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Executive function deficits in attention-deficit/hyperactivity disorder and autism spectrum disorder

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Abstract

Executive function deficits have been reported in both autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD). However, little is known regarding which, if any, of these impairments are unique vs. shared in children with ADHD versus ASD. In this Review, we provide an overview of the current literature with a critical eye toward diagnostic, measurement, and third-variable considerations that should be leveraged to provide more definitive answers. We conclude that the field's understanding of ASD and ADHD executive function profiles is highly limited because most research on one disorder has failed to account for their co-occurrence and the presence of symptoms of the other disorder; a vast majority of studies have relied on traditional neuropsychological tests and/or informant-rated executive function scales that have poor specificity and construct validity; and most studies have been unable to account for the well-documented between-person heterogeneity within and across disorders. Currently, the most parsimonious conclusion is that children with ADHD and/or ASD tend to perform moderately worse than neurotypical children on a broad range of neuropsychological tests. However, the extent to which these difficulties are unique vs. shared, or attributable to impairments in specific executive functions subcomponents, remains largely unknown. We end with focused recommendations for future research that we believe will advance this important line of inquiry.

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Author contributions

All authors researched data for the article and contributed substantially to the discussion of the content. M.J.K., E.F.S., L.J.S., S.L.H, E.M.J., J.N.S., K.E.F., and E.D.M. wrote the article and reviewed and/or edited the manuscript before submission.

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Introduction

Attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) are among the most common neurodevelopmental disorders observed in children[1], as shown by prevalence rates of roughly 5% and 1%, respectively.[2–5] ADHD is characterized by symptoms of inattention and/or hyperactivity or impulsivity that must be present prior to age 12 years and are associated with impairment. ASD is characterized by persistent deficits in social communication and social interaction and by restrictive, repetitive patterns of behavior, interests, or activities. The rates of co-diagnosis of ASD and ADHD are as high as 70%[6], and both diagnoses share common clinical characteristics such as onset during childhood, developmental deficits or delays in brain development, and behavioral difficulties and impairments across social and academic domains[1]. However, the nature of these difficulties differs across disorders. For instance, the social difficulties observed in ADHD seem to reflect a performance deficit (such as intrusiveness, inattention to social cues, and impulsive social behavior that result in peer rejection), rather than a lack of social knowledge or skills.[7–9] By contrast, the social difficulties in ASD seem to reflect deficits in social knowledge[10] that result in social disengagement, isolation, and indifference to social cues[11,12] (although the importance of social performance difficulties in ASD is being increasingly recognized[13]).

Deficits in executive function (a set of higher-order neurocognitive processes that enable goal-oriented behavior) have been hypothesized to play key roles in the development and/or maintenance of core behavioral symptoms[14–17] and assessment of executive function plays a significant role in clinical practice, including early detection and intervention planning.[40] Clinical researchers typically use cognitive and behavioral models[18–20] to study shared and unique executive function components within and across diagnostic groups and symptom clusters. An expansive literature in ADHD spanning nearly three decades[29,31–34] posits that executive function deficits are either causal mechanisms that give rise to ADHD behavioral symptoms; non-causal factors that nonetheless aid in developmental recovery from ADHD; or epiphenomenal (neither causes of ADHD nor involved in symptom expression).[38] Similarly, the executive dysfunction hypothesis of ASD (one of several etiological theories of ASD) describes how executive function deficits contribute to core ASD diagnostic symptom domains, including disruptions in social communication and increases in restricted interests and repetitive behaviors.[5,35–38]

One of the most empirically supported and influential models of executive function[21,22] — the unity and diversity model[18]— proposes that there are three interrelated but uniquely specific and separable executive function components: working memory, inhibitory control, and set shifting.[9] ‘Unity’ in this model refers to correlations between the three components, which are presumed to reflect a common underlying ability, whereas ‘diversity’ acknowledges that the components are also unique and separable.[22] Developmental studies suggest that executive function abilities are present before three years of age, but specific executive function components are not yet discernible at this age.[23] Indeed, executive function abilities continue to develop exponentially in early childhood,[24] with working memory and inhibitory control becoming separable abilities in preschool and early school-aged children,[21,25,26] and set shifting emerging as a unique ability in late adolescence or

early adulthood.[21] All three executive function components continue to develop and peak in young adulthood (approximately age 25) before plateauing and/or naturally diminishing with age.[24]

Furthermore, the three components in the unity and diversity model support a host of secondary higher-level cognitive processes. For example, deficits in one or more of these executive function components have been implicated theoretically and/or experimentally in functional and behavioral outcomes relevant to ASD and/or ADHD, including difficulties with organizational skills,[27] planning,[18,27,28] interference control (the suppression of interference due to resource or stimulus competition),[29] goal-maintenance,[30] vigilance,[31] response consistency,[15,29] delay tolerance,[32] academic achievement and success,[33] learning behaviors such as task engagement and persistence,[34] social skills,[35] emotion regulation,[36] on-task behavior and visual attention,[37] and self-control and regulation of motor activity.[38] This evidence provides a clear and compelling rationale for clarifying the unity and diversity of executive dysfunction in ASD and ADHD.[39]

In this Review, we provide the first critical review of studies examining executive function profiles in ADHD vs. ASD based on rigorous methodological criteria informed by the 'unity and diversity' model and current best practice recommendations from the cognitive literature. We begin with a non-critical overview of the current evidence supporting and/or refuting executive function deficits in ADHD and ASD. Whereas prior reviews[41,42] have generally accepted the 'executive function' construct labels used by the cited authors, our narrative review builds on prior work by introducing critical conceptual and measurement limitations, as well as construct validity concerns with clinical and neuropsychological executive function tests and behavioral ratings[41,42]. Then, we unpack executive functions to introduce the idea that the overlap in deficit profiles between ADHD and ASD might be due to deficits in different subcomponents that produce similar performances on executive function tests but for different reasons. Despite advancements in the methods and techniques to measure executive function in children, accurate assessment of executive function components remains challenging. Based on insights from what we believe are current best practices for executive function measurement and differential diagnostics, we then critically revisit the literature using a set of rigorous methodological benchmarks. Finally, we conclude with a series of evidence-based recommendations that we hope researchers will use to develop and conduct new studies that provide more definitive answers regarding the unity and diversity of executive function profiles in ASD and ADHD.

Non-critical overview

In this section, we summarize the evidence supporting or refuting the presence of deficits in the three executive function components (working memory, inhibitory control, and set shifting) and non-specific executive functioning (studies that combine scores from tests intended to measure multiple executive function domains) in children and youth with ADHD (Table 1), ASD (Table 2) and co-occurring diagnoses (Table 3). We prioritized available meta-analytic and systematic reviews in our summary of findings. In this initial overview, we have generally accepted the diagnostic and test construct labels used by the cited authors. We describe the results in terms of effect size, which in this context refers to

the estimated magnitude of the impairments for each disorder. A Cohen's *d* effect size of 0.20 is considered small (noticeably smaller than medium but not so small as to be trivial), *d* = 0.50 is considered medium (deficits that are visible to the naked eye of a careful observer) and *d* = 0.80 is considered large.[43]

Working memory

Working memory refers to the active, top-down manipulation of information held in short-term memory, including the mental ability to hold, manipulate, and update multiple pieces of information.[16,20] Working memory is arguably the most common executive function deficit in youth with ADHD.[53] Deficits on tests on tests intended to measure working memory are consistently among the largest deficits of any executive function component[54] in studies of youth with ADHD, with some meta-analytic estimates as high as *d*=0.69–0.74.[55] Meta-regression estimates reach higher effect sizes (*d*=2.01–2.05)[55] when analyses focus specifically on tests that place sufficient demands on the 'working' components of working memory: processes that require active monitoring of incoming information and replacing outdated information with relevant information (continuous updating), maintaining information in mind while performing a secondary task (dual-processing), and/or maintaining and rearranging information in mind (or serial and temporal reordering).[55–58] Meta-analytic estimates of working memory deficits are smaller in preschoolers with ADHD compared to children and adolescents with ADHD (*d*=0.32).[59] This result might be due to substantial differences in the tasks administered to preschool-aged youth, which sometimes are simplified versions of the tasks given to children and adolescents, or are research-assistant administered game-like tasks.[60]

Meta-analyses have also consistently identified deficits on tests of working memory among individuals with ASD[61–63]. Studies suggest greater deficits on visuospatial (*d*>0.72) compared to verbal (*d*=0.44–0.67) tests.[61–63] Further, effect sizes are larger among school-aged children (*d*=0.62) compared to adolescents (*d*=0.20),[61] although some meta-analyses report no age effects.[62,63] Additionally, some limited evidence suggests substantial working memory deficits in preschoolers with ASD compared to neurotypically developing peers.[64]

