FIFTY YEARS OF EXECUTIVE CONTROL RESEARCH IN ATTENTION-DEFICIT/HYPERACTIVITY DISORDER:WHAT WE HAVE LEARNED AND STILL NEED TO KNOW

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Abstract

For 50 years, attention-deficit/hyperactivity disorder (ADHD) has been considered a disorder of executive control (EC), the higher-order, cognitive skills that support self-regulation, goal attainment and what we generally call "attention." This review surveys our current understanding of the nature of EC as it pertains to ADHD and considers the evidence in support of eight hypotheses that can be derived from the EC theory of ADHD. This paper provides a resource for practitioners to aid in clinical decision-making. To support theory building, I draw a parallel between the EC theory of ADHD and the common gene–common variant model of complex traits such as ADHD. The conclusion offers strategies for advancing collaborative research.

Keywords: ADHD, Executive control, Attention, Review, Theory

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1 Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a childhood-onset, neurodevelopmental disorder characterized by developmentally inappropriate and impairing restlessness, inattentiveness, and impulsiveness. The condition affects approximately 5% of children globally and is a risk for substantial impairment in later life, which makes it a major mental health concern (Faraone et al., 2021). ADHD is common among adults as well as children, but the current discussion focuses on children and youth because research in childhood ADHD more often includes measures of EC and lessons learned about cognition and childhood ADHD can be generalized to adults with ADHD.

Prevailing theories about the nature of ADHD have changed considerably over the last century. In the late 19th century what was later known as ADHD was considered a disorder of moral dyscontrol or intellectual deficiency that ran in families due to adverse genetic and social circumstances (Still, 1902; Tredgold, 1908). In the 20th century, the neural and behavioral consequences of encephalitis lethargica (1918–1920) highlighted the possible role of acquired brain injury (Kahn and Cohen, 1934). If severe brain injury could cause serious behavioral and intellectual consequences, doctors surmised that mild or minimal brain damage (MBD) arising from undetected or subtle trauma (such as that arising during pregnancy or delivery) could cause the less severe restlessness, inattentiveness, and impulsiveness that defined ADHD (Knobloch and Pasamanick, 1959).

Fifty years ago, the prevailing dogma regarding ADHD's central, underlying abnormality shifted once again to emphasize cognitive deficit; specifically, impaired attention, or executive control (EC). Virginia Douglas (Douglas, 1972, pp. 259) posited that "a core group of symptoms involving inability to sustain attention and to control impulsivity can account for most of the deficits found in the hyperactive group." The theory that ADHD reflects disordered attention arising from impaired EC processes has served as a durable organizing principle in practice and research for five decades and led to the disorder's renaming as attention deficit hyperactivity disorder (ADD) in the third edition of the Diagnostic and Statistical Manual (*DSM III*)(American Psychiatric Association, 1980) and as attention deficit hyperactivity disorder (ADHD) in *DSM IV*. There have been 93,000 scientific papers on ADHD/ADD and attention or EC since 1972. Almost all large-scale studies of ADHD include measures of EC skills (Epstein et al., 2022). Current diagnostic criteria for ADHD (*DSM-5*; American Psychiatric Association, 2013) largely reflect impaired EC: poor focused attention (fails to give close attention or is easily distracted); difficulty sustaining attention (avoids tasks that require mental effort over a long time); problems planning and organizing attention (has trouble organizing); weak working memory (loses things, is forgetful); and deficient inhibitory control (has difficulty waiting for their turn, interrupts). The overlap between the diagnostic criteria for ADHD and definition of EC skills raises the possibility of circularity in research studies of EC and ADHD. Those individuals who meet criteria for the inattentive subtype of ADHD might exhibit EC reflecting poor focused and sustained attention and those with the hyperactive-impulsive subtype might have greater problem with inhibitory control.

The EC theory of ADHD posits that EC deficits lie at the core of ADHD and mediate the link between underlying causes — whether they be neural, genetic, or environmental — and the

behavioral manifestations of ADHD. The EC theory of ADHD is not just an abstract theoretical construct, but one that enters and shapes clinical encounters when patients and parents ask about the validity of an ADHD diagnosis, the underlying cause of ADHD, and the nature of therapies. Increasingly, measures of EC are becoming part of standard clinical assessment (Conners et al., 2003; Epstein et al., 2003) and EC is beginning to shape therapy, as can be seen in the surge of interest in digital therapeutics and attention training. Practitioners want to know whether EC deficits can help them understand the disorder and explain it to their patients and hope that measuring EC skills can improve diagnostic accuracy, outcome prediction, and treatment.

This review presents a primer on EC and its role in ADHD. First, I define EC, and discuss the diverse methods of measuring EC. Then, I summarize evidence supporting eight hypotheses derived from the EC theory of ADHD. Finally (section 4), I suggest areas for further research and offer a reconceptualization of the role of EC in ADHD — an omni-EC theory of ADHD, which parallels current omnigenic models of ADHD and other complex disorders. Has the EC theory advanced our understanding of ADHD as predicted by its early advocates? Has it resulted in better clinical practice? I argue in conclusion that it has and that EC fills a critical gap in our understanding of the link between ADHD's causes and its symptoms. However, scientific challenges remain even after 50 years of EC research.

2 What is EC?

EC processes organize and manage more basic processes, such as perception, thinking, memory, affect, and action, so that we can meet our immediate and longer-term goals in an ever-changing and uncertain environment. In everyday parlance, EC is often called "attention," because it is key to our ability to "pay attention." According to Mesulam (1998) EC is a function of cognition which reflects all neural processing linking sensation to adaptive responses (thinking, feeling, and action) and to meaningful experiences, including consciousness. In animals, cognition tends to be largely reactive, reflexive, unvarying, and sensation-driven. As a result, amphibians are great fly catchers! Humans have larger brains and more synapses than amphibians, allowing for complex association networks that permit more rapid configuration and reconfiguration of the cognitive system to optimize both immediate and longer-term goals. Consequently, humans can influence the way that sensations link to responses and experience. We can withhold, stop, and shift our actions, feelings, and thoughts; we can prepare, sustain our attention, and divide our attention so that we can respond quickly and accurately; and we can hold and update information for future use. We can flip among these abilities as circumstances and goals dictate and deploy them in rapid succession if needed. These abilities are what we mean by EC. Through their influence on lower-level processes, such as memory, action, and sensation, EC processes are key to this adaptive configuration and volitional self-regulation — and possibly to conscious perception (Martín-Signes et al., 2019).

EC links to mental health in several ways. Poor EC makes a person less able to cope with changing environmental circumstances and stress or to manage emotions, make plans, and learn from experience. EC abilities influence one's attraction to short-term over long-term rewards. The vulnerability associated with EC difficulties might be nonspecific, such that every

EC deficit leads to poor outcomes, or there might be more specific links between certain EC processes and particular outcomes. For example, poor inhibitory control has a prima facie link to the impulsivity associated with ADHD, conduct disorder, or substance use disorder.

There is considerable diversity in the way that EC processes are defined. There is also a wide variation in the tasks that are used to measure EC and in the measures of EC that are derived from EC tasks (Banich et al., 2009; Diamond, 2013; Jurado and Rosselli, 2007). Most models of EC include inhibition, working memory, and cognitive flexibility (Miyake et al., 2000; Stuss, 1993; Zelazo, 2020). But there are other EC processes, such as planning, error processing (monitoring for, detecting, and adjusting to errors), dividing and sustaining attention, delaying responses, and temporal processing. The distinction between processes that involve EC and those that do not can be blurred as in the role of inhibition in memory retreival (Penolazzi et al., 2014). Some EC models distinguish between processes that are effortful (controlled) and those that are not (automatic) (Friedman and Robbins, 2022), and some distinguish between cold cognition (rational, top down, prefrontal cortical) and hot cognition (emotional, reward-driven, bottom up, limbic and ventral prefrontal) (Rastikerdar et al., 2023).

