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Does Anxiety Systematically Bias Estimates of Executive Functioning Deficits in Pediatric Attention-Deficit/Hyperactivity Disorder?

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Abstract

Growing evidence suggests that childhood ADHD is associated with larger impairments in working memory relative to inhibition. However, most studies have not considered the role of co-occurring anxiety on these estimates – a potentially significant confound given prior evidence that anxiety may increase working memory difficulties but decrease inhibition difficulties for these children. The current study extends prior work to examine the extent to which co-occurring anxiety may be systematically affecting recent estimates of the magnitude of working memory/inhibitory control deficits in ADHD. The carefully-phenotyped sample included 197 children with ADHD and 142 children without ADHD between the ages of 8 and 13 years (N=339; M_{age}=10.31, SD=1.39; 144 female participants). Results demonstrated that ADHD diagnosis predicted small impairments in inhibitory control (d=0.31) and large impairments in working memory (d=0.99). However, child trait anxiety assessed dimensionally across multiple informants (child, parent, teacher) did not uniquely predict either executive function, nor did it moderate estimates of ADHD-related working memory/inhibition deficits. When evaluating anxiety categorically and controlling for ADHD, anxiety diagnosis predicted slightly *better* working memory (d=0.19) but not inhibitory control for clinically evaluated children generally. Findings from the current study indicate that trait anxiety, measured dimensionally or categorically, does not differentially affect estimates of executive dysfunction in pediatric ADHD. Further, results suggest that trait anxiety is generally not associated with executive dysfunction above and beyond the impact of co-occurring ADHD. Future research is needed to further assess the role of anxiety in ADHD behavioral symptomatology, neurocognitive functioning, and mechanisms underlying these relations.

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Keywords

ADHD; anxiety; executive function; working memory; inhibitory control

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder that impacts approximately 5% of children and is associated with chronic functional difficulties across the lifespan (Polanczyk et al., 2014). In addition to difficulties that arise from ADHD, co-occurring conditions are common and can affect the valence and magnitude of functional outcomes for these children (Larson et al., 2011; Chan et al., 2022a). For example, approximately 25% of children with ADHD meet diagnostic criteria for an anxiety disorder (Jarrett & Ollendick, 2008; Tannock, 2009). While the exact nature of the co-occurrence between ADHD and anxiety remains unclear (for reviews, see Jarrett & Ollendick, 2008; Schatz & Rostain, 2006), emerging evidence suggests that co-occurring anxiety may exacerbate or present new difficulties for children with ADHD in some functional domains (Bishop et al., 2019; Overgaard et al., 2016) while potentially buffering against or masking difficulties in other domains (Chan et al., 2022b; Maric et al., 2018). Executive functions are one area in which this interplay may be of particular importance, such that co-occurring anxiety symptoms may help to explain the larger working memory relative to inhibitory control deficits seen in this population (Jarrett & Ollendick, 2008; Karalunas et al., 2017; Kofler et al., 2019; Tannock, 2009). To that end, the present study examined the extent to which child trait anxiety, measured dimensionally and categorically, produces systematic over- or under-estimates of ADHD-related executive function deficits.

Executive Functions and Anxiety

Executive functions are interrelated, higher-order processes that regulate and enable cognition, goal-directed behaviors, and problem solving (Miyake et al., 2000). Various models of executive function based on empirical and theoretical work have included three primary executive function domains including working memory, inhibitory control, and set shifting (e.g., Huizinga et al., 2006; Miyake et al., 2000; St Clair-Thompson & Gathercole, 2006). However, inhibitory control and working memory are the focus of the current study due to developmental research suggesting that set shifting likely does not become a distinct ability until adolescence or early adulthood, while earlier performance on set shifting tasks depends heavily on working memory and/or inhibition processes (Irwin et al., 2019, 2022; Karr et al., 2018). Briefly, *inhibitory control* refers to processes that support the ability to withhold or stop a dominant and/or on-going response (Alderson et al., 2007), while *working memory* involves the active, top-down manipulation of information held in short-term memory through interrelated functions of updating, dual-processing, and temporal/serial reordering (Fosco et al., 2020).