When ADHD symptoms are controlled for among ASD samples, effect sizes remain medium for verbal (*d*=0.53) and spatial tests (*d*=0.50).[42,65] Impairments on tests intended to measure working memory remain notable among youth with ASD when controlling for symptoms of ADHD[65] and among individuals without co-occurring ADHD.[66 67] Similarly, these impairments remain notable among youth with ADHD when controlling for symptoms of ASD[68] and among individuals without co-occurring ASD.[69,70] Studies that include both ADHD and ASD groups consistently report substantial impairments on tests intended to measure working memory relative to neurotypically developing peers across both groups.[42,68,71] Evidence suggests greater working memory impairment among individuals with ADHD compared to ASD.[71] Studies also demonstrate greater impairment in ADHD and ASD co-occurring groups relative to neurotypical peers (*d*=0.65),[67,69,70] but similar working memory performance relative to ASD-only groups.[69,70,72,73]

Inhibitory control

Inhibitory control refers to the ability to withhold or stop an on-going response, particularly within the context of goal-directed behavior.[74] Youth with ADHD show deficits on tests intended to measure inhibitory control, with medium effect sizes (d=0.52)[54] compared to neurotypical youth, and preschoolers with elevated ADHD symptoms show small-to-medium deficits overall (d=0.49).[60] Two of the most commonly used inhibitory control tasks in the ADHD literature are the stop-signal task and go/no-go task, which test response inhibition.[59,74–76] Studies based on these tasks show medium meta-analytic effect sizes in school-aged youth through adulthood (d=0.49–0.63)[75–77] and medium effect sizes in preschoolers (d=0.37–0.87).[59] By contrast, findings related to inhibitory control are generally mixed and meta-analytic effect sizes are null or small-to-medium when interference control tests are used (such as Stroop, flanker and Simon tasks).[59,60,78,79]

Early meta-analyses of inhibitory control deficits among youth with ASD revealed small to medium effect sizes on tasks similar to those used in the study of youth with ADHD (response inhibition: d=0.55; interference control: d=0.31)[80]. A meta-analysis identified a small-to-medium effect (d=0.48), with younger children showing more pronounced deficits than adolescents (preschool d=0.72; vs. school-aged d=0.56; vs. adolescence d=0.42).[81]

Youth with either ADHD or ASD demonstrate impaired performance on tests intended to measure inhibitory control compared to neurotypically developing peers.[42,67,68,70] Children with ADHD continue to show poor inhibitory control test performance after controlling for ASD symptoms.[68] Similarly, children with ASD continue to show poor inhibitory control test performance after controlling for ADHD symptoms.[65] When comparing ADHD to ASD groups, some evidence suggested greater inhibitory control impairment among individuals with ADHD compared to ASD,[67,71] but other findings indicated the two groups exhibit equal levels of impairment.[72] Co-occurring ADHD and ASD groups demonstrated impaired inhibitory control relative to neurotypically developing peers[67,69–71] and the ASD-only group.[67,69,71] By contrast, one review demonstrated comparable inhibitory control test performance among co-occurring ADHD and ASD and ASD-only groups.[72] There are also mixed findings regarding inhibitory control skills among co-occurring groups relative to ADHD-only groups. Co-occurring ADHD and ASD groups exhibited inhibitory control skills comparable to the ADHD-only groups,[69,70,72] but one empirical study demonstrated that the co-occurring ADHD and ASD group exhibited better inhibitory control relative to the ADHD-only group.[71] Further, one review suggested there were no differences in inhibitory control among co-occurring and ADHD-only groups.[72]

Set shifting

Set shifting (also called cognitive flexibility) is defined as the ability to switch flexibly between mental sets.[22] Set shifting has been understudied compared to working memory and inhibitory control among youth with ADHD, and has been associated with relatively smaller effect sizes (youth d=0.35, preschool d=0.26)[54,60]. However, interpreting these findings is challenging given the evidence that set shifting only develops as a separate, unique ability in late adolescence or early adulthood.[21]

Consistent with this developmental evidence, the impairments on set shifting tests in youth with ADHD might not be due to the tests' set shifting demands per se, but to the inhibitory control and/or working memory demands required to perform these tests.[83–85] Set shifting has also been studied less than the other executive function components among youth with ASD. However, empirical work shows that youth with ASD exhibit very large deficits compared to neurotypical children on tests assessing perseverative errors (continuation of same response strategy following a rule change) (d=2.17–2.55)[86] and small-to-medium-sized difficulties maintaining a new ruleset following a successful initial shift (d=0.46).[66,87] Additionally, set shifting performance seems to be more impaired among children than adolescents with ASD.[88]

When controlling for ASD symptoms, the evidence is mixed as to whether or not youth with ADHD continue to display deficits on set shifting tests.[68,89] By contrast, children with ASD continue to show impaired performance on tests intended to measure set shifting compared to neurotypical individuals after controlling for co-occurring ADHD symptoms, with a medium effect size (d=0.61) across tasks.[65] Youth with ASD also show worse set shifting compared to youth with ADHD[69] and co-occurring ADHD and ASD;[72] however, some studies show no group differences[42] and one study observed better performance among youth with ASD compared to neurotypical and co-occurring ADHD and ASD peers.[71] Co-occurring ASD and ADHD groups show medium-sized deficits relative to neurotypical peers (d=0.60).[71]

Non-specific executive functioning

Under 'non-specific' executive functioning we review findings that are collapsed across tests of the three executive function components, as well as tests of additional neurocognitive and behavioral processes that are, at least in part, considered to be outcomes of the three core executive functions in the 'unity and diversity' framework (such as planning, organizing, and attentional focus) .[22,27,30] Several large meta-analyses and mega-analyses offer conclusions about the magnitude of non-specific executive function deficits in youth with ADHD and ASD. According to a review of 34 meta-analyses, youth with ADHD exhibit medium-magnitude deficits compared to neurotypical youth on non-specific executive functioning (d=0.45), with larger deficits among children (d≈0.50) than adolescents (d≤0.30). Comparable meta-analytic effect sizes have been identified among preschoolers with ADHD (d=0.32–0.64).[42] Similarly, according to a meta-analysis of 235 studies, the average non-specific executive functioning deficit in youth with ASD compared to neurotypical youth is in the medium range (d=0.48).[61]

Consistent with patterns reported among neurotypical populations,[90] estimates of group-level impairments do not necessarily inform between-person heterogeneity in executive function across individual youth with ADHD and ASD. Specifically, although a majority of youth with ADHD (89%) have a deficit in one or more executive function components, individuals differ in which component is impaired (approximately 75–85% of youth with ADHD have impairments in working memory, 21–46% in inhibitory control, and 10–38% in set shifting).[53] Only 4% of children with ADHD show impairments in all three components, highlighting the limited clinical utility of non-specific measures of executive functioning.[53]

Concerning ASD, approximately half of youth with ASD (47%) show executive function deficits in one or more executive function components.[91] Thus, not all children with ADHD or ASD have executive function deficits. However, emerging evidence suggests that this executive function heterogeneity may prove fruitful for understanding heterogeneity in functional impairments for children with ADHD and/or ASD.[8,53,91–93]

Taken together, the available literature on school-aged children suggests that ADHD and ASD might be associated with moderate deficits on non-specific indices of executive functioning, and moderate-to-very-large deficits in working memory specifically. Further, ADHD might be associated with greater working memory and inhibitory control deficits compared to ASD (medium-to-very large deficits in ADHD vs. small to medium deficits in ASD), whereas ASD might be associated with greater set-shifting deficits compared to ADHD (medium-to-very-large deficits in ASD vs. small-to-null deficits in ADHD). However, methodological and diagnostic issues call these preliminary conclusions into question and highlight the need for more rigorous research to clarify the unity and diversity of executive function profiles in ASD vs. ADHD.

Limitations of the current evidence base

In this section, we discuss the challenges in drawing conclusions about similarities and differences in executive function profiles in ASD and ADHD from the current literature base. Specifically, we discuss the diagnostic challenges in differentiating between ASD and ADHD and consider the accuracy, precision and comprehensiveness of traditional test batteries.

Diagnostic uncertainty

The extent to which ADHD and ASD are associated with similar executive function impairments is complicated by several diagnostic challenges.[115,116.] Specifically, there is concern regarding the validity of current gold-standard diagnostic methods to differentiate between ASD and ADHD symptoms. For example, none of the items on the gold-standard Autism Diagnostic Inventory – Revised (ADI-R) adequately differentiate ASD from ADHD.[11,94] By contrast, select items from the Behavior Assessment System for Children-3 (BASC-3) and the Autism Diagnostic Observation System (ADOS-2) might accurately differentiate ASD from ADHD, although both measures fail to adequately differentiate ADHD from ASD.[11,95] Differential diagnosis is also challenged by diagnostic overshadowing (the attribution of co-occurring symptoms to a disorder that has already been diagnosed when it is actually indicative of a co-occurring condition).[96] For example, symptoms of ASD might be misattributed as ADHD symptoms in a child diagnosed with ADHD and vice versa.[96,97] Such problems with differential diagnoses can reduce the validity of the research base and negatively impact children and their families due to inaccurate diagnoses and delays in implementing appropriate treatments.[97]

Co-occurring conditions and symptom variability pose further challenges. Unfortunately, current methodological and classification approaches are likely not sensitive enough to capture the full range of within-group and between-group symptom variability. For example, intellectual developmental disorder (defined as a standardized intelligence score at least 2

standard deviations below the mean with associated impairment) is more common among individuals with ASD (19–35%) than among the general population (2–3%).[98] Further, the prevalence of co-occurring ASD and intellectual developmental disorder is 30–40%[98]. Similarly, 8%-39% of children with mild and borderline intellectual developmental disorder have ADHD.[14,41,99] However, ASD and ADHD studies typically exclude children with low intellectual quotient (IQ) and/or intellectual disability[100]. This is an important limitation because working memory is a (likely causal) predictor of global IQ[101–103] and age-related improvements in working memory lead directly to improvements in IQ.[103] Thus, the methodological decision to exclude individuals based on IQ has the unintended consequence of excluding children based on their working memory abilities. This decision inadvertently yields an incomplete picture of the heterogeneity and nature of executive functioning profiles in ASD and ADHD.