Each EC process can be subdivided even more. For example, inhibitory control has been operationalized as the ability to suppress the influence of goal-irrelevant stimuli so as to inhibit distraction (Stroop or Eriksen Flanker tasks); to withhold responses before they are initiated (go/ no-go task, continuous performance task (CPT), or delay-discounting tasks); or to cancel responses after they have begun (stop-signal task or Wisconsin Card Sorting Test (WCST)). Bari and Robbins (2013) found that the concept of inhibition had been defined, operationalized, and measured in 18 different ways across the studies they reviewed. The same diversity of definitions is found in all EC processes. Sustained attention can be operationalized as maintaining performance over a prolonged period (such as minutes or hours as required by a radar operator, sentry, or assembly-line worker), without slowing one's responses (increased reaction time, RT) or showing lapses in attention (reaction time variability, RTV) (Chee et al., 1989; van Schie et al., 2021). And working memory similarly can be parsed into verbal and visuospatial working memory and into holding and manipulating information in memory (Cowan, 2022).

A clear understanding of EC skills is also impeded by the lack of correspondence between EC processes and the performance metrics that researchers choose to measure these skills (Figure 1). Not only are multiple laboratory tasks used to measure response inhibition, but also each task uses different metric to describe performance. The stop-signal task calculates stop-signal reaction time (SSRT), which is an estimate of the latency of the stopping process measured in milliseconds (Logan and Cowan, 1984). The CPT indexes inhibition as the proportion of false positive responses (responding when withholding was required); the WCST reports inhibition as perseverative errors (continuing to respond even when sorting rules have changed; Miles et al., 2021); and the Stroop task assesses inhibition as the effect of distraction (e.g., the speed of reading color names printed in black versus those printed in conflicting colors) (Scarpina and Tagini, 2017). Even though each of these tasks and metrics purports to measure "inhibition," heterogeneity in performance metrics creates challenges for those wishing to aggregate data across multiple studies (Mar et al., 2021).

To make matters even more complex, almost every laboratory task has been used to measure more than one EC process. For example, in addition to indexing inhibition, both the stop-signal task and the CPT measure RT and RTV to assess preparedness, attention, and arousal; in addition, the CPT measures lapses of attention (omission errors) and sustained attention (deterioration in performance on a task over time) (van der Meere et al., 1991). Similarly, multiple performance indices can be derived from the WCST (e.g., failures to stick with an established sorting rule and the difficulty switching to a new sorting rule) (Scarpina and Tagini, 2017).

Insert Figure 1: Mapping EC processes to tasks and performance metrics

Finally, to understand what EC is and isn't, it is important to distinguish it from IQ. Whereas EC measures are intended to reflect specific cognitive processes, intelligence as measured in IQ tests reflects a global estimate of overall EC, neurocognition, comprehension, learning, motivation, and social context, and is beyond the scope of this review (Dennis et al., 2009; Gustavson et al., 2022). When IQ has been used as a covariate in studies of specific EC processes, it accounts for a small proportion of the variance in scores but typically does not alter group comparisons (e.g., ADHD-diagnosed individuals compared with typically developing peers) (Schachar et al., 2023).

2.1 One or many EC processes?

How many EC processes are there and how critical is the distinction among them? There is a vigorous "unity" versus "diversity" debate (Marklund et al., 2007) in EC research, akin to the lumping and splitting debate in theories of psychopathology (McMahon and Insel, 2012; Thaxton et al., 2022) and intelligence (Howard, 1993; Sternberg and Kaufman, 1998). The unity argument holds that all EC processes are manifestations of a single, underlying process or mechanism, such as a supervisory attention system (Norman and Shallice, 1981) or central executive (Baddeley and Hitch, 1974). Unity theories argue that there is a general factor akin to global IQ that accounts for much of the variation in EC measures.Therefore, it might not matter which EC processes are measured in a particular study because they are essentially equivalent.By contrast, the diversity argument holds that EC processes are substantially distinct. Observed interrelatedness arises because EC processes are influenced by some developmentally prior EC function that shapes performance (e.g., Barkley, 1997) or because they depend on partially overlapping subprocesses or neural substrates (see section 4, Discussion). Task choice matters a great deal if diversity is the most accurate explanation.

As is usual in lumping versus splitting debates in biology, the evidence lands between unity and diversity but tends to indicate that there is considerable diversity among EC processes. Correlations between EC processes are rather low (e.g., working memory and flexibility = 0.15; inhibitory control and flexibility = 0.15) (Blokland et al., 2017; Lee, 2014; Macare et al., 2014). Intercorrelation across tasks tend to be greater among younger individuals compared to older ones, hinting at increasing diversity with age (Bardikoff and Sabbagh, 2021; Best and Miller, 2010; Brydges et al., 2014; Buss and Spencer, 2014; Friedman et al., 2011; Garon et al., 2008; Gulberti et al., 2014; Karr et al., 2018; Lehto and Elorinne, 2003; Miyake et al., 2000; Müller et al., 2012; Wiebe et al., 2011). The diversity argument is supported by the different patterns of

neural activity associated with various EC processes (Chevrier and Schachar, 2010; Hannah and Aron, 2021). On the other hand, twin studies show that there tends to be considerable sharing of genetic influences across EC processes (Bollen, 2002; Friedman and Miyake, 2017). While the debate is ongoing, this review takes the position that diversity in EC is important. We revisit this point in the discussion.

2.2 Why are there so many different laboratory measures of EC?

EC tasks arise from two psychological traditions. Most have evolved from a clinical neuropsychology tradition with its focus on the localization of brain injury (e.g., WCST). Fewer tasks have developed from a cognitive or experimental psychology tradition (e.g., stop-signal paradigm). Neuropsychological tests acknowledge the multiplicity of cognitive processes that are invoked when performing a task in addition to the specific cognitive process purportedly being measured (task impurity). But most neuropsychologically inspired tasks do not use designs that allow distinction of the target process from the overall task performance.

By contrast, tasks evolving from the cognitive experimental tradition explicitly aim to isolate a single EC process by using multiple conditions (factorial design) to probe the integrity of the process. N-Back working memory, which manipulates working memory load to study the effect on immediate recall, and the Stop-Signal Task , which manipulates stop-signal delay (time between signal to respond and the signal to stop the response) to examine the effect on probability of stopping a response, are examples of cognitive experimental tasks. Critically, cognitive paradigms are interested in the difference in performance when conditions that manipulate the process under study are changed (the effect on recall of increasing working memory load, the effect on probability of stopping a response of variation in stop-signal delay). In cognitive experimental tasks, overall perfromance (main effect) is affected by multiple EC processes and nonspecific factors, such as motivation, comprehension, or anxiety, but the interaction between conditions supposedly reflects the basic process that the task was designed to study.

There are advantages and disadvantages of each approach. Experimental tasks are purer measures of a specific process but are usually longer to administer (because of the need for factorial design) and therefore tax motivation and attention. Neuropsychological tasks tend to be more familiar and available, but it is difficult to know which specific EC process is being measured and more difficult to construct equivalent versions for repeated measurement as are required in longitudinal and intervention research where the effect of practice could influence performance. The task impurity problem likely contributes to the higher inter-correlation among neuropsychological tasks than experimental cognitive tasks described above.