Interestingly, whereas several theoretical models conceptualize executive function deficit(s) as underlying *causes* of ADHD (Barkley, 1997; Kofler et al., 2020; Rapport et al., 2001; cf. Halperin & Schulz, 2006; van Lieshout et al., 2013), they tend to be viewed as *outcomes* of anxiety disorders or involved in development and maintenance of anxiety symptoms (Eysenck et al., 2007; Ferreri et al., 2011; Hirsch & Mathews, 2012). Greater trait anxiety

in childhood and adolescence has been associated with both impaired working memory and inhibitory control in some non-ADHD studies, however, results have been mixed (e.g., Alfonso & Lonigan, 2021; Moran, 2016; Ursache & Raver, 2014; Wright et al., 2014), which may reflect methodological differences including measurement of both working memory/inhibitory control (e.g., different tasks, questionnaire-based reports) and anxiety (e.g., dimensional versus categorical, state versus trait), lack of control for co-occurring ADHD symptoms/diagnoses, as well as collapsing across adult and child samples versus examining these relations in children specifically. In contrast, a different pattern appears to emerge when examining the impact of anxiety on executive functions in children with ADHD as described below.

ADHD, Anxiety, and Executive Functions

Inhibitory Control

Although trait anxiety may be associated with *increased* inhibitory control difficulties in non-ADHD children and adolescents as noted above, emerging evidence suggests that anxiety may *decrease*, or buffer against, inhibition difficulties in individuals with ADHD (Jarrett et al., 2016; Schatz & Rostain, 2006; Tannock, 2009), thus obfuscating attempts to estimate the magnitude of inhibition deficits in children with ADHD. Conceptual models propose that this buffering or masking effect occurs via an anxiety-related increase in motivation, effort, and impulse control (Jarrett et al., 2016; Tannock, 2009), greater recruitment of cognitive resources and increased effort to compensate for ADHD-related impairments in attentional control (Eysenck et al., 2007), and/or increased physiological/cortical arousal that in turn temporarily actuates underdeveloped prefrontal structures that support executive functioning (Arnsten, 2009; Ruf et al., 2017; Shaw et al., 2007). Stated differently, these models suggest that the hypothesized over-inhibited behavioral style characteristic of anxiety may interact with the under-inhibited behavioral style characteristic of ADHD, resulting in inhibition performance that falls in between either condition and thus more similar to their neurotypical peers.

Indeed, meta-analytic evidence suggests that individuals with ADHD and a co-occurring anxiety disorder demonstrate better inhibition than those with ADHD alone (Lipszyc & Schachar, 2010; Maric et al., 2018), although their performance continues to fall below that of healthy controls (Lipszyc & Schachar, 2010). In the time since those reviews, additional evidence has emerged suggesting that children with ADHD and co-occurring anxiety demonstrated better inhibitory control compared to children with ADHD alone (Menghini et al., 2018; Yurtbaşı et al., 2018). At the same time, most prior research in this area has utilized categorical definitions of anxiety despite increased emphasis on examining the role of anxiety symptoms dimensionally given that anxiety disorders reflect extremes along natural continuums of characteristics that are normally distributed across the general population (Castagna et al., 2019; Read et al., 2020). Among studies that have examined anxiety symptoms dimensionally, it remains unclear whether clinically significant levels of anxiety are necessary to see effects of anxiety on inhibition performance in ADHD due to mixed results that may be an artifact of using single tasks to estimate inhibition (Ruf et al., 2017; Bloemsma et al., 2013; Adamo et al., 2021; Read et al., 2020) given that the majority

of variance in any given neurocognitive task is likely due to processes other than the specific ability we intend to measure (e.g., the *task impurity* problem; Snyder et al., 2015).