Similarly, most ASD samples are limited to children with milder or more subtle symptom presentations who require minimal support (for example, participants meeting criteria for the lowest severity category, 'Level 1, Requiring Support'), rather than individuals who present with more severe symptoms and require substantial support for daily living. Consequently, executive function results of ASD samples might not generalize across the broader autism spectrum.

An additional limitation stems from the fact that ADHD and ASD are usually diagnosed using nosological frameworks that conceptualize psychological disorders as fundamentally distinct and orthogonal (such as the *Diagnostic and Statistical Manual of Mental Disorders*)[104]. This categorical approach often fails to adequately capture clinically relevant symptoms that fall outside the diagnostic criteria in complex, heterogeneous, and highly co-occurring diagnoses and thus likely conflates ADHD and ASD between-group differences[104,105]. By contrast, dimensional approaches that conceptualize clinical presentations based on the frequency and severity of broad symptom dimensions (such as the "RDoC" Research Domain Criteria Initiative[95,104]) capture variability within ADHD and ASD and are used in research to differentiate disorder-specific deficits within broad areas of impairment[106–108]. However, dimensional diagnoses have not been linked to reimbursable mental healthcare services and their use in clinical practice is limited. Reconciling categorical and dimensional approaches within the realities of managed care healthcare systems will be critical to avoid further widening the research-to-practice gap.

Construct validity of neuropsychological tests

Interest in executive function and its measurement has grown significantly across diverse fields including clinical science, cognitive science, neuropsychology, and developmental psychology. This piqued interest likely resulted from theory, research and empirical evidence linking executive function deficits to various psychopathologies (including neurodevelopmental disorders) and to adverse functional outcomes in clinical and non-clinical pediatric populations.[19,69,109] As research and clinical interest grew, there was a corresponding commercial interest that featured a proliferation of executive function tests and measures marketed to clinicians. In some cases, conceptually-derived (rather than empirically-derived) executive function subscales based on pre-existing questionnaire

items were added to broadband rating scales based on pre-existing item content. In other cases, performance-based 'executive function' tests for children were published that were fully or partially comprised of pre-existing tests originally designed to detect gross neuropsychological and frontal lobe deficits in adults.[110] For example, the popular digit span test transitioned from a measure of verbal IQ to a measure of freedom from distractibility and is now reified as a test of working memory. Similarly, the trail making test was repurposed from a test of brain injury or gross neuropsychological functioning to a specific test of set shifting.[111] Finally, new tests and measures were developed psychometrically, but frequently had smaller normative samples and were in most cases not adopted in widespread clinical and clinical-research practice.[112] Thus, it might be unsurprising that the now-traditional executive function tests most frequently used in clinical practice lack the sensitivity and specificity necessary to capture the global and specific executive function deficits that are characteristic of children with neurodevelopmental disorders.[109]

Specifically, there is a preponderance of evidence questioning the construct validity and test specificity of most of the traditional norm-referenced, performance-based neuropsychological tests of executive function widely used in clinic settings,[29,109] including the Delis–Kaplan Executive Function System,[113] the Woodcock-Johnson III Tests of Cognitive Abilities,[114] the Developmental Neuropsychological Assessment–II,[115] and executive function-relevant factors from the several editions of the Wechsler intelligence test batteries[116]. Much of the criticism of these tests points to the fact that these measures are too broad in scope, lack specificity to assess executive function components, and were developed to assess gross frontal lobe dysfunction (for example, secondary to traumatic brain injury or in people with dementia) rather than the more subtle executive function deficits associated with psychopathology.[109,117] Independent evaluations of these test batteries indicate that their subtests contribute meaningfully to a composite measure of global IQ or global neuropsychological functioning (psychometric *g*)[118] , but do not provide a valid assessment of executive function components and distinct constructs when compared to well-validated performance-based executive function tasks from the cognitive literature.

For example, a sizeable proportion of the variance in the Delis–Kaplan Executive Function System executive function subtests was attributable to a general factor *g* rather than to the specific executive function components described in the test's interpretation manual.[119] Similarly, a re-analysis of the Developmental Neuropsychological Assessment–II standardization sample indicated that the 23 evaluated subtests do not meaningfully contribute to the assessment of psychometric *g*, or to the tests' intended neuropsychological domains (general factor loadings for most subtests were less than .50, and domain-specific effects for all subtests were even lower). All subtests demonstrated strong subtest-specific effects, but it is not clear what constructs these subtest-specific effects represent.[120]

A similar pattern has been found for traditional clinically-used tests of working memory. For example, factor-analytic studies of the Wechsler Intelligence Scale for Children-V[116] show that up to half (ranging from 24–50% across subtests) of the variance on working memory subtests is attributable to a general psychometric *g* factor, whereas a minimal proportion of1 variance (less than 3%) is attributable to a working memory factor after accounting for the general factor.[118] The same issue has been identified with the working memory factor across

the rest of the Wechsler intelligence scales using the tests' original norming samples as well as independent samples.[118,121–127]. These findings indicate that the Wechsler working memory factor "possesses too little true score variance to support clinical interpretation"[123] and is "not sufficiently reliable for clinical decisions."[128] Thus, the working memory factor in these scales cannot be used for the identification of working memory deficits in research or clinical practice.

For research purposes, modifications to the administration and scoring rules might help overcome some of the limitations of these scales. Modifications might include ignoring the rule to discontinue the test administration after several consecutive fails, administering all trials regardless of patient performance, and scoring patient responses using partial credit unit scoring (counting each stimulus recalled in the correct serial position) rather than the traditional all-or-nothing scoring (awarding a point only if the patient's response was perfect for the complete trial).

A study in a sample of children with ADHD[129] tested these recommendations for working memory assessment from the cognitive literature.[130] Consistent with the factor analytic evidence above, the results revealed that traditional scoring of the Wechsler Intelligence Scale for Children-IV digit span backward subtest (a commonly used task assumed to test working memory) failed to predict working memory or achievement and instead showed moderate correspondence with fluid reasoning (general factor *g*)[131]. However, modifications to the subtest administration and scoring decreased its association with fluid reasoning (from a statistically significant r=.49 to a non-significant r=.15) and substantially increased the magnitude of its associations with latent estimates of working memory, specifically reordering and dual-processing (r=.53-.58) and academic achievement (r=.49).[129] The results indicated that "digit span backward becomes a valid measure of working memory at exactly the point that testing is traditionally discontinued".[129]

We and others have also argued that the working memory tests commonly used in clinical practice place relatively minimal demands on the executive components of working memory and thus might be better conceptualized as measures of short term memory .[53,101,130,132,133] In either case, the construct valid measurement of working memory specifically, and executive functioning more broadly, is currently significantly limited in clinical practice, which has led some cognitive scientists to describe the clinical and neuropsychological literature as engaging in "parallel play" when it comes to executive function measurement.[109] Other construct validity concerns include administration features and analysis and research considerations that should be taken into account when evaluating the utility of the available neuropsychological tests of working memory (Table 4; Supplementary information)

Construct validity of rating scales

Informant-rated rating scales are a convenient and cost-effective method for assessing many of the constructs, syndromes, and symptoms encountered in clinical practice and research. The combination of informant-rated executive function scales and performance-based tests has often been considered the gold standard for clinical and neuropsychological assessment of executive function in children and adolescents.[134,135] However, these two measurement

modalities have shown non-significant to weak associations.[131,136–138] Informant-rated scales only correlate $r \approx .20$ or lower with construct valid, performance-based executive function tests.[131,135,138] Stated differently, 96% of a person's executive function abilities are not captured by informant-rated scales ($.2^2$ = 4% shared variance between informant-rated and performance-based methods for assessing the same construct)[131].

Further, methodological and conceptual issues limit the interpretation of informant-rated executive function rating scale scores and the conclusions that can be drawn from them. Several authors have questioned the content validity of informant-rated executive function rating scales and the evidence supporting their construct and predictive validity. For example, some popular informant-rated executive function rating scales have been criticized for being, essentially, recycled ADHD rating scales, with a majority of items on at least some subscales appearing identical, or nearly identical, to DSM-5 ADHD symptom criteria[131,135,138]. Based on the current literature, our conclusion is that informant-rated executive function rating scales cannot be used to assess neurocognitive abilities, and that more work is needed to clarify what these scales are actually measuring (Box 1).