2.3 The role of self-rated EC skills

Should EC processes be measured using rating scales rather than laboratory measures as some have proposed (Barkley and Murphy, 2010; Biederman et al., 2008; Gioia et al., 2002)? Research shows poor agreement between executive skills rated by parents or by patients and the same apparent skills measured with laboratory tasks. Instead of predicting performance on laboratory tasks measuring EC processes, parent- and self-reported EC skills tend to correlate with ADHD symptom ratings (Anderson et al., 2002; Mcauley et al., 2010; Toplak et al., 2009;

Vriezen and Pigott, 2002). Apparently, ratings and laboratory measures reflect largely distinct phenomena. What a parent or teacher thinks is poor cognitive flexibility, for example, may not be the same as what is measured in the laboratory, and EC skills measured in the laboratory might not reflect the EC processes that are typically deployed in a person's day-to-day life. While rating scales may be less precise, they have the advantage of reflecting behavior over a far longer time frame than laboratory measures. Self-rating of poor attention could be helpful to practitioners in formulating a comprehensive understanding of a person's ADHD symptoms and impairment, whether or not it correlates with performance on laboratory tasks. Consequently, parent-rated, self-rated, and laboratory measures of EC skills might each contribute to a complete understanding of a person's ADHD symptoms (Biederman et al., 2008; Dhillon et al., 2020; Toplak et al., 2009). This review, however, focuses exclusively on laboratory measures of EC.

3 Hypotheses for the role of EC in ADHD

Insert Table 1 about here

3.1 EC deficits are commonly found in ADHD

There is an extensive body of research demonstrating inferior performance on EC measures in participants with an ADHD diagnosis compared with the performance of typically developing age-matched peers (see Pievsky and McGrath [2018] for a meta-analysis of meta-analyses). People with an ADHD diagnosis have deficits in a wide range of EC processes, including verbal and visuospatial working memory (Ramos et al., 2020), response inhibition (Lipszyc and Schachar, 2010; Wright et al., 2014), the ability to shift one's attention from one task to another (Irwin et al., 2019), sustained attention (RTV; Kofler et al., 2013), delay aversion (Jackson and MacKillop, 2016; Sonuga-Barke et al., 2008), decision-making (Dekkers et al., 2022; Patros et al., 2016), verbal fluency (Gustavson et al., 2019), and planning (Patros et al., 2019). The overall size of the observed deficits is small to moderate (mean = 0.45; SD = 0.27) with estimates ranging from a low of 0.35 (set shifting) to a high of 0.66 (RTV) (Pievsky and McGrath, 2018). In other words, variation in an individual person's EC explains only a modest amount of variation in their ADHD symptom severity. EC deficits are more pronounced in younger individuals with ADHD (Pievsky and McGrath, 2018), coinciding with the age at which they are most challenged by academic tasks and self-regulation (Beauchaine et al., 2018).

What this impressive body of research does not prove is that one EC process is more critical to the understanding of ADHD than any other (c.f., Castellanos and Tannock, 2002). Many rather than few EC deficits are found in individuals with ADHD, with each one helping to explain a small-to-medium amount of the variation in ADHD symptom severity. How theories of ADHD can accommodate this multiplicity of EC deficits is discussed below.

3.2 EC deficits confirm an ADHD diagnosis

ADHD is a diagnosis based on descriptions of child behavior in various settings gathered from parents, teachers, and other knowledgeable informants, along with a practitioner's direct observation and judgement. In making the diagnosis, the clinician considers age-appropriateness, impairment, other psychopathologies, and the broader social, psychological, and academic context along with the severity and persistence of ADHD symptoms. But there

are circumstances when a more objective way of making an ADHD diagnosis is desirable (Groom et al., 2016; Hamadache et al., 2021). There is often disagreement between mothers and fathers and/or between parents and teachers on the presence and severity of ADHD traits and therefore on the appropriateness of an ADHD diagnosis. Disagreements arise for various reasons. Impressions of ADHD traits can be influenced by the mood of the informant, their familiarity with normal child development, or the presence of co-occurring child behaviors, such as aggression, anxiety, or social-skills deficits (Abikoff et al., 1993; Molina et al., 1998; Schachar et al., 1986). Under these circumstances, clinicians desire objective means of diagnosis. Unfortunately, individual EC measures, even the ones that seem to best predict ADHD, fall far short of the sensitivity and specificity needed for a valid diagnostic test (Zakzanis, 2001). For example, an effect size of 0.5 for the difference between ADHD and control cases in a single measure of EC corresponds to an overlap between ADHD-diagnosed and typically developing children of 66.6%. Stated another way, only 33.4% of people with an ADHD diagnosis would have performance that falls outside of the range exhibited by age-matched, typically developing controls. The other 66.7% would be false negatives: Their performance would be within the normal range on that measure of EC. Moreover, a substantial proportion of typically developing children would perform poorly and be false positives for an ADHD diagnosis. Raising diagnostic thresholds to reduce false positives among unaffected children would reduce the ability to correctly detect ones who are truly affected .

One intriguing possibility is that a more sensitive and specific diagnostic test can be constructed by measuring multiple EC deficits, rather than any single one, or by combining behavioral and EC measures (Nikolas et al., 2019; Tallberg et al., 2019). For example, Solanto et al. (2001) found that the combination of a measure of delay aversion and a measure of response inhibition correctly classified more individuals with ADHD than either measure alone. It is not clear whether this increase in accuracy indicates that a) some ADHD participants have one kind of EC deficit while others have a different deficit, as argued in Sonuga-Barke's (2002, 2003) dual-pathway model; b) whether the combination of two particular deficits is critical; or c) whether multiple measures improve precision because they generate a more precise estimate of cognitive deficit. There are not many studies addressing what might be called the EC-profile question. Given the considerable number of EC measures, far more of this kind of research will be necessary to validate a diagnostic test. Using multiple EC measures to construct deficit profiles is discussed further below.

3.3 EC measures can clarify diagnosis in the presence of comorbidity

Comorbidity is the co-occurrence of two or more distinct disorders in a particular patient at the same time. This is not a rare event in the case of ADHD. In fact, most people diagnosed with ADHD meet the criteria for one or more concurrent disorder, such as oppositional defiant disorder, conduct disorder, anxiety disorder, or a learning disability (Faraone et al., 2021). Comorbidity challenges the practitioner to determine which of the comorbid disorders is the correct or primary diagnosis.

The comorbidity of ADHD and autism spectrum disorder (ASD) is a good example of the challenge presented by comorbidity. Until *DSM–5*, a diagnosis of ASD precluded another diagnosis (a hierarchical diagnostic rule). With *DSM–5*, it became possible to make more than

one diagnosis in the presence of ASD. This nosological shift revealed the high comorbidity of ADHD and ASD (ASD+ADHD). Individuals with an ASD diagnosis present with comorbid ADHD in 40%–70% of cases (Antshel and Russo, 2019; Grzadzinski et al., 2016; Joshi et al., 2017; Lyall et al., 2017; Mutluer et al., 2022). Even those who do not meet the criteria for comorbid ADHD show high levels of ADHD traits (Pehlivanidis et al., 2020). The overlap of ASD and ADHD is asymmetrical: More ASD cases exhibit comorbid ADHD than ADHD cases exhibit a formal ASD diagnosis or ASD traits, but many people with ADHD have marked social-skills deficits of the type that can be seen in ASD (Luteijn et al., 2000; Nijmeijer et al., 2009; Pehlivanidis et al., 2020).