Working Memory

In contrast to co-occurring trait anxiety potentially producing significant *under*estimates of inhibition deficits in children with ADHD, it may be producing significant *over*estimates of ADHD-related working memory deficits (Jarrett et al., 2016; Schatz & Rostain, 2006; Tannock, 2009). Similar to findings in studies of non-ADHD children and adolescents (for meta-analysis, see Moran, 2016), influential theoretical models suggest that anxiety may impede retention and processing of information within working memory (Jarrett et al., 2016; Tannock, 2009). Conceptually, this exacerbating effect is thought to occur because anxiety reduces encoding/filtering efficiency by prioritizing bottom-up, threat-related cues (e.g., worry thoughts or external stimuli; Moran, 2016). These threat-related cues are thought to reduce bandwidth for storing/processing task-relevant information and create additional dual-processing demands by allocating resource-limited internal focus of attention and short-term storage space to anxiety-provoking stimuli/thoughts (Eysenck et al., 2007; Fosco et al., 2020). Relatedly, it has been suggested that greater levels of trait anxiety further impair performance by eliciting concerns about competency and performance (state anxiety) during working memory tests, which may be even more likely to occur for children with ADHD given their objective difficulties on these types of tests (Read et al., 2020; Schatz & Rostain, 2006; Karalunas et al., 2017; Kofler et al., 2019).

Indeed, Read et al. (2020) found that higher levels of anxiety symptoms were related to worse working memory above and beyond ADHD symptom severity in children diagnosed with ADHD and comorbid anxiety. Similarly, Castagna et al. (2019) found that greater ADHD symptoms were related to worse working memory in children with high, but not low, anxiety in a sample of non-clinical youth, suggesting that anxiety symptoms may impact working memory test performance across the continuum of anxiety symptoms rather than only at clinically elevated levels. In contrast, a meta-analytic review found no working memory differences between children with ADHD and those with co-occurring ADHD+anxiety (Maric et al., 2018), although conclusions may be limited because most included studies used working memory tests that have been criticized for measuring short-term memory and/or gross neuropsychological functioning rather than working memory specifically (for reviews, see Rapport et al., 2013; Snyder et al., 2015). Taken together, the available evidence, while mixed, positions anxiety as a critical confound that may be systematically producing overestimates of working memory deficits in ADHD – a critical consideration given that most ADHD studies have not controlled for anxiety when reporting estimates of working memory (or inhibition) deficits in this population.

Current Study

Taken together, prior literature suggests that trait anxiety may be both obfuscating (inhibition) and inflating (working memory) the field's estimates of the magnitude of executive function impairments in pediatric ADHD (Bloemsma et al., 2013; Maric et al., 2018; Ursache & Raver, 2014). The current study seeks to clarify the extent to which anxiety

may reflect a key confound in these efforts by examining anxiety's impact on the magnitude of working memory and inhibition deficits in children with ADHD relative to children without ADHD in a carefully phenotyped, clinical child sample, including those with and without co-occurring anxiety disorders, using well-validated assessments of working memory and inhibitory control. Based on the evidence reviewed above, we hypothesized that greater anxiety would significantly reduce estimates of ADHD-related inhibitory control deficits. In contrast, based on prior mixed evidence, we predicted that greater anxiety would inflate estimates of working memory deficits in children with ADHD, and be associated with lower working memory abilities in both children with and without ADHD.

Method

Participants

The sample included 339 children between the ages of 8 and 13 years (M = 10.31, SD = 1.39; 144 female participants) from the Southeastern U.S. recruited through community resources for participation in a clinical research study of the neurocognitive mechanisms underlying pediatric attention and behavior problems. The Florida State University IRB approved the study prior to and throughout data collection, and parents and children gave written informed consent/assent. Sample ethnicity consisted of 228 White Not Hispanic or Latino (67.3%), 47 Black or African American (13.9%), 36 multiracial (10.8%), 23 Hispanic or Latino (6.8%), and 5 Asian (1.5%) children. Children were excluded from the larger study if they presented with gross neurological, sensory, or motor impairment; non-stimulant medications that could not be withheld for testing; or history of seizure disorder, psychosis, or intellectual disability.

Group Assignment

Children and caregivers completed a comprehensive psychoeducational evaluation that included detailed semi-structured parent clinical interviewing using the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Aged Children (K-SADS; Kaufman et al., 1997) and multiple norm-referenced parent and teacher questionnaires, including the Behavior Assessment System for Children (BASC-2/3; Kamphaus & Reynolds, 2015) and ADHD Rating Scale for DSM-IV/5 (ADHD-4/5; DuPaul et al., 2016). A psychoeducational report was provided to parents.

