhibitory control and resting state heart rate variability in children with ADHD. *Scientific Reports*, *10*, Article 19958. https://doi.org/10.1038/s41598-020-76859-9

Zamani Sani, S. H., Fathirezaie, Z., Sadeghi-Bazargani, H., Badicu, G., Ebrahimi, S., Grosz,

R. W., Sadeghi Bahmani, D., & Brand, S. (2020). Driving accidents, driving violations, symptoms of attention-deficit-hyperactivity (ADHD) and attentional network tasks. *International Journal of Environmental Research and Public Health*, *17*(14), Article 5238. https://doi.org/10.3390/ijerph17145238

Zang, Y. (2019). Impact of physical exercise on children with attention deficit hyperactivity disorders. *Medicine*, *98*(46), Article e17980. https://doi.org/10.1097/MD.0000000000017980

Zhang, J., Díaz-Román, A., & Cortese, S. (2018). Meditation-based therapies for attention-deficit/hyperactivity disorder in children, adolescents and adults: a systematic review and meta-analysis. *Evidence-Based Mental Health*, *21*(3), 87–94. https://doi.org/10.1136/ebmental-2018-300015

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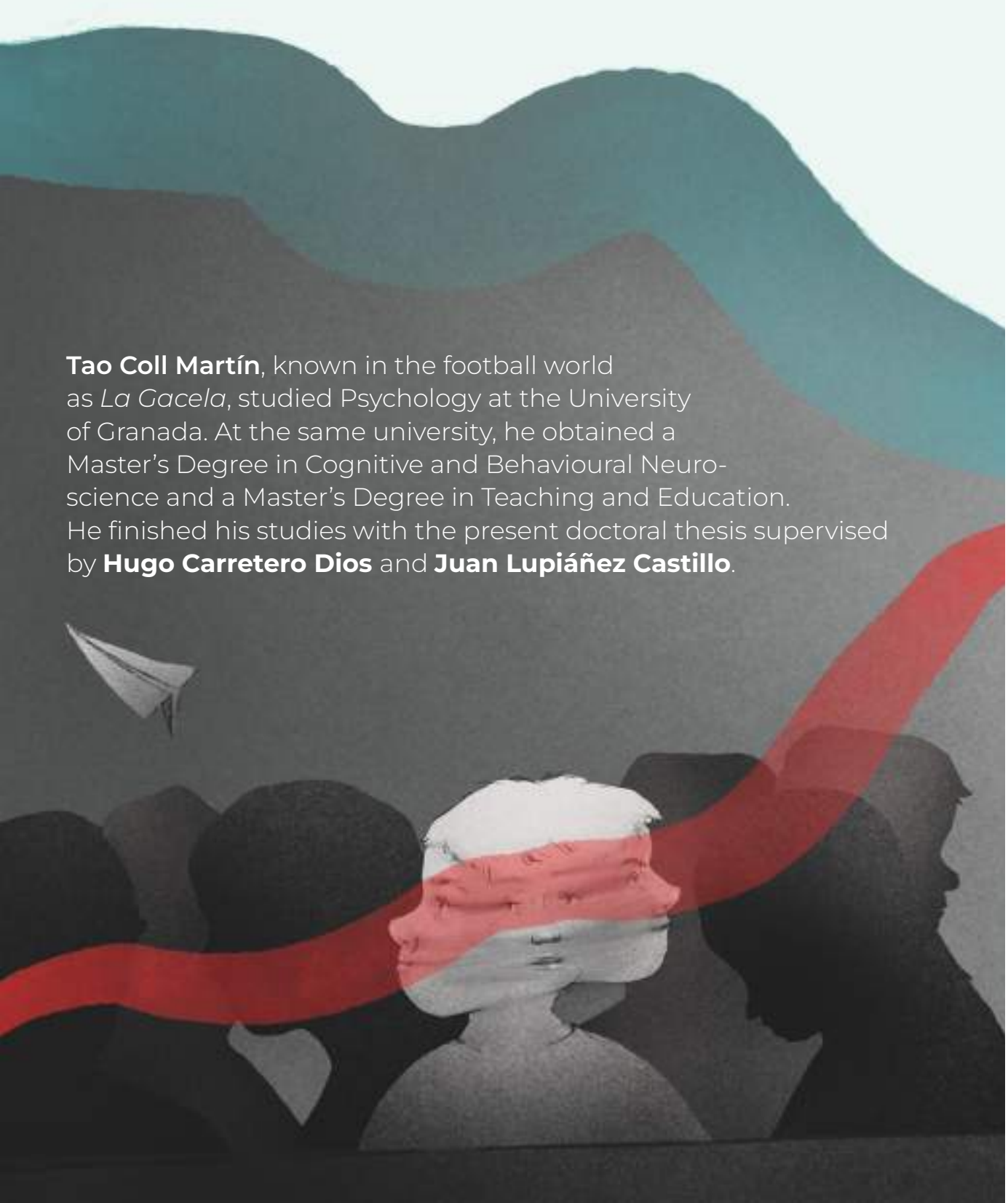

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