The high rate of comorbid ASD+ADHD raises numerous important clinical and theoretical questions. There are different possibilitied. The ADHD diagnosis given to those with ASD could represent a true comorbidity which carries the clinical and etiological features of ADHD or it could be a phenocopy of ADHD, with none of the clinical or etiological correlates typical of ADHD. A third possibility is that ASD+ADHD is a condition that differs from both ASD and ADHD and has its own distinct correlates (Caron and Rutter, 1991). One way to determine which of these hypotheses is accurate is to see if the correlates (biomarkers) of ADHD traits found among ASD patients are the same as the correlates of ADHD traits found among ADHD patients who do not have an ASD diagnosis. If a particular individual has true comorbid ASD+ADHD, then that individual should display the biomarkers (i.e., EC deficits in this context) of both ADHD and ASD. If the patient does not show biomarkers of both conditions (e.g., they show biomarkers of ASD but not ADHD), then the conclusion could be reached that the ADHD was an epiphenomenon of ASD — something that looks like ADHD but is not. These distinctions could have implications for treatment and the scientific understanding of ASD+ADHD.

We would understand comorbid ADHD and ASD better if there were distinct EC biomarkers of ASD and ADHD. According to some researchers, people with a diagnosis of ADHD have poorer response inhibition and sustained attention than those with ASD (Antshel and Russo, 2019; Corbett et al., 2009; Craig et al., 2015; Sanz-Cervera et al., 2015), individuals with ASD exhibit worse cognitive flexibility and planning than those with ADHD (Craig et al., 2015; Happé et al., 2006; Lukito et al., 2017; Salcedo-Marin et al., 2013) and those with ASD+ADHD show all of these deficits. This pattern of EC deficits supports the conclusion that ASD+ADHD is a true comorbidity. However, these conclusions are based on indirect comparisons, with individuals with ADHD compared to typically developing controls (Lipszyc and Schachar, 2010; Pievsky and McGrath, 2018; Pineda-Alhucema et al., 2018) and those diagnosed with ASD similarly compared to control subjects (Demetriou et al., 2018). A better understanding of the distinct EC profiles of these two disorders requires direct comparison of individuals with ASD, those with ADHD, as well as control subjects in order to control for comorbidity, experimental tasks, choice of performance metric, testing conditions (e.g., in or out of an MRI machine), age, sex, and medication status during testing (Buti et al., 2011).

We conducted a scoping review and meta-analysis of studies in which there was a direct comparison of participants with a diagnosis of ADHD or ASD along with a control group of typically developing children of the same age with neither ADHD or ASD. We found 58 studies involving a total of 9,923 ADHD, ASD, and control participants (Townes et al., in press).

Although this is an impressive body of research, its limitations were readily apparent. There was considerable variation in the EC skills that were measured and in the indices of performance derived from each EC measure. Response inhibition was the most frequently studied EC process (N = 32 studies), followed by working memory (N = 20), focused attention (N = 18), flexibility (N = 15) and planning (N = 12). However, a wide array of measures for each EC was employed and different performance indices were reported for each skill, making the synthesis of the literature unconvincing. Ninety-eight separate tasks were involved in these studies, including the Go/No-Go task (N= 11), WCST (N = 9), Stroop Color Word Test (N = 6), CPT (N = 7), CANTAB Spatial Working Memory Task (N = 6), and Stop-Signal Task (N = 5). Our meta-analysis found no differences between ASD and ADHD in any EC skill, although people with ADHD and those with ASD performed worse than controls in all domains except planning. The largest of the five studies reporting on response inhibition using the Stop-Signal Task (Karalunas et al., 2018) suggested that individuals with an ASD diagnosis had a deficit in response inhibition compared to people with an ADHD diagnosis, even when controlling for comorbid ADHD (see also Van Hulst et al., 2018). But two studies found no difference in response inhibition between people with ASD, those with ADHD, or controls (Kuijper et al., 2015, 2017), and one study reported that individuals with ADHD, but not ASD, showed a deficit in response inhibition (Albajara Sáenz et al., 2020). Few studies included a group with ASD+ADHD (Demetriou et al., 2018; Pievsky and McGrath, 2018; Pineda-Alhucema et al., 2018).

We took a closer look at the difference between ADHD and ASD in a comparison of children with ASD and those with ADHD on measures of response inhibition, reaction time variability, and reaction time using a large sample of clinical cases and a general-population sample (Schachar et al., 2023b). (Our study did not include putative EC markers of ASD, such as measures of cognitive flexibility, planning, or emotion processing.) In both clinical and community samples, individuals with ASD and with ADHD were compared to each other and to controls. As in the Karalunas et al. (2018) study, we found comparable deficits in response inhibition in individuals with ASD and with ADHD compared to controls. Similarly, reaction time variability was greater in both ASD and ADHD patients than control participants. We did not observe group differences in reaction time. When we controlled for ADHD traits among individuals in the ASD group, EC deficits were reduced to non-significance, showing that the observed EC deficits were largely manifestations of comorbid ADHD, not of ASD. By contrast, the EC deficits in the ADHD group remained after controlling for ASD traits, for which we used a rating scale of social communications. There was a clear correlation between the severity of ADHD traits and the EC abilities that we measured in this study and that relationship was similar across participant in the ASD, ADHD and control groups (no statistical interaction was seen between ADHD trait severity and diagnosis of ASD, ADHD or controls). The more severe the ADHD traits, the greater the observed EC deficit. These results show that deficits in response inhibition and RTV are markers of ADHD traits across disorders. Stated another way, ADHD that presents as part of ASD carried the same implications for response inhibition and reaction time variability as did ADHD in the absence of ASD, suggesting that the ADHD found in individuals with ASD is a true comorbidity, rather than a non-specific epiphenomenon or feature unrelated to “primary” ADHD. On the other hand, people diagnosed with ASD who lack ADHD traits or symptoms did not show atypical inhibitory control or reaction time variability. Because ADHD

proved to be a trait that carried equivalent implications across disorders, our findings support dimensional notions of psychopathology over categorical ones. We have found the same general pattern with ADHD and comorbid anxiety (Korenblum et al., 2007), conduct disorders (Schachar and Tannock, 1995), and obsessive-compulsive disorder (OCD) (Mar et al., 2022). There is a need for similar research into the EC skills associated with comorbid ADHD in other conditions. Future research should examine whether the characteristics of ADHD traits, for example genetic or neural, are the same across disorders.

3.4 EC deficits are impairing

Practitioners and patients can see the impairing consequences of restless, inattentive, and impulsive behaviors more readily than they can judge the impairment associated with variation in EC abilities that are measured in a laboratory setting. However, research increasingly shows that EC skills predict academic, emotional, and mental health impairments (c.f., Barkley, 1991). For example, EC measures predict emotional dysregulation, even after controlling for ADHD traits (Groves et al., 2022); social skills, including the ability to read facial emotions (Jaisle et al., 2023); scholastic attainment, literacy and writing (Al Dahhan et al., 2022; Horowitz-Kraus, 2015; McDougal et al., 2022; Soto et al., 2021); and quality of life (Irwin et al., 2021). EC deficits predict ADHD-trait severity, and response to stress (Marko and Riečanský, 2018; Shields et al., 2016).

Using the data set in the comorbidity study described above, we estimated that ADHD is associated with a delay in development of about two years in response inhibition and reaction time variability compared to typically developing peers (Schachar et al., 2023b). A developmental lag of this size is substantial in children 6 to 14 years of age. And we found that the EC deficits in individuals with ADHD were as great as those found in children who suffered a closed head injury (Leblanc et al., 2005; Ornstein et al., 2013). By following children with brain injury, we learned that their EC deficits often moderate over time and normalize about two years after injury (Leblanc et al., 2005). By contrast, EC deficits in ADHD persist from childhood into adolescence (Mcauley et al., 2013). Taken together, these data suggest that EC deficits of the size found in ADHD are likely to be developmentally significant and impairing.