Children that met all of the following criteria were included in the ADHD group (n=197): 1) DSM-5 diagnosis of ADHD combined (n = 132), inattentive (n = 57), hyperactive/impulsive (n = 6), and other-specified (n = 2) by the directing clinical psychologist and multidisciplinary team based on K-SADS and differential diagnosis considering all available clinical information indicating onset, course, duration, and severity of ADHD symptoms consistent with the ADHD neurodevelopmental syndrome; (2) borderline/clinical elevations on at least one parent and one teacher ADHD subscale (i.e., >90th percentile); and (3) current impairment based on parent-report. Children with any current ADHD presentation specifiers were eligible given the instability of ADHD presentations (e.g., Willcutt et al., 2012).

Our standard assessment battery also included norm-referenced child internalizing disorder screeners, and additional standardized measures were administered clinically as needed to inform differential diagnosis and accurate assessment of comorbidities (e.g., semi-structured child clinical interviews, additional testing). Several children with ADHD also met criteria for common comorbidities based on this comprehensive psychoeducational evaluation, including 66 anxiety disorder (33.5%), 12 depression (6.8%), 17 oppositional-defiant disorder (8.6%)[1], and 18 autism spectrum disorder (9.1%). To improve generalizability given that comorbidity is the norm rather than the exception for children with ADHD (Wilens et al., 2002), these children were retained in the sample. Further, 50 children with ADHD (25.4%) screened positive for a specific learning disorder. Positive screens for learning disorders were defined based on scores ≥ 1.5 SD below age-norms on one or more KTEA-3 academic skills battery reading and math subtests, as specified in DSM-5 (APA, 2013). Forty-seven children (23.9%) with ADHD were prescribed psychostimulant medication, which was withheld ≥24 hours for neurocognitive testing.

The Non-ADHD group comprised 142 consecutive case control referrals who did not meet ADHD criteria, and included both neurotypical children and children with psychiatric disorders other than ADHD. Neurotypical children (57.0%) had normal developmental histories and nonclinical parent/teacher ratings and were recruited through community resources. Clinically-referred and evaluated children who did not meet ADHD criteria were also included in the Non-ADHD group. These Non-ADHD disorders were included to control for comorbidities in the ADHD group, and included diagnoses of anxiety (31%), depression (7.7%), and autism spectrum disorder (9.9%). Ten children in the Non-ADHD group (7.0%) screened positive for a learning disorder. The ADHD and Non-ADHD groups did not differ significantly in the proportion of children with clinical disorders other than ADHD (anxiety, depression, ASD; $p>.54$); however, the ADHD group had higher proportions of ODD and SLD as expected ($p<.001$).

Ninety-four Non-ADHD participants underwent identical evaluations to the ADHD group. Due to funding constraints, the remaining 48 Non-ADHD participants (33.8%) completed abbreviated evaluations that included parent BASC-3 and ADHD-RS-5, a 1 to 2-subtest IQ screener (described below), and detailed developmental, medical, educational, and psychiatric histories. Teacher BASCs were obtained for a subset of the abbreviated cases recruited during the school year ($n=14$). Neurotypical children that received the abbreviated evaluation had slightly higher child-reported anxiety symptoms ($p=.01$), but did not differ from the full evaluation neurotypical subgroup in terms of parent-reported or teacher-reported anxiety symptoms, age, IQ, SES, sex, working memory, or inhibitory control (all $p>.14$).

Procedure

Children completed the executive function tasks as part of a larger battery of neurocognitive testing that involved two to three sessions of approximately three hours each. All tasks were counterbalanced to minimize order effects. Children received brief breaks after each task

[1]As recommended in the K-SADS, oppositional-defiant disorder (ODD) was diagnosed only with evidence of multi-informant/multi-setting symptoms. ODD prevalence in the ADHD group was 34.9% based on meeting parent-reported symptom counts.

and preset longer breaks every 2–3 tasks to minimize fatigue. For all testing, performance was monitored at all times by the examiner, who was stationed just outside of the testing room (out of the child's view) to provide a structured setting while minimizing performance improvements associated with examiner demand characteristics (Gomez & Sanson, 1994).