Taken together, our goal of understanding the unity and diversity in executive function profiles across ADHD and ASD is limited by substantial clinical (differential diagnosis), cognitive (accuracy, precision and comprehensiveness of available test batteries), and research (exclusion criteria, dimensional vs. categorical approaches) challenges. Although traditional neuropsychological tests provided a promising starting point by highlighting the importance of executive functions for understanding both ASD and ADHD, converging evidence indicates that they generally do not provide the necessary level of specificity and construct coverage for reliably measuring the more subtle deficits associated with psychopathology.[109] In the next section, we describe how we believe that leveraging advances in cognitive science can improve understanding of executive functioning profiles in ADHD vs. ASD.

Overcoming limitations

Cognitive and clinical scientists have developed modern performance tests of executive function that are based on models of executive function from the cognitive literature ('cognitively-informed').[22,143] In this section, we discuss these measures and how the results of studies that have used them have begun to improve the field's understanding of executive function deficits in ADHD and ASD.

Modern performance-based tests

In contrast to traditional performance-based neuropsychological tests, modern performance-based executive function tests are supported by cognitive models of executive function.[22,143] These 'cognitively-informed' performance-based tests provide reliable and valid estimates of the executive function components defined in the unity and diversity model[56,109,135]. These tests have also demonstrated ecological validity via robust prediction of important functional outcomes such as academic achievement, social functioning, attentive behavior, and organizational skills.[18,26,27,38,58,131]

The utility of using cognitively-informed measures of executive function for evaluating children with ADHD and ASD is particularly evident when impairment estimates are compared to the estimates yielded by traditional neuropsychological tests. For example, across meta-analyses using traditional neuropsychological executive function tests for children with ADHD, 33%-50% of cases exhibited executive function deficits (30%-37% impaired working memory, 21%-46% impaired inhibitory control).[90,144–149] By contrast, studies using cognitively-informed measures report that 89% of ADHD cases exhibited impairments in at least one executive function (75–85% impaired working memory, 21–46% impaired inhibitory control, 10–38% impaired set shifting).[53,56] Indeed, a study using meta-regression techniques[55] concluded that 98% of children with ADHD score below average or worse on cognitively-informed working memory tests with high demands on the 'working' components of working memory. The large increases in impairment rates yielded by cognitively-informed tests are consistent with critiques suggesting that traditional neuropsychological tests often lack sensitivity and specificity for detecting the subtle executive function deficits associated with these disorders.[109]

Similarly, an empirical study using traditional neuropsychological tests suggested that about 47% of children with ASD demonstrate deficits in one or more executive function components .[91] However, a meta-analysis revealed that these measures generally do not differentiate children with ASD from children without ASD, and concluded that the evidence did not support using these tests to fractionate children's performance into specific executive function components.[61] By contrast, meta-analytic work revealed that using cognitively-informed tests of executive functioning greatly improved discrimination in executive function deficits such as working memory[62] and reaction time parameters[68] in individuals with ASD compared to a typically developing group and/or a ADHD group. Cognitively-informed executive function tests also demonstrated superior predictive and ecological validity compared to informant-rated scales.[131] This result was confirmed using informant-rated scales and performance-based scales of functional outcomes such as academic achievement, and was reported using latent variable analysis that would be expected to maximize test-rating correlations by removing error.

We echo recent recommendations[109] to employ multiple, cognitively-informed measures of each executive function component – ideally assessed across multiple sessions on separate testing days – to maximize construct validity and yield more accurate impairment estimates relative to traditional neuropsychological batteries[109]. Together with latent estimation that models both unique and shared variance across the executive function components,[22,26,58,150] these methodological refinements are expected to substantially increase the specificity and sensitivity of the scores produced by these modern cognitively-informed tests. However, the clinical utility of these tests remains limited because, with few exceptions,[151] they lack the large, nationally representative normative samples needed to draw conclusions about individual patients. In addition, careful attention to these tests' outcome metrics will be critical. For example, the stop-signal test is often considered the gold standard for inhibition measurement but produces fictitious inhibitory deficits if scored using the traditional method (Box 2).

Measuring subcomponents

Similar to how executive function can be fractionated into three primary components, the three primary components seem to be separable into subprocesses. For example, working memory can be divided into its 'working' component (the mental processes that operate on mentally held information) and 'memory' component (short-term memory, the passive storage and rehearsal mechanisms that temporarily hold information in mind). Further, the 'working' component can be fractionated into interrelated but distinguishable subprocesses involving continuous updating, dual-processing and serial-temporal reordering, and the 'memory' component can be fractionated into distinct verbal, visual and spatial short-term storage systems (Figure 1).[20,56,152] Similar subdivisions are also apparent for inhibitory control and set shifting. Using specifically designed test batteries that enable performance to be fractionated into the three primary executive function components and their specific subcomponents will be imperative for better understanding the nuances of executive function strengths and difficulties in ADHD and ASD.

To date, research investigating executive function subcomponents in ADHD and/or ASD has been scarce. Thus, even if research confirmed that ASD and/or ADHD are associated with deficits in a specific executive function component, it would remain unclear whether these deficits are due to the same subprocesses. For example, deficits on inhibition tests might be related to perseverative processes in children with ASD that, in turn, predict engagement in restrictive or repetitive behaviors. By contrast, the same overall test scores in children with ADHD might be related to action-cancellation processes (stopping an in-progress behavior) that, in turn, predict impulsive or verbally intrusive behaviors.[153] Similarly, a conclusion that ADHD or ASD is not associated with deficits in a specific executive function component might be premature if overall null findings are due to strengths in some subprocesses that mask deficits in other subprocesses.

A notable exception to this critique is a study that used a specifically designed test battery to evaluate the three subprocesses of 'working' component of working memory in children with ADHD[56]. Compared to children without ADHD, children with ADHD exhibited large impairments in serial/temporal reordering (d=1.34) and medium-sized impairments in continuous updating (d=0.64), but generally intact dual-processing working memory. This initial study highlights the importance of construct specificity. However, additional analyses also showed that what is shared between these three subprocesses—rather than their unique features—is critical for predicting ADHD symptoms. Thus, careful attention to both the unity and diversity within and across executive function components is needed to advance research in ADHD and ASD.

Executive function profiles revisited

In this section, we revisit the available evidence base to critically review the studies examining executive function profiles in ADHD and ASD. To that end, we developed rigorous methodological criteria derived from our examination of the limitations of available reports and what we believe to be current best practices from the cognitive (executive function measurement) and clinical (differential diagnostics) literatures. We used three primary criteria to re-review the available literature. First, studies should include

both ADHD and ASD samples. Second, studies should describe differential diagnostic methods suggesting reasonable certainty regarding the labeling of comparison groups as ADHD, ASD, co-occurring ADHD and ASD, or neurotypical. In the case of studies using a dimensional approach, construct-valid symptom assessments should be used. Third, studies should include valid cognitively-informed measurements of one or more executive function components (Table 5). Additional criteria including issues of representativeness and generalizability were also considered and impacted the level of certainty/strength of our conclusions and will be further discussed in the following section.

We were able to locate several executive function studies that included both ADHD and ASD samples and provided sufficient diagnostic details to suggest reasonable certainty regarding their clinical groups. However, almost all of the current literature relied on traditional neuropsychological tests that have been criticized for poor construct validity.[109] Specifically, no study to date fully met our benchmarks regarding construct-valid working memory or set shifting measurement. Our conclusion that most extant ADHD and ASD executive function studies failed to meet methodological quality benchmarks to allow firm conclusions is consistent with a recent review[72] that judged every extant co-occurring ADHD and ASD study as methodologically poor or fair (none received a rating of strong).

A partial exception to this conclusion is a study that explored a computationally derived ex-Gaussian index of response inconsistency (called tau)[154], which has been shown to be a causally linked outcome of working memory but not inhibitory control[15,29,155]. In this study, children with ADHD and co-occurring ADHD and ASD demonstrated elevated tau relative to both children with ASD (without ADHD) and neurotypical children, and ADHD but not ASD uniquely predicted tau. Although concluding that working memory is implicated in ADHD but not ASD is arguably a stretch because tau is not solely a reflection of working memory[15,156], this result speaks to the lack of robust evidence regarding unique vs. overlapping working memory profiles in ASD vs. ADHD.

Similarly, a series of studies by some of the authors of the current Review included children with ADHD and ASD, controlled for ASD when evaluating executive functions in ADHD, and used a battery of construct-valid working memory tests. Results indicated that children with ADHD have large working memory deficits and medium-to-null inhibitory control and set shifting deficits relative to children without ADHD.[51,53,56,58,75] Interestingly, however, poor performance on inhibitory control tests was attributable, in large part, to the tests' working memory demands rather reflecting actual inhibition deficits in children with ADHD.[31,152,153] These studies also showed that working memory but not inhibition deficits predict ADHD-related difficulties with emotion regulation, academic achievement and productivity, organizational skills, activities of daily living, inattentive and hyperactive/impulsive symptom severity, information processing speed, and peer relationships.[8,27,31,36,37,58,156,158–161] However, children with ASD comprised only about 10% of our ADHD and non-ADHD samples, and methodological control for ASD was limited to including an equal number of ASD cases in both the ADHD and non-ADHD comparison groups and conducting sensitivity analyses (comparing results when including versus excluding children with ASD). Thus, although these studies provide preliminary

evidence for working memory deficits as a key, likely causal, factor in ADHD, they do not provide data on executive functioning for children with ASD.