3.5 EC deficits predict outcome

Like ADHD itself, EC deficits tend to persist and predict future mental health and wellbeing (Brocki et al., 2010; Crosbie et al., 2013; Manfro et al., 2021). Yang et al. (2022) conducted a meta-analysis of 167 longitudinal studies involving 66,119 participants that addressed the relationship of EC abilities when first measured (baseline) and future mental health and found that baseline EC scores predicted ADHD, conduct disorder, oppositional defiant disorder, substance use, and depressive symptoms, but not anxiety symptoms. Predictions were significant but not strong and were greater in clinical samples than in community ones. There was little evidence for relationships between particular EC deficits and specific outcomes, except that so-called “hot” EC processes (those involving reward) were more strongly predictive of substance use and so-called “cold” EC processes were clearly associated with internalizing symptoms, like depression. Prediction was stronger in younger children than older children. Yang et al. found a clear gap in research into the effect of EC capabilities on individuals' outcomes beyond adolescence. In addition to the aforementioned outcomes, individuals with

EC problems identified at a young age are less likely than their unaffected peers to attend school and more likely to have lower achievement, levels of literacy, and adult earnings (Allan et al., 2014; Alloway and Alloway, 2010; Blair and Razza, 2007; Deer et al., 2020; Devine et al., 2016; Manfro et al., 2021; Ribner et al., 2023; Tamm et al., 2021). EC problems identified early in life predict social-skills deficits later in life (Devine and Hughes, 2014; Rinsky and Hinshaw, 2011), worse physical and mental health, greater criminal activity, and a lower quality of life (Kenworthy et al., 2022; van der Plas et al., 2021).

3.6 EC skills play a role in medication response

Stimulants (methylphenidate and dextroamphetamine) are the most common drugs used to treat ADHD. These drugs are indirect dopaminergic and noradrenergic agonists, acting in prefrontal-cortex and striatal-catecholamine pathways. Stimulants significantly reduce the severity of ADHD behaviors and associated impairment (effect sizes of -0.78 for methylphenidate and -1.02 for amphetamines; Cortese et al., 2018). Within the range of the typical dosage (0.3 to 1.0 mg/kg), higher doses tend to have greater impact on ADHD symptoms, even though residual symptoms can remain even at higher doses.

If EC plays an important role in the mechanism of ADHD, then stimulants should have a beneficial impact on EC. That seems to be the case, although the impact on EC is less obvious than on ADHD behaviors (Cortese et al., 2018; Tamminga et al., 2016) and responses vary with the EC process being measured. Differential effects of stimulants on various EC skills could arise because different areas of the brain are "tuned" to distinct levels of dopamine activity, according to the Yerkes–Dodson law. Consequently, the beneficial effect of stimulants varies by EC process and dose (Williams and Goldman-Rakic, 1995; Witt et al., 2021; Zahrt et al., 1997), and can have maximal effect at lower doses than the dose that effects the greatest reduction in ADHD behavior (Tannock and Schachar, 1992;Tannock et al., 1995; Rubio Morell and Hernández Expósito, 2019).

The partial dissociation of behavioral and EC effects in drug response raises an important clinical question. Under typical clinical conditions, the dosage of stimulants is adjusted to address ADHD behaviors, not EC performance. If behavior and EC processes are optimized at different doses (higher for behavior; moderate for EC processes; Tannock et al., 1992, 1995), it is likely that EC is not optimized in clinical practice. This dissociation might explain why some EC skills, such as vigilance, divided attention, phasic and tonic alertness, response inhibition, and focused attention, are improved by medication (Fallon et al., 2017; Mckenzie et al., 2022; Scheres et al., 2003), but others, such as task-switching, are impaired (Rajala et al., 2020).

Consistent with the hypothesis that EC plays an etiological role in ADHD, EC skills measured before treatment begins predict both behavioral and cognitive response, but not in all ADHD cases or in all studies (Coghill et al., 2007; Fosco et al., 2021; Golubchik et al., 2019; Idema et al., 2021; Kapur, 2020a, 2020b; Mckenzie et al., 2022; Molitor and Langberg, 2017; Rubio Morell and Hernández Expósito, 2019; Scheres et al., 2003). A review of 25 relevant studies found that task performance was useful for predicting the response to treatment with methylphenidate (Molitor and Langberg, 2017). For example, Scheres et al. (2003) found that children with inferior inhibitory control prior to treatment benefited less from methylphenidate at standard

doses and needed higher doses for best symptom relief than children with better inhibition. More research is needed to better understand the dose-response relationship of stimulants on multiple EC processes in clinical practice and the potential for pretreatment EC performance to predict treatment response (Coghill, 2021).

3.7 EC training improves ADHD

If EC plays a central role in the mechanism of ADHD, improving EC directly through training or medication should "rescue" the deficit and improve ADHD symptoms. Lambez et al. (2020) reviewed studies of various non-drug interventions for EC deficits and ADHD. These therapies included neurofeedback (Van Doren et al., 2019), physical activity (Cerrillo-Urbina et al., 2015), cognitive training (Peñuelas-Calvo et al., 2022), and cognitive behavior therapy (CBT; Catalá-López et al., 2017). The effects on EC processes of these non-pharmacological interventions were comparable in medicated and unmedicated participants with an effect size of 0.67. Physical exercise showed the highest effect size of 0.93, comparable to the effect of pharmacological interventions. Cognitive training showed the lowest effect size of 0.45. CBT interventions and neurofeedback showed moderate effects (0.70). Non-drug interventions tended to affect inhibitory control the most and working memory the least, even though working memory has been targeted more often than other EC skills.

Westwood et al. (2023) conducted a meta-analysis of the rapidly growing literature involving computerized cognitive training (CCT). Of the 36 randomized control trials that met the inclusion criteria for their review, 17 studies focused on memory training. In studies where the informant was probably unaware of whether that participant was receiving active or placebo treatment (the gold standard in treatment studies), there was no effect on ratings of the patient's hyperactivity-impulsivity symptoms but there was a small improvement in their inattentiveness. Effects were larger when ADHD participants were assessed in the setting where the intervention was delivered. CCT improved working memory and visuospatial skills, but not other EC processes or academic outcomes (e.g., reading, arithmetic). There were some persistent effects in verbal working memory and reading comprehension — as well as in the ratings of EC skills — but Westwood et al. identified few relevant trials targeting these EC processes. The authors found no evidence that CCT training that targetted multiple EC at the same time was superior to working-memory training alone. The beneficial effect of cognitive rehabilitation appears to be greater for preschoolers than for school-age children with ADHD (Scionti et al., 2020). In summary, there is growing evidence that EC can be improved with non-pharmacological interventions, but to date, there is less evidence that improvement in EC performance predicts ADHD symptoms.

3.8 EC deficits play a mechanistic role in ADHD

ADHD fits the model of a multifactorial, threshold disorder with a major genetic component. Multiple common (single nucleotide polymorphisms [SNPs]), rare genetic (copy number variants [CNVs] and single nucleotide variants [SNVs]) variants contribute to ADHD, along with various environmental risk factors, which could act directly or indirectly via epigenetic mechanisms (Faraone and Larsson, 2019; Castelo et al., 2022; Massera et al., 2023). According to this theory, genetic variation, brain injury, or environmental influences are not the proximal causes of restlessness, inattentiveness, or impulsiveness. Instead, various risks increase the likelihood of

alterations in proteins, brain structures and neurotransmitters that support vital neural functions such as EC, and it is the variation in EC processes that causes ADHD traits (Bernier et al., 2012; Castelo et al., 2022; Castro et al., 2023; Diamond, 2020; Galván and Rahdar, 2013; Shields et al., 2019; Yan and Rein, 2022; Zelazo, 2020).