Measures

Working Memory Tasks

Rapport working memory tasks.: The Rapport et al. (2009) computerized phonological and visuospatial working memory test and administration instructions are identical to those described in Kofler et al. (2019). The variance common to these tasks has been proposed to reflect central executive abilities (Rapport et al., 2009). Reliability and validity evidence includes high internal consistency (α = .82-.97; Kofler et al., 2018); 1- to 3-week (.76-.90; Sarver et al., 2015) and 10-week (.73-.84; Kofler et al., 2023) test-retest reliability; and expected magnitude relations with working memory updating and complex span tasks (r = .61-.69; Wells et al., 2018). Each working memory test consisted of six trials at each set size (3–6 stimuli/trial), administered in randomized/unpredictable order as recommended (e.g., Kofler et al., 2017), yielding 24 trials. Five practice trials were administered before each task (80% correct required).

For the *phonological working memory task*, children were presented a series of jumbled numbers and a capital letter. The letter never appeared in the first or last position of the sequence to minimize potential primacy and recency effects, and was counterbalanced across trials to appear an equal number of times in the other serial positions (i.e., position 2, 3, 4, or 5). Children were instructed to verbally recall numbers in order from smallest to largest, and to say the letter last (e.g., 4H62 is correctly recalled as 246H). For the *visuospatial working memory task*, children were shown nine squares arranged in three offset vertical columns. A series of 2.5 cm diameter dots (3, 4, 5, or 6) were presented sequentially in one of the nine squares during each trial, such that no two dots appeared in the same square on a given trial. All dots presented within the squares were black with the exception of one red dot that was counterbalanced across trials to appear an equal number of times in each of the nine squares, but never presented as the first or last stimulus to minimize potential primacy and recency effects. Children reordered the dot locations (black dots in serial order, red dot last) and responded on a modified keyboard. Partial-credit unit scoring (i.e., stimuli correct per trial) was used to index overall working memory performance as recommended (Conway et al., 2005), computed separately for the phonological and visuospatial working memory tests. Higher scores reflect better working memory.

Inhibitory Control Tasks

Stop-signal inhibitory control.: The stop-signal task and administration instructions are identical to those described in Alderson et al. (2008). Psychometric evidence includes high internal consistency (α = .83-.89); 3-week test-retest reliability (.72); and convergent validity with other inhibition tests (Soreni et al., 2009). Go-stimuli were uppercase letters X and O presented for 1000 ms at the center of the screen (500 ms ISI; total trial duration = 1500 ms). Xs and Os appeared with equal frequency throughout the experimental blocks. The stop-stimulus was a 1000 Hz auditory tone presented randomly on 25% of trials.

Stop-signal delay (SSD; the latency between go- and stop-stimuli presentation) was initially set to 250 ms and dynamically adjusted ±50 ms contingent on the child's performance on each stop trial. Children completed two practice blocks prior to four experimental blocks of 32 trials/block using a modified response pad. Data validity was checked for each participant for each of the 4 task blocks using the Verbruggen et al. (2013) criteria for violations of the race model of inhibition (e.g., mean RT on failed stop trials > mean RT on go trials, probability of responding on stop trials between 25% and 75%, 15%+ omission errors); invalid blocks were treated as missing and imputed as described below. Inhibitory control was operationalized as the speed of the inhibitory stop process (iSSRT) and was estimated separately for each of the task blocks using the Verbruggen et al. (2013) integration method. Lower scores indicate better inhibition.