By contrast, there is some, albeit still limited and mixed, evidence suggesting that ADHD might be more strongly associated with difficulties on tests of the action restraint (preventing a behavior before it starts) component of inhibitory control than ASD.[42,69,71,72,82] However, these findings are preliminary because they are based on relatively small samples and because none of the available studies also included construct-valid tests of working memory and/or set shifting. This latter point is important because the three executive function components are moderately interrelated, and there is experimental evidence demonstrating that working memory impacts performance on inhibitory control tests (but not vice versa), which could potentially explain why children with ADHD show deficits on inhibition tests.[152,157–159] Indeed, studying any executive function in isolation limits certainty because it is unclear whether the observed deficits are specific to the tests' inhibitory control demands or to the myriad of other executive, neurocognitive, motor, or perceptual processes required for successful performance.[53,83,117]

Evidence-based recommendations

The current literature indicates a substantial problem with task impurity and emphasizes the need for viable solutions.[109,165] We recommend a set of study methods we hope researchers will adopt as guidelines for developing and conducting new studies to provide more definitive answers regarding the unity and diversity of executive function profiles in ADHD and ASD.

Our first recommendation is to use construct-valid, performance-based executive function batteries. Using multi-test approaches for each construct of interest will be critical for identifying the unique and overlapping executive functioning weaknesses – and potentially strengths – associated with these neurodevelopmental conditions. Stated bluntly, it is tenuous — if not scientifically indefensible — to reify any single test as measuring any single executive function.[53,150] Test combinations should be selected to isolate executive function components and subcomponents, and performance should be assessed across multiple sessions on separate testing days – ideally at different times of day – to increase the specificity and reliability of executive function scores, to reduce participant fatigue, and to account for random and time-of-day effects.[130,166,167]

Our second recommendation is to carefully attend to differential diagnostics and consider dimensional assessment. ASD is often viewed as qualitatively different from other neurodevelopmental and clinical disorders. However, replicated evidence supports the view that conditions such as ASD[168,169] and ADHD[170] are extreme ends along natural continuums of characteristics that are normally distributed across the general population. Relatedly, the majority of existing studies rely on diagnostic status to define and compare ADHD and ASD groups, which limits understanding of symptom overlap and heterogeneity within and across these conditions. Using dimensional approaches to conceptualize these two conditions will more fully capture the underlying neurocognitive profiles and etiologies of these symptom dimensions and potentially link specific symptom clusters with specific

executive function vulnerabilities.[171] Of course, studies using categorical approaches will also be valuable. For these types of studies, clear consideration and communication of differential diagnostic challenges and clinical decision-making will be important for readers to assess the integrity of the grouping variable and the generalizability of the findings.

Our third recommendation is to assess and model unique and overlapping aspects of both syndromes. Most ADHD and ASD studies examined executive functioning in one disorder without consideration of the other, and even fewer studies included a co-occurring ADHD and ASD group. More research is needed to examine similarities and differences in executive functioning abilities across these two disorders by including ADHD, ASD, and co-occurring ADHD and ASD samples (as well as neurotypical children) in studies to capture the full range of symptom quantity, frequency, and severity for each ASD and ADHD symptom cluster and functional outcome. Capturing the full range of symptom severity and impairment is particularly important given that most studies to date have recruited only relatively high functioning children with ASD.

Our fourth recommendation is to reconsider IQ exclusionary criteria, and to not covary IQ in data analyses. Most reviewed studies set exclusion criteria based on IQ, which typically excludes children with borderline intellectual disability and intellectual developmental disorder. This methodological decision results in an incomplete picture of the heterogeneity of executive function profiles in ASD and ADHD. Further, it is important not to include IQ scores as a covariate in statistical analyses of neurodevelopmental disorders (see ref[172] for a compelling statistical, methodological, and conceptual rationale). Researchers should reconsider study criteria and exclusionary cut points for intellectual functioning and instead build an executive function test battery based on the expected mental age of study participants.

Our fifth recommendation is to consider third-variable explanations. If overlapping executive function deficits are identified, additional research will benefit from determining whether this overlap is due to shared deficits between ASD and ADHD, to demographic similarities, and/or the increased risk that each condition carries for additional co-occurring syndromes (Box 3).

Our sixth recommendation is to increase access to research studies and improve generalizability of research findings. Understanding differential outcomes based on race, ethnicity, gender, sex, age, and socioeconomic factors[173,174] is highly limited. Although efforts have been made to incorporate more diverse samples, most existing research has studied children, specifically boys. This bias is likely driven by the higher prevalence of ADHD and ASD diagnoses in school-aged boys versus girls.[175,176] Boys also present with earlier onset and more serve symptoms, making them more likely to be identified as having ADHD and/or ASD at a younger age than girls.[177,178] Given this bias, it is difficult to parse apart the extent to which sex differences in executive functions might be present in school-aged children with ADHD and/or ASD. More broadly, like most areas of psychological inquiry, ADHD and ASD research has historically been conducted primarily with White Non-Hispanic children from western, educated, industrialized, rich, and democratic (‘WEIRD’) societies.[179,180] In light of continued inequalities regarding

access to research studies, we recommend active strategies to optimize participation by diverse populations,[181] with a focus on traditionally underserved populations to assess and reflect a broader range of experiences.[182,183]

Summary and future directions

Given the substantial conceptual and construct validity issues discussed in our Review, firm conclusions regarding the unity and diversity of executive function profiles in children with ADHD vs ASD are not warranted at this time. Instead, the most parsimonious conclusion is that children with ADHD and/or ASD tend to perform moderately worse than neurotypical children on a broad range of performance-based neuropsychological tests that likely place at least some demands on executive functions.[109,117] However, the extent to which these deficits are attributable to impairments in executive function components and subcomponents remains largely unknown. The unfortunate consequence is that there is currently very little knowledge about specific strengths and weaknesses in executive functioning within and across ASD and ADHD.

Future work guided by the methodological considerations described herein holds considerable promise for improving our understanding of the neurocognitive causes, outcomes, and sequelae of these neurodevelopmental disorders. Specifically, future studies would benefit from using cognitively-informed, construct-valid executive function tests and symptom measures; adopting dimensional approaches to capture the full range of symptom frequency, quantity and severity for each symptom cluster and functional outcome of interest; improving sampling strategies and access to clinical research services for populations that have been traditionally excluded from these types of studies; and considering third-variable explanations for any detected overlap in executive function profiles. Work guided by these methodological considerations holds considerable promise for improving the field's understanding of the neurocognitive causes, outcomes, and sequelae of ADHD and ASD. Clinicians might also be interested in maximizing the limited utility of current, commercially available executive function tests (Box 4).

At the same time, the nature and structure of executive function remains actively debated in the cognitive literature.[184] Despite emerging experimental and longitudinal studies providing functional (and probably causal) evidence linking cognitive/behavioral models of executive function with important behavioral and functional outcomes in ADHD and ASD,[33,37,92,158–161] alternate executive function conceptualizations and measurement approaches (such as neurobiological and sociocultural insights) are clearly warranted. We have taken the position that the improved sensitivity and specificity of cognitively-informed performance tests for differentiating ADHD and ASD from control groups will help future work to more definitively disentangle shared vs. unique neurocognitive deficits in these disorders. However, we acknowledge that bigger effect sizes are not inherently better. Ultimately, the utility of adopting cognitive or behavioral (or any other) models of executive function lies in their usefulness and the extent to which they help clinicians understand – and communicate to parents, teachers, and other stakeholders – why these children exhibit challenging behaviors.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Box 1:

Informant-based executive function scales

The evidence strongly indicates that informant-rated executive function scales are not valid for assessing cognitive abilities such as executive functioning.[131,135,138] These rating scales are reliably measuring something, but more work is needed to determine exactly what that something is.

Some authors have proposed that everyday executive functions could be divided into 'executive function abilities' (measured by performance-based tests) and 'executive function skills' (measured by informant-rated scales), or into cognitive (performance-based) vs. behavioral (informant-rated) manifestations of executive function (for a review see[131]).

However, these distinctions are problematic because using the same term ('executive functions') for minimally related constructs evokes the 'jingle fallacy' – the logical error in which two tests are assumed to assess the same construct because they share similar names or labels.[185]

Adopting alternative terms for behavioral ratings to refer to the specific behaviors and/or skills of interest will help to resolve the conceptual and measurement conflict. For example, the term 'executive function skills' can be replaced with more specific descriptions of the behaviors and/or skills of interest, such as 'organization, time management, and planning skills'[159,186]

Using alternative terms can also promote additional research by highlighting abilities not directly included in the construct of executive function that are important for understanding executive function behaviors. To that end, currently available informant-rated 'executive function' scales have been theorized to measure externalizing behaviors broadly,[138] success of goal pursuit,[135] ADHD symptoms, or organizational/planning skills.[131]

Box 2:

The case of the stop-signal task.