Insert Figure 2: Multifactorial threshold model of ADHD

Individual genetic variants that have been identified in ADHD account for a trivial amount of the variance in ADHD trait severity across patients. However, the combination of these variants explains a significant, although small amount of variance in ADHD (Green et al., 2022). A considerable proportion of the variants that influence ADHD (84%–98%) is shared with other psychiatric disorders (ASD, schizophrenia, and major depressive disorder), and with traits such as smoking initiation, educational attainment, and insomnia (Demontis et al., 2023). Among the variants associated with ADHD, there is an overrepresentation of those that are upregulated during early embryonic development and those that are expressed in the dorsolateral prefrontal cortex and in midbrain dopaminergic neurons. These are the brain regions that are involved in EC processes and in stimulant effects. To date, only a small proportion of the estimated number of variants affecting ADHD have been found, but more are expected to be revealed by whole genome sequencing.

According to the EC theory of ADHD, EC skills are influenced by the genetic risks for ADHD and variation in EC skills is manifest in ADHD behavioral traits. If so, EC skills ought to be heritable and share genetic risk with ADHD. Twin studies indicate that EC skills are indeed heritable (Blokland et al., 2017; Friedman and Miyake, 2017; Guimaraes et al., 2020; Gustavson et al., 2022). And polygenic risk scores for ADHD (a measure of disease risk arising from common genetic variants) are associated with EC processes, such as attention; working memory; and facial memory (Aguilar-Lacasaña et al., 2022; Demontis et al., n.d.; Maier et al., 2015; Moses et al., 2022; Nigg et al., 2018). The total number (burden) of CNVs that are found in ADHD patients also predict inhibition and sustained attention (Zarrei et al., 2023). And there is growing evidence that the statistical association between polygenic risks scores for ADHD and ADHD symptom severity is reduced when variation in EC processes are included as mediators in the analyses. This kind of mediation analysis indicates that genetic risks generate variation in EC skills such as working memory, reaction time variability, and inhibition, and that this variation in EC skills predicts variation among individuals in ADHD behavior (Nigg et al., 2018; Moses et al., 2022).

4 Discussion

4.1 Summary of evidence in support of Executive Control theory of ADHD

This review finds support for many of the hypotheses derived from the EC theory of ADHD. There is consistent evidence that children and teens with ADHD perform more poorly on a range of EC processes than their age-matched peers who don't have ADHD, supporting the most fundamental hypothesis of the EC theory of ADHD. Those with an ADHD diagnosis show a lag in development of about two years or more in EC skills, and the lag seems to be greatest among younger individuals. EC deficits are impairing and persistent and they compromise

future scholastic achievement, social function, and mental health. There is wide variation in EC skills in the general population and in affected individuals, with a considerable overlap of the two groups making the boundary between healthy and impaired EC difficult to discern. Many typically developing individuals have EC performance in the same range as those with ADHD and many with ADHD have EC in the normal range. The relationship of ADHD and EC appears to be quantitative: The greater the EC deficit, the more severe are the ADHD traits. The quantitative relationship of ADHD traits and EC holds across ADHD and unaffected individuals which indicates that the trait-based model of psychopathology can be extended to include EC measures.

Although some EC deficits show larger effect size than others, no single EC process, task, or performance metric is definitively more strongly and reliably associated with ADHD than any other, and none explains more than a modest amount of the variation in ADHD traits among individuals. The association of EC processes and ADHD is not sufficiently strong for a single EC measure to diagnose ADHD. By contrast, there is evidence that combinations of EC skills deficits can distinguish affected from unaffected individuals with greater sensitivity and specificity than deficits in single EC abilities. Which combination of EC measures to use and how they should be combined (e.g., profiles, clusters, or latent traits) remains to be determined.

Further research is needed to determine if ADHD and other mental health conditions have similar EC deficits. So far, it looks as if the apparent overlap in EC performance between ADHD and other conditions such as ASD arises from ADHD traits that are commonly found in the other condition. When ADHD or ADHD traits are taken into account, commonly comorbid disorders such as ASD do not show the same EC performance deficits that are observed in ADHD. However, only a limited number of EC processes have been studied in ADHD and comorbid conditions with adequate control over comorbidity at a disorder and at a trait level (c.f., Karalunas et al., 2018). The lack of control over comorbidity in cross-disorder studies of EC limit conclusions about whether there are EC measures that can distinguish ADHD from another disorder.

This review does not support any of the existing theories that argue for a necessary and sufficient deficit in a particular EC process in ADHD patients. For example, Barkley's (1997) posited that response inhibition is the central neurocognitive problem in ADHD. However, the size of the deficit in response inhibition in ADHD patients is comparable to that of other EC processes and is not the largest effect seen. That honor belongs to RTV an index of EC that deserves far more research (Epstein et al., 2022; Sonuga-Barke and Castellanos, 2007). Models that stress the leading role of working memory or error processing find no more support than those that emphasize inhibition (Castellanos and Tannock, 2002). On the other hand, models that incorporate multiple EC processes better fit existing evidence (Sergeant, 2005; Sonuga-Barke, 2003).

Stimulant medication impacts EC performance and behavior. But EC processes are often optimized at a lower dose of stimulant medication than behavior is, suggesting an incomplete overlap of biological mechanisms. Given that drug response is usually adjusted to optimize behavioral improvement, EC is unlikely to be optimized in clinical practice. Little is known about the long-term implications for learning and behavior of a cognitively oriented dose-optimization

strategy. EC measures partially predict who will have a favorable behavioral response to drug treatment, but again, a limited range of EC processes have been evaluated as predictive markers.

Research suggests that games that train EC processes can improve their performance in various cognitive domains after playing video games for brief periods (e.g., Green and Bavelier, 2003), but replication has been inconsistent (Boot et al., 2008; Sala et al., 2018; Sala and Gobet, 2019). So far, there has been little evidence for short- or long-term reduction in ADHD behaviors of computerized cognitive intervention. Typically, training has targeted a single cognitive process, most commonly, working memory. Given the heterogeneity in EC performance deficits among those with a diagnosis of ADHD, it is not surprising that the effects of such narrowly focused interventions might be inadequate. However, there is little evidence that targeting multiple EC processes improves behavioral outcomes (Westwood et al., 2023).

Evidence is mounting that EC processes mediate the link between underlying genetic or neural risks and ADHD behaviors. Both ADHD and performance on EC tasks are heritable, and they are associated cross-sectionally and prospectively. ADHD and EC processes share some genetic risks, neural correlates, and neurochemical pathways. However, the relationships between genetic risk, EC processes, and ADHD appear weak given current evidence: Neither EC performance nor genetic risk factors individually explain more than a modest amount of the variation among people in ADHD symptom severity.

4.2 Research agenda

4.2.1 Improving our understanding of EC in ADHD

Despite the progress that has been made, gaps remain in the basic understanding of EC and in the measurement of EC skills. There has been a tendency to “reify” EC processes. For example, the N-Back task is understood as a measure of working memory; the Stop-Signal Task , a measure of response inhibition; and so on. It is necessary to keep in mind that the EC processes measured in these tasks reflect the outcome of multiple interacting subprocesses. For example, the ability to stop a speeded response involves variation in preparing to execute a response, preparing for the possibility that you might have to stop, stopping per se, detecting performance errors, and adjusting to these errors. The multiplicity of subprocesses involved in EC processes sets up the possibility of convergence and pleiotropy in genetic, neural, and psychopathological characteristics of EC.

For example, multiple EC processes might contribute to ADHD because they converge along a common neural pathway. Thus, poor performance on two different EC tasks could predict an ADHD diagnosis because both EC tasks share some common subprocess, such as error detection. A cognitive pathway, such as deficient response inhibition, might be a result of problems in one subprocess in one disorder or affected individual (e.g., deficient error detection) and a different subprocess in another disorder or affected individual (e.g., inefficient stopping process), creating the impression of pleiotropy. An apparently common EC deficit could arise in diverse disorders for different reasons (poor preparation to stop in ADHD versus sluggish information processing in schizophrenia).

Additionally, there is both degeneracy and redundancy in the neural substrates of EC. Multiple neuronal systems can support a single EC process or subprocess (many-to-one) and a single neuronal system can support multiple EC processes (one-to-many). The absence of a one-to-one mapping of structure and function arising from degeneracy and/or redundancy helps account for the recovery of cognitive processes after injury but adds complexity to the study of structure-function relationships (Noppeney et al., 2004). Imaging of performance on EC tasks that allow for the separation of the various subprocesses that occur within a single trial will facilitate needed research into the subprocesses involved in the performance of EC tasks relevant to ADHD (Chevrier and Schachar, 2020). Future research could examine which of the EC subprocesses are more strongly and specifically associated with ADHD than with other disorders, and which are more biologically informative and therefore the best targets for discovery, assessment, intervention, or personalized risk prediction.

4.2.2 Improving EC measurement

Substantial strides have been made in standardizing experimental measures for use in mental health research (NIH Toolbox)(Gershon et al., 2010; Karr et al., 2023; Taylor et al., 2022). However, the ADHD research community needs a set of measures that is both pertinent to ADHD in particular and to mental health in general in order to facilitate cross-disorder comparisons. The tasks need to be suitable for children, teens, and adults if developmental trajectories are to be studied. Creating standardized measures for EC processes would allow for better estimates of patients' developmental lag to facilitate evaluation, and the change over time to estimate treatment effects.

The ADHD/EC toolkit needs to include instructions for standardizing administration, task parameters, and performance metrics to facilitate the amalgamation of data. Verbruggen and the community of stop-signal-task researchers (2019) proposed such a set of parameters to guide research, including direction about how the task should be administered and analyzed, definitions of outliers and deficit, and standard language for labelling critical variables to facilitate data synthesis. Similar guidelines need to be developed for other ADHD-related EC tasks. Any proposed experimental guidelines need to consider the unique constraints of research with children and youth, especially those with mental health challenges. Tasks should be evaluated to see if they can be shortened without the loss of validity and accuracy. Priority should be given to EC measures that can be modified for use in animals and in neuroimaging to facilitate studies that recreate and rescue attention deficits that are seen in ADHD and other disorders to advance understanding of the effects of neurotransmitters, neural substrates, and medications on attention (Clatworthy et al., 2009; Hvoslef-Eide et al., 2015; Liu et al., 2017; Skandali et al., 2018).

Ideally, the ADHD/EC toolkit should be available on mobile devices to support population-based and longitudinal research (Crosbie and Schachar, 2001; Parsey et al., 2021). Game-like versions of laboratory tasks can increase participant engagement and allow for a longer and repeated testing in order to generate more reliable measures of EC performance. But attention must be paid to the precision of measurement. Tablets and cell phones are not designed to collect reaction time data in milliseconds, although efforts have been made in this regard (https://www.neurobs.com/menu_presentation/menu_features/mobile). A lack of precision

will result in increased measurement error and the loss of statistical power, requiring larger study samples.

4.2.3 Optimizing research design

The field needs to move beyond the study of ADHD patients in comparison with typically developing peer control subjects using a limited set of EC measures. We now need direct comparisons of ADHD and other disorders, particularly those that are most frequently comorbid with ADHD such as ASD and Tourette syndrome. More and more studies feature clustering strategies of various kinds. These studies should go beyond behavioral measures to included measures of performance on EC tasks and other levels of the multifactorial model of ADHD described above. These studies would help determine the extent of EC pleiotropy, the sharing of biological mechanisms and could discover novel groups of patients who share EC, structural, genetic and other informative characteristics (Kushki et al., 2019). Studies of EC in comorbid disorders will require the measurement of comorbid traits as well as EC, and they should include EC measures that are hypothesized to be related to the comorbid disorders (e.g., social cognition or cognitive flexibility in ASD). These studies could yield insight into the mechanism of ADHD, clarify the nature of comorbidity, and help refine nosology. Longitudinal studies are needed to evaluate the association between EC deficits detected early in life and later ADHD traits and EC skills. The combination of family and longitudinal designs will help identify EC deficits that confer risk for development of ADHD and other psychopathologies.

Drug and non-drug treatment studies should consider a person's unique profile of EC strengths and deficits (a precision EC approach). To date, drug effects on EC processes have been assessed as if all cases of ADHD have the same EC deficits — an observation that is not supported by this review. Many EC deficits improve with medication, but some may deteriorate; drug effects might depend, in part, on baseline EC performance, which undoubtedly differs among participants (Golubchik et al., 2019). The consequences of the differential impact of stimulants on EC and on behavior require further study. The lack of a precision EC approach might account for the failure of cognitive interventions to improve the behaviors associated with ADHD. Future digital therapeutic research should be tailored to each person's actual EC deficits rather than assume that everyone has the same deficits as current studies tend to do. Training to improve an EC process in someone who has no problem in that EC skill might be both a waste of effort and detrimental to patient engagement. Treating every participant in a clinical study as if they have the same EC performance deficigts would decrease the measured impact of these interventions.

4.1.4 Omni-EC deficit model of ADHD

The parallels between the findings on EC summarized above and the current state of genetic research into ADHD are striking and suggest directions for future EC research. Early expectations for genetic research into ADHD were great because twin studies showed that ADHD was highly heritable. Candidate gene research in ADHD was predicated on the notion that one or a few genes conferred risk for ADHD (oligogenic) and that these genes would likely be ones that involved neurotransmitters, given the known effect of stimulant medication. However, few of these initial findings could be replicated (Dumas-Mallet et al., 2016). We now know that there are many genetic risks for ADHD and that they involve a wide range of

potential biological pathways. There is no “gene for ADHD.” The largest contribution to the ADHD phenotype comes from the cumulative impact of common variants, those that are found in more than 1% of the population — not just in affected individuals. There is a small contribution from rarer CNVs and SNVs found in less than 1% of the population. Some risks for ADHD are inherited from parents, and some arise *de novo* (Lionel et al., 2011).

Although certain genetic risks might be more penetrant (have a greater effect) than others, for the most part each one makes a very small contribution to the phenotype. Collectively, common variants contribute significantly to the risk of developing ADHD, as is evident in polygenic risk scores (Demontis et al., 2023). Genetics researchers predict that studies with larger samples and more precise genetic methods such as whole genome sequencing will lead to the identification of more risk variants. From what we know currently, there is both a many-to-one relationship (equifinality) and a one-to-many relationship (pleiotropy) linking genetic variants to various mental and physical health conditions. Some of this apparent genetic heterogeneity will be reduced when we discover the way that genes effect biological mechanisms. ADHD is rightly considered a genetically complex trait. But rather than being daunted by this genetic complexity, the field is looking toward a time when progressively more sensitive genetics techniques will increase the amount of variation in ADHD that we can explain.