In addition to concerns regarding the lack of specificity of executive function tests commonly used in the clinical literature,[53,109,117,119,129] the specific metrics used in these tests warrant scrutiny. A compelling example is the stop-signal test, which is often considered a gold standard for measuring inhibitory control in the cognitive and clinical literature.[187]

The stop-signal test is a choice-response task based on the racehorse model of inhibition, which conceptualizes successful inhibition as the outcome of a race between independent 'go' and 'stop' processes. In this model, children successfully inhibit a response if the 'stop' process finishes first, whereas they fail to inhibit if the 'go' process finishes first. In the stop-signal task, children are instructed to choose between two 'go' response options after seeing a specific stimulus (for example, press X in a keyboard when they see an X on the screen or press O when they see an O).[74,75] On a subset of trials, a stop signal is presented (often an auditory beep) a short time after the 'go' response option. The stop signal cues children that they should withhold or stop (inhibit) their response on that trial.

The traditional index of inhibitory control obtained from this test is the stop-signal reaction time (SSRT), which reflects the difference between the child's mean response time on 'go' trials (how fast or slow they respond when they are expected to respond), and the stop-signal delay (the maximum time between the stimulus and the stop signal that can still produce a successfully completed inhibition response).

The SSRT metric from the stop-signal task has been used to build a substantial proportion of the evidence base suggesting inhibitory control deficits in ADHD and ASD.[75] Unfortunately, this metric produces "fictitious inhibitory differences",[187] particularly for conditions such as ADHD that are linked with skewed reaction time distributions on speeded response tasks.[132] In other words, it produces false positives indicating that children with ADHD have impaired inhibition when in reality they might not.

An alternative scoring approach based on an integrated method of stop-signal reaction time (iSSRT) has been recommended to assess inhibitory control in children with ADHD and ASD[187]. However, to date very few ADHD and/or ASD studies have adopted this metric.[82]

Box 3:

Cognitive disengagement syndrome

ADHD and ASD overlap not only with each other, but each disorder also demonstrates increased risk for a host of other clinical conditions[41] including multiple forms of anxiety, depression, learning disabilities, and cognitive disengagement syndrome. In turn, these conditions have also been linked with neuropsychological/executive functioning sequalae..[188,189] However, the extent to which these co-occurring conditions and symptoms contribute to the seemingly similar executive function profiles across ADHD and ASD is still largely unknown.

Cognitive disengagement syndrome is one understudied condition that warrants increased scrutiny as a potential third-variable explanation for the overlap between ADHD and ASD executive function profiles. Cognitive disengagement syndrome refers to a constellation of symptoms that include slowed behavior and/or thinking, reduced alertness, excessive daydreaming, and getting lost in one's thoughts.[190–192] This syndrome was formerly called 'sluggish cognitive tempo', and the shift in terminology reflects increased awareness that the former might be not only pejorative and offensive but also inaccurate.[191,193]

Cognitive disengagement symptoms are distinguishable from ADHD and ASD symptoms, but occur at significantly elevated rates in both ADHD (31–40%) and ASD (49%),[194] and might account for the association between ADHD and ASD symptomology.[188] Cognitive disengagement symptoms also predict a host of outcomes implicated in both ASD and ADHD (in many cases even after controlling for ADHD and/or ASD symptoms) including social difficulties, informant-reported cognitive problems, academic challenges, internalizing impairments, and reduced global functioning above and beyond ADHD and ASD.[188,189,194,195]

Although the original moniker sluggish (slow) cognitive (mental) tempo (speed) presumes that the symptoms are attributable to slow processing speed, the evidence of associations between cognitive disengagement symptoms and slowed processing speed is mixed[196]. Our current read of this literature is that children with elevated cognitive disengagement symptoms show accurate but moderately slowed performance across a wide range of neuropsychological tests.[197] However, this pattern is not attributable to a globally 'sluggish' cognitive tempo, but to executive dysfunction characterized by working memory systems that are too slow and inhibition systems that are too fast.[193] Behaviorally, requiring extra time to rearrange the active contents of working memory delays responding, whereas an overactive inhibition system likely terminates thoughts too quickly and therefore prevents the initiation or completion of intended behaviors. These independent executive functioning difficulties combine to give the appearance that children with cognitive disengagement syndrome are absent-minded or fail to act when expected.

Taken together, cognitive disengagement symptoms occur at elevated levels in both ADHD and ASD, are linked with executive function deficits, and might account for the presence of – and/or overlap between – important behavioral and functional impairments

in ASD and ADHD. To our knowledge, however, no study of executive functioning in ADHD or ASD has accounted for this unique neurocognitive-behavioral-affective syndrome. Thus, it will be critical for future studies to assess and control for cognitive disengagement symptoms as the field moves toward clarifying the unity and diversity of executive function profiles in ADHD and ASD.

Box 4:

Evidence-based clinical recommendations

It is increasingly clear that the gap between basic and applied research is at least as long[198] as the well-documented 17-year research-to-practice gap[199,200], with many seminal executive function studies approaching the 25th anniversary of their publication[18,101]. We are not aware of any commercially available, norm-referenced tests to assess executive function in clinical settings that would meet cognitive benchmarks for valid executive function assessment. Until construct-valid and reliable clinical measures of executive function are available, clinicians are limited to 'executive function' tests that in most cases were not originally developed to assess executive functioning and were re-purposed from classic frontal lobe tests.

Clinicians have an ethical responsibility to continually evaluate the evidence supporting and/or refuting the validity of tests, and to use this evaluation to guide test battery selection and test score interpretation. This obligation is explicitly defined in the *Standards for Educational and Psychological Testing*, jointly published by the American Psychological Association, American Educational Research Association, and National Council on Measurement in Education.[201]

In that context, clinicians should avoid interpreting subtest level scores when using traditional neuropsychological test batteries of executive functions, and instead focus on composite or full scale scores based on multiple subtests. The majority of the variance in any single neuropsychological subtest score is attributable to processes other than the 'executive function' that clinicians are trying to measure. There is overwhelming evidence indicating that individual subtests from these batteries contribute meaningfully to psychometric *g* (global neuropsychological functioning or IQ) but do not adequately or accurately capture strengths and weaknesses in specific executive function components.[96–98,100–103,105,106]. By contrast, subtest-level scores from traditional neuropsychological executive function test batteries do not adequately or accurately capture strengths and weaknesses in specific executive functions. Instead, individual differences on these subtests seem to primarily reflect non-executive factors that influence test performance, including gross and fine motor demands, visual and verbal perception, and attention lapses.[109]

Thus, we recommend using traditional neuropsychological executive function test batteries as screening tools for severe impairment and limiting clinical interpretation to global neuropsychological functioning rather than inferring domain-specific patterns. In terms of specific test recommendations, the current best options for assessing working memory might be the recall of sequential order subtest of the Differential Abilities Scale-II and the NIH Toolbox List Sorting test. This recommendation comes with the caveat that these tests have not been compared head-to-head with construct-valid working memory tests from the cognitive literature. Also, both tests possess several features to ease administration at the cost of construct validity (such as all-or-nothing scoring and discontinue rules). We also considered the letter-number sequencing subtest of the Wechsler Intelligence Scale for Children-V, which is a face valid working

memory reordering test. However, the factor analytic studies described in the main text indicate strongly that the WISC-V working memory subtests contribute to measuring IQ but do not have enough reliable variance left to produce a unique working memory factor.[118,122–125,128]

Similarly, commonly used neuropsychological tests of inhibition show construct validity limitations. This issue might be due to tasks presenting test trials with 100% incongruent stimuli, which significantly reduces inhibition demands by minimizing the interference effects of the non-dominant rule set and reducing goal maintenance requirements by inadvertently reinforcing task goals[52,162].

Set shifting is likely not a unique executive function in school-aged children[21], and so we do not offer recommendations regarding its measurement.

For research purposes, several excellent options are either freely available online, available by request from study authors, or can be programmed using free or relatively low-cost experiment presentation software. Examples include the Online Working Memory Lab,[202] working memory tests designed to assess patients with ADHD and ASD[37,51,58] and child versions of classic updating, inhibition, and shifting tests.[18,22]

A helpful list of recommended executive function tests for research purposes has also been published.[109] These tests and measures were developed psychometrically, but have not been adopted in widespread clinical and clinical-research practice.[112] Finally, a 5-item working memory teacher rating scale has shown initial promise for predicting short-term memory (r=.25-.40) and academic achievement (r=.42-.60) test performance up to 18 months later. However, as with other executive function rating scales, the items in this scale are highly similar to ADHD diagnostic criteria, which inflates the apparent scale validity.[203]