Similarly, ADHD is a cognitively complex disorder, which might be termed “omni-EC” (all EC processes). Just as single genetic variants do not account for much of the variance in ADHD, no single “candidate” EC process explains more than a moderate amount of the variance in ADHD. It is important to note, however, that the explanatory power of a single EC measure, although modest, is far greater than that of any single genetic variant. In that respect, ECs have narrowed the search for pathways leading from first causes to the disease phenotype. Both genetic risks and EC show pleiotropy and equifinality. Individual EC processes seem to confer risk for various psychopathologies and different EC deficits clearly converge on single disorders.

Just as polygenic risk scores have increased the power of genetics to predict disease risk, there is preliminary evidence that the cumulative effect of multiple EC deficits explains more of the variation in ADHD in individual patients than does any single EC measure. And, like genes, we do not yet understand how EC processes combine to influence ADHD. EC effects could be additive, interactive, or multiplicative and are highly likely to be influenced by sex and environmental factors, such as neural trauma, social experience, and environmental toxins, in the same way that genes are affected by epigenetic factors.

4.1.5 Multi-EC risk index

The parallels between genetic and EC theories of ADHD suggest strategies for combining the effects of all EC deficits to improve the prediction of psychopathology, treatment response, and outcome, and to explore similarities and differences among mental disorders (Anttila et al., 2018). The effects of multiple EC deficits could be combined by putting each EC performance measure on a common metric, such as T-scores (using population norms). T-scores could be used to create an index of overall EC impairment or to identify profiles of EC strengths and weaknesses that distinguish ADHD from typically developing children and from those with other

disorders. Total EC-risk scores and profiles could be tested for their ability to predict risk for ADHD in high-risk populations, as well as outcome, treatment response, and various biological characteristics. In addition to multi-EC indices, the field could use clustering strategies to find associations among EC processes and biologically informative characteristics. For example, all EC processes that predict cortical thickness, future comorbidity, learning difficulties, or a particular genetic pathway could be considered as a cluster for further genetic study.

Most of what we have learned about EC and ADHD has been based on relatively small-scale studies involving one or a few EC skills. The future will require larger samples, greater standardization of measures and reporting, and intensive collaboration akin to the Pschiatric Genetics Consortium (PGC) or Enigma consortia (De La Vega and Bustamante, 2018; Xiong et al., 2019).

5 Conclusion

The early protagonists of the EC theory of ADHD would hopefully be satisfied with the progress that we have made since their groundbreaking insights (Douglas, 1972). There is now a substantial body of evidence supporting the key hypotheses derived from the EC theory of ADHD, and EC measures are being applied in genetic, neuroimaging, and therapeutic research. Understanding of EC offers considerable advantages in the study of psychopathology. EC measures can be standardized more easily than clinical assessments, thereby reducing the need for training of research staff and improving the reliability of data in the kind of cross-site studies that are required to address outstanding issues. EC processes are not directly focused on psychopathology, which reduces ethical and participant concerns (especially in general-population and classroom-based studies), and they can be useful in imaging research, because they can be directly related to measures of function, which can be parsed into subprocesses.

The EC-deficit hypothesis for ADHD has generated multiple insights into the role of EC in psychopathology and offers hope for sharpening our understanding of the causes of this common, costly, and impairing mental health condition. Fundamentally, EC processes are links between the suspected genetic and neural causes of ADHD and the disorder itself. They are a bridge between biology and action, thought and choice (Martín-Signes et al., 2019). Even if we were to identify all genetic, neural, and other correlates of ADHD, we would still be left with the challenge of explaining how those risks “cause” ADHD. ADHD is a problem of self-control that is best studied by examining self-control. Better understanding EC can play a significant role in bridging this gap.

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8 Figures and Captions

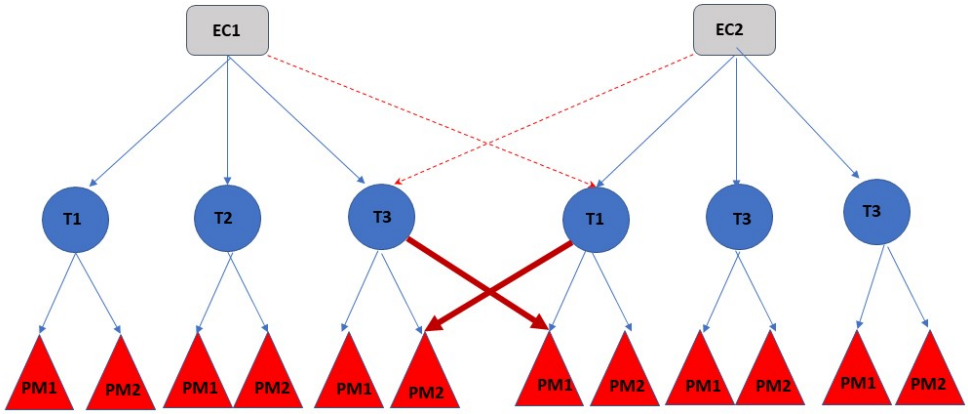

Figure 1: Mapping EC processes to tasks and performance metrics

Multiple EC process (ECP1…n; grey rectangles) can be measured with many different tasks (T1…n; blue ovals), each of which report performance using various performance metrics (PM1…n; red triangles). PMs are not unique to a single task (or EC process) but similar PMs can be derived from multiple tasks measuring various EC processes. Blue arrows depict the mapping of EC processes to various tasks and tasks to performance metrics. Dotted red arrows illustrate how an EC process can be measured by a task typically associated with a different EC process. Solid red arrows show how a task can generate performance metrics relevant to more than one EC process.

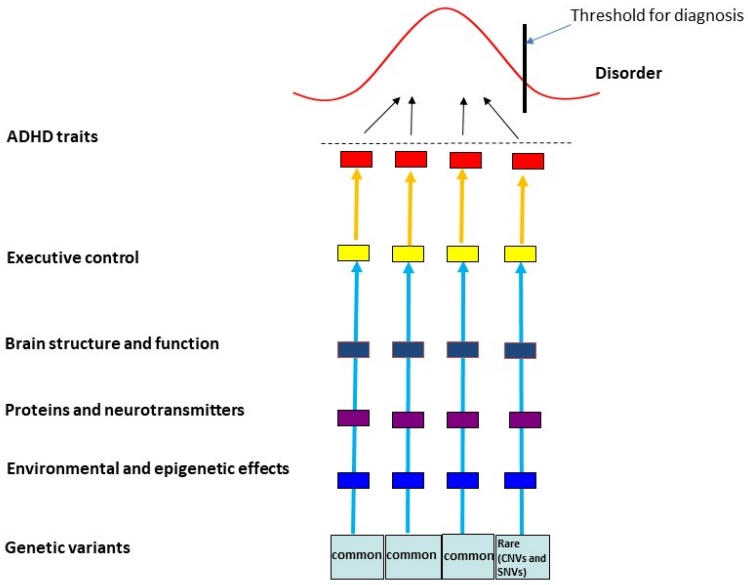

Figure 2: Multifactorial threshold model of ADHD

Genetic (common and rare) variants, acting in combination with environmental risk factors, start a cascade of effects on brain structure (proteins and neurotransmitters) and function, which impacts executive control and generates a wide range of ADHD traits (red normal distribution) in a cumulative, quantitative fashion. When the net effect of these risks on behavior exceeds a certain threshold (perpendicular black line), a disorder is diagnosed.

9 Tables

Table 1: Eight hypotheses derived from the Executive Control theory of ADHD

3.1	EC deficits are commonly found in ADHD
3.2	EC deficits confirm an ADHD diagnosis
3.3	EC measures can clarify diagnosis in the presence of comorbidity
3.4	EC deficits are impairing
3.5	EC deficits predict outcome
3.6	EC skills play a role in medication response
3.7	EC training improves ADHD
3.8	EC deficits play a mechanistic role in ADHD