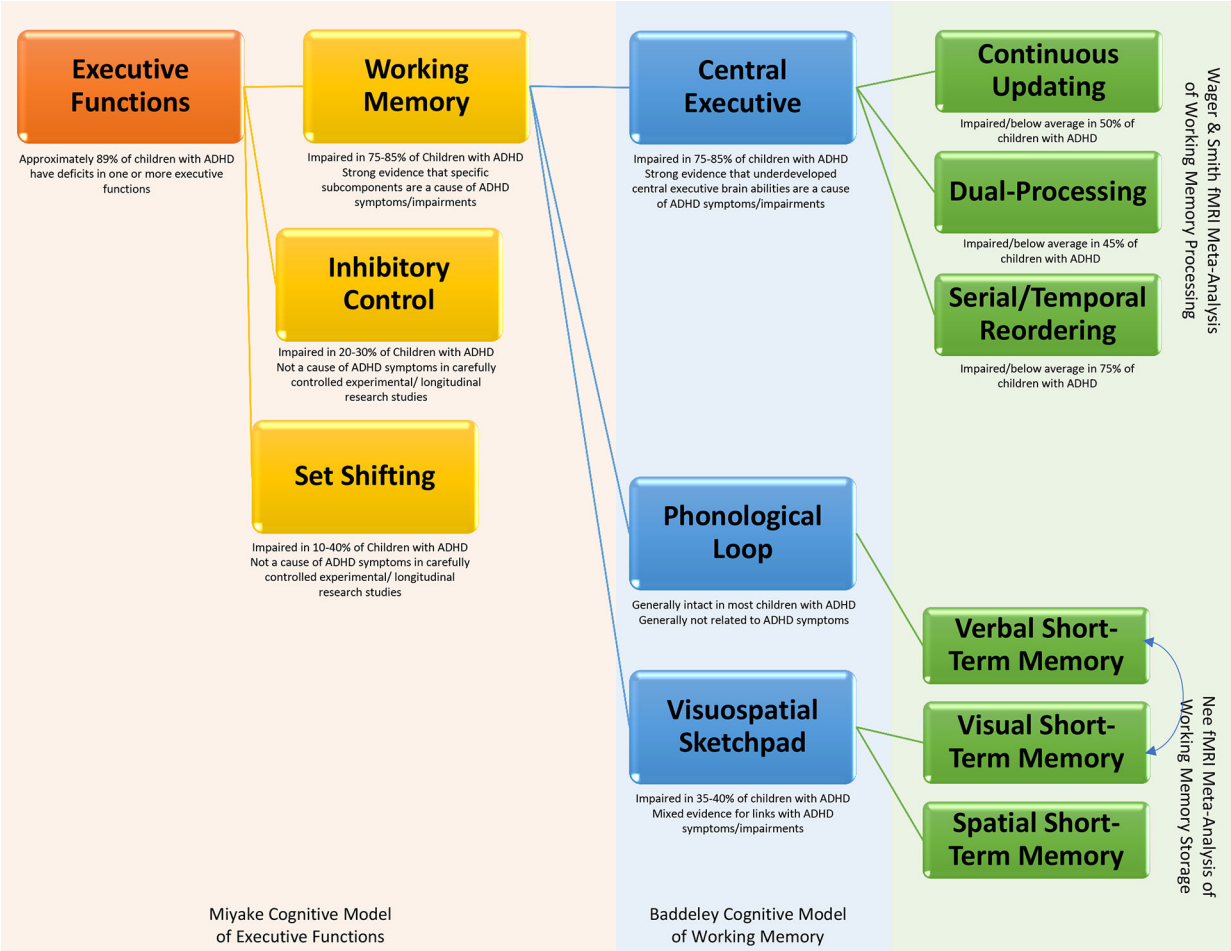

Figure 1. Conceptual model of executive function components and working memory subprocesses.

The unity and diversity model identifies a broad executive function construct (red) with three primary components: working memory, inhibitory control, and set shifting (yellow). The working memory model fractionates working memory into a 'working' component called the central executive, and two storage-rehearsal 'memory' components that temporarily hold verbal vs. visual-spatial information[20] (blue). However, meta-analytic fMRI evidence suggests a differentiation between spatial and non-spatial (verbal, visual/object) content[152]. Meta-analytic fMRI evidence also indicates that the central executive component can be further subdivided into more specific mental processes including updating, dual-processing, and serial-temporal reordering[57](purple). There are also subprocesses of inhibitory control and set shifting that are not depicted here.

Table 1.

Systematic reviews and meta-analyses of executive functioning in ADHD

Executive function domain	Findings and effect sizes	Number of studies or meta-analyses	Sample size	Age range of sample (years)	Ref.
Working memory	Working memory mean effect weighted by *k* = .54 (*k*=156)	34 meta-analyses	*n* by group not available. Total *n* range=1,010 – 1,721.	8.5 – 34.1 (mean across studies)	54
	Working memory effect size ESzr = .17	22 studies of youth with externalizing behavior problems	Externalizing *n* range=1,161–1,653 Control *n*=523	3 – 6	60
	Visuospatial hedge's *g*=.74 Phonological hedge's *g*=.69	45 studies	ADHD n = 866 TD n = 2,128	8 – 16	55
	Working memory effect size *d*=.32	25 studies	*n* by group not available. Total *n*=3,005	3–6	59
Inhibitory control	Inhibitory control mean effect weighted by *k* = .52 (*k*=438)	34 meta-analyses	*n* by group not available. Total *n* ranges = 136 – 6,403.	8.5 – 34.1 (mean across studies)	54
	Inhibitory control effect size ESzr = .24	22 studies of youth with externalizing behavior problems	Externalizing *n* range =3,093–3,604 Control *n*=523	3 – 6	60
	Average weighted effect size = .24.	18 studies	ADHD *n*=757 TD *n*=605	7 – 50	78
	ADHD vs. control response time: *g*=0.62 Effect sizes youth > adults	71 studies	TD *n*=3,656 Clinical sample (not limited to ADHD) *n*=5,593	6 – 58	77
	ADHD vs. control commission errors *g*=.40 ADHD vs. control omission errors *g*=.59 Mean reaction time of combined measure (Go/No Go, continuous performance task, and sustained attention and response) *g*=.29	318 studies	Clinical sample (not limited to ADHD) n= 11,211 Control sample n= 11,577	No age restriction; further information not available	59
	Mean reaction time *g*=.45 Stop signal reaction time *g*=.63 Standard deviation of the reaction time *g*=.73.	24 studies	ADHD n = 808 TD n = 695	7 – 12	74
	Reaction time congruency effects: 3 studies found ADHD > TD, 7 studies not significant, one study ADHD = TD Accuracy congruency effects: 2 studies found ADHD > TD, 8 studies not significant, 1 study did not report data.	12 studies	ADHD n = 272 TD n = 280	6 – 17	79
	Inhibitory control effect size *d*=.55, response inhibition effect size *d*=.64	25 studies	*n* by group not available; total *n*=3,005	3–6	59
Set shifting	Set shifting mean effect weighted by *k* = .35 (*k*=260).	34 meta-analyses	*n* by group not available. Total *n* range = 584 – 691.	8.5 – 34.1 (mean across studies)	54
	Set shifting effect size ESzr = .13	22 studies of youth with externalizing behavior problems	Externalizing *n*=605–1,038 Control *n*=188	3 – 6	60
	Set shifting effect size *d*=.63	25 studies	*n* by group not available Total *n*=3,005	3–6	59
Non-specific executive functioning	Overall magnitude of the effect size comparing ADHD and TD youth SMD=.45	34 meta-analyses	*n* by group not available. The *n* used to calculate summative SMDs ranged from 136 to 21,804.	8.5 – 34.1 (mean across studies)	54

Executive function domain	Findings and effect sizes	Number of studies or meta-analyses	Sample size	Age range of sample (years)	Ref.
	Overall effect size in externalizing compared to control children was ESzr=.22	22 studies of youth with externalizing behavior problems	Externalizing *n* range=3,238–3,749 Control *n*=739	3 – 6	[60]

Note: ADHD = attention-deficit/hyperactivity disorder, ASD = autism spectrum disorder, EF = executive function, TD = typically developing, ESzr = mean correlation effect size, SMD = standardized mean difference, *k*=number of studies.

Table 2.

Systematic reviews and meta-analyses of executive functioning in ASD

Executive function domain	Effect size and other findings	Number of studies or meta-analyses	Sample size	Age of sample (years)	Ref.
Working memory	Working memory among school-aged children *g*=.62 Working memory among adolescents *g*=.20	235 studies	ASD n = 6816 Control individuals n = 7265	≥6	61
	Phonological working memory *d*=.67	11 studies	ASD n = 271 TD n = 256	11 – 38	62
	Visuospatial working memory *d*=.73	23 studies	ASD n = 647 TD n = 700	8 – 63	
	Working memory *d* = .61 Greater deficits in spatial compared to verbal working memory.	28 studies	ASD n = 819 Control individuals n = 875	ASD mean = 6.5–63.6 Control mean = 6.3–63.7	63
Inhibitory control	Overall effect size *k*=103, *g*=.46	235 studies	ASD n = 6816 Control individuals n = 7265	≥6	61
	Inhibition small-to-medium effect in ASD vs. non-ASD Interference control: ES = .31 Response inhibition: ES = .55	2 meta-analyses (including 41 studies)	ASD n = 1091 TD n = 1306	ASD mean = 14.8 TD mean = 13.8	80
	Similar deficits across inhibitory control and response inhibition Effects when tasks were used *g*=0.48 (preschoolers > school-aged > adolescents). Age effect was not significant when ADHD comorbidity was included. Effects when parent-report measures were used *g*=1.33	Meta-Analysis of direct measures: 164 studies	ASD n = 5140 Control individuals n = 6075	ASD mean = 14.26	81
		Meta-Analysis of indirect measures: 24 studies	ASD n = 985 Control individuals n = 1300	ASD mean = 9.75	
Set shifting	Overall effect size *k*=38, *g*=.48	235 studies	ASD n = 6816 Control individuals n = 7265	≥6	61
Non-specific executive functioning	Effect size of executive function measures excluding rating scales *k*=221, *g*=0.48 (95% CI 0.43–0.53, *p*<.001). Impairment was similar across domains. Overall effect size *k*=70, *g*=.47	235 studies	ASD n = 6816 Control individuals n = 7265	≥6	61

Note. ADHD = attention-deficit/hyperactivity disorder, ASD = autism spectrum disorder, EF = executive function, TD = typically developing, ES = effect size.

Table 3.

Systematic reviews and meta-analyses of executive functioning in co-occurring ASD and ADHD samples, or in ADHD or ASD samples while controlling for the other syndrome

Executive function domain	Findings and effect sizes[a]	Number of studies or meta-analyses	Sample size	Age of sample	Ref.
Working memory	ADHD+ASD = ASD = ADHD	26 studies	Total ADHD, ASD, ADHD+ASD, and ASD+ID n=4,458	Range = 4 – 22 Mean = 10	72
	TD generally > ADHD+ASD = ASD = ADHD	26 studies	ASD n=646 ADHD n=789 ADHD+ASD n=101 TD n=723	Range = 3 – 18	69
	TD > ASD (*g*=0.50–0.53)	98 studies	ASD n=2,986 TD=3,005	Mean ASD = 10.65 Mean TD = 10.81	65
	ADHD > ASD (*g*=0.43) TD > ASD TD > ADHD	58 studies	ASD n=2,092 ADHD n=2,800 TD n=3,367	Range = 3 – 18	42
Inhibitory control	ADHD+ASD = ASD = ADHD	26 studies	Total ADHD, ASD, ADHD+ASD, and ASD+ID n=4,458	Range = 4 – 22 Mean = 10	72
	TD = ASD > ADHD = ADHD+ASD	26 studies	ASD n=646 ADHD n=789 ADHD+ASD n=101 TD n=723	Range = 3 – 18	69
	TD > ASD (*g*=0.32)	98 studies	ASD n=2,986 TD n=3,005	Mean ASD = 10.65 Mean TD = 10.81	65
	ADHD > ASD (*g*= -1.23–0.46) TD > ASD TD > ADHD	58 studies	ASD n=2,092 ADHD n=2,800 TD n=3,367	Range = 3 – 18	42
Set-shifting	ADHD+ASD > ASD	26 studies	Total ADHD, ASD, ADHD+ASD, and ASD+ID n=4,458	Range = 4 – 22 Mean = 10	72
	ADHD > ASD	26 studies	ASD n=646 ADHD n=789 ADHD+ASD n=101 TD n=723	Range = 3 – 18	69
	TD > ASD (g=0.61)	98 studies	ASD n=2,986 TD=3,005	Mean ASD = 10.65 Mean TD = 10.81	65
	ASD = ADHD (*g*=−0.91–0.23) TD > ASD TD > ADHD	58 studies	ASD n=2,092 ADHD n=2,800 TD n=3,367	Range = 3 – 18	42

Note. ADHD = attention-deficit/hyperactivity disorder; ASD = autism spectrum disorder; TD = typically developing;

[a]Clinical groups are ranked from best to worst performance (lower ranking = more impaired)

Table 4.

Desirable characteristics of working memory tests and methods

Desirable test characteristic and study methods	Brief rationale	Examples of tests that do not meet the criterion	Refs.
Patient responses require recall, not just recognition	Meta-analytic correlation between recall-based working memory tests and recognition-based working memory tests is only *r*=.20, suggesting that recognition and recall tasks tap largely independent constructs.	*N*-back tasks	52,111,171–173
Correct responses require 'working' components of working memory, not just passive storage or simple reversal	A simple reversal of list order can be performed without removing attention from the mental representation of the list items. Simple reversal is more strongly related to fluid reasoning than working memory in children with ADHD and children in general.	Digit span backward, spatial span backward, Corsi block tapping, CANTAB spatial WM	56,57,86,97,99–102,105,107,109,174,175
Partial-credit unit scoring (count each stimulus correct, not just each trial)	Partial-credit scoring produces more reliable estimates (higher internal consistency), increased sensitivity for detecting individual differences, and stronger concurrent and predictive validity estimates than all-or-nothing scoring.	To our knowledge, all commercially available tests	107,108,174,176
All trials are administered (do not discontinue test based on patient's performance)	'Discontinue rules' are convenient for clinical administration, but greatly limit test sensitivity by blunting individual differences, reducing variability in scores across patients, and resulting in the majority of variance in scores coming from the lowest memory loads.	Most tests commonly used in clinical practice	107,108,174,176,177
Memory sets are unpredictable (test takers are not able to anticipate the number of stimuli they will have to remember on a given trial)	Memory set predictability lowers the task's working memory demands because it allows patients to use strategies to decrease the task's executive demands and develop task expertise over time (for example, when tests start at lower memory loads and increase sequentially, or present the same number of items every trial).	To our knowledge, all commercially available tests	108,118,167,178,179
The range of memory loads captures the full range of abilities in the population of interest	Memory load refers to the number of discrete 'bits' (pieces of information) that can be temporally held in the forefront of one's mind for immediate access and processing. Memory load is affected by a number of factors including number of stimuli presented, stimulus modality, information complexity, and cultural factors.	N/A	108,180,181
Test includes a sufficient number of trials at each memory load and in total	The ideal number of trials (suggested 6+ trials per memory load) reflects a tradeoff between reliability and efficiency, and likely differs across different working memory tests, emphasizing the need for psychometric work.	To our knowledge, all commercially available tests	118,167,174
Multiple tests from different modalities (for example, verbal/visual vs. spatial) are used and latent estimates are derived	The majority of variance in any single test is attributable to processes other than working memory Formative and/or summative approaches (such as SEM latent factors and Bartlett factor scores) increase specificity and maximize the extent to which conclusions can be drawn about working memory vs. other processes.	N/A	86,108,127,129
Analyses do not covary IQ	Covarying IQ is problematic given that working memory is a likely a causal factor affecting performance on IQ tests, rather than vice versa.	N/A	109,153–155,182,183
Participants are monitored during testing. In online administration, stimuli are selected to improve validity	Some patients might use alternative methods to improve performance during working memory tests (for example, writing down the stimuli). Using stimuli that cannot be easily put into words (for example, "Klingon" symbols) and/or spatial tasks improves validity for remote and unmonitored administration.	N/A	147
Research studies are preregistered	Preregistration refers to publicly documenting the research plan prior to running the study.	N/A	osf.io

Note. See Supplementary Materials for expanded discussion of each criterion. IQ = intelligence quotient; N/A = not applicable; SEM = structural equation modeling; CANTAB = Cambridge Neuropsychological Test Automated Battery; WM = working memory

Table 5.

Unique and overlapping executive function deficits in ADHD and ASD

Construct	Definition	Components	Conclusions regarding ASD vs. ADHD
Non-specific executive functioning	The 'unity' in the unity and diversity model. Refers to 'common EF,' or the cognitive processes that are shared across the three primary executive functions.	The specific processes remain poorly understood but presumably involve active maintenance of task goals and goal-related information, and using that information to effectively bias lower-level processing.	No studies met diagnostic and methods criteria to allow firm conclusions.
Working memory	The active, top-down manipulation of information held in short term memory, including the mental ability to hold, manipulate, and update multiple pieces of information.	The 'working' components include reordering (maintaining and rearranging information in mind), updating (active monitoring of incoming information and replacing outdated with relevant information), and dual-processing (maintaining information in mind while performing a secondary task). The 'memory' components include verbal-visual short-term memory and spatial short-term memory.	No studies met diagnostic and methods criteria to allow firm conclusions. Evidence for large working memory deficits in ADHD when controlling for ASD (via inclusion of children with ASD in both ADHD and non-ADHD groups or controlling for ASD diagnostic status). Possible preliminary evidence for greater impairment in ADHD vs. ASD based on indirect metrics. No direct evidence for or against shared or unique impairments in specific subcomponents.
Inhibitory control	The ability to withhold or stop an on-going response, particularly within the context of goal-directed behavior.	Subcomponents vary across models and include differentiating action cancellation (stopping an in-progress behavior) and action restraint (preventing a behavior before it starts), as well as cognitive vs. behavioral inhibition. Interference control (the suppression of interference due to resource or stimulus competition) is also considered a subcomponent of inhibition in some models, whereas others ascribe this function to working memory.	Initial evidence suggesting potentially greater impairment in ADHD vs. ASD for the action restraint (go/no-go) component of inhibition. No direct evidence for or against shared or unique impairments in other inhibitory control subcomponents.
Set shifting	The ability to switch flexibly between mental sets. Also called cognitive flexibility. Likely not a unique executive function in school-aged children.	Rule switching (implementing the correct response based on changing cues) and perceptual switching (moving visuospatial attention away from one set of features to selectively attend to a different set of features).	No studies met diagnostic and methods criteria to allow firm conclusions. Impaired performance on set shifting tests in ADHD seem to be due to non-shifting aspects of the tests.