Go/no-go inhibitory control.: The go/no-go test and administration instructions are identical to those described in Kofler et al. (2019). Psychometric evidence includes high internal consistency (α=.95), as well as convergent validity with other inhibitory control measures (Kofler et al., 2019). Children were presented with a randomized series of vertical (go stimuli) and horizontal (no-go stimuli) rectangles at the center of the screen (2000 ms presentation, ISI jittered 800–2000 ms to minimize anticipatory responding). Children were instructed to quickly click the mouse when a vertical rectangle appeared, but to withhold a response when a horizontal rectangle appeared. A ratio of 80:20 go:no-go stimuli was selected to maximize prepotency (Kane & Engle, 2003). Children completed a 10-trial practice (80% required) followed by 4 continuous blocks of 25 trials each. Commission errors reflect failed inhibitions (i.e., incorrectly responding to no-go trials) and served as the primary index of inhibitory control during the task. Lower scores indicate better inhibition.

Executive Function Dimension Reduction—Task impurity was controlled by computing Bartlett maximum likelihood component scores based on intercorrelations among all 4 executive function tests (Distefano et al., 2009), which parsed the 8 working memory and 8 inhibitory control task blocks/set size variables into 2 component scores (42.82% of variance explained; Supplementary Table 1). A two-component, orthogonal solution was specified *a priori* to derive separate estimates of working memory and inhibitory control based on theory and previous empirical work (e.g., Miyake et al. 2000). These principal components analysis-derived component scores provide estimates of reliable, construct-level variance attributable to domain-general working memory and inhibitory control. This formative method for estimating executive functioning was selected because (a) such methods have been shown to provide higher construct stability relative to confirmatory/reflective approaches (Willoughby et al., 2016); and (b) estimating executive functioning at the construct-level rather than measure-level was expected to maximize associations with the study's outcomes via the removal of task-specific and error variance. These component scores were used for all analyses. Higher scores reflect better working memory, but worse inhibitory control.

Anxiety Symptoms—The *Multidimensional Anxiety Scale for Children* 2nd Edition Self-Report (MASC-2; March, 2013) was completed by children to assess symptoms related to anxiety disorders. Child self-reported anxiety was utilized as our primary indicator of

anxiety due to prior work demonstrating that child report of anxiety appears to show greater associations with neurocognitive functions than parent report (Bloemsma et al., 2013; Read et al., 2020) and appears to be more sensitive to early symptom emergence than parent reports (Cole et al., 2002). The MASC-2 consists of 50 items (4-point Likert scale) and the total score measures the overall extent to which the child is experiencing anxiety symptoms. The MASC-2 total score has demonstrated high internal consistency (α=.92) and 1- to 4-week test-retest reliability (*r*=.89; March, 2013). Higher raw scores reflect greater quantity/severity of anxiety symptoms.

The *Behavioral Assessment System for Children* (BASC-2/3; Kamphaus & Reynolds, 2015) parent and teacher forms were used in sensitivity analyses and consist of 139–175 items that assess internalizing, externalizing, and adaptive behaviors in children and adolescents ages 2–21. Psychometric support includes high internal consistency (α=.85-.96) and 1–10 week test-retest reliability (*r*=.84-.90). Age- and sex-normed T-scores were obtained via conversion of raw scores based on the national standardization sample. Parent and teacher Anxiety subscales were used to probe our *a priori* decision to define child self-reported anxiety as our primary indicator of anxiety (7–14 items; 4-Point Likert scale). Higher scores indicate higher levels of parent- or teacher-perceived anxiety.

Socioeconomic Status (SES) and Global Intellectual Functioning (IQ)

Hollingshead SES was estimated based on caregiver(s)' education and occupation (Cirino et al., 2002). In addition, children were administered either a 4-subtest (full evaluation) or a 1–2 subtest (abbreviated battery) Short-Form of the WISC-V (Sattler et al,, 2016; Wechsler, 2014)

Data Analysis Overview

The study's primary questions involved the extent to which ADHD is associated with deficits in each executive function, and the extent to which child trait anxiety confounds our estimates of the magnitude/presence of these expected impairments. Thus, a series of hierarchical regressions were conducted, with separate models for each executive function outcome (inhibitory control, working memory). Step 1 included demographic covariates (child age, sex, SES). ADHD diagnostic status (yes/no) was then added (Step 2) to estimate the magnitude of executive function impairments prior to accounting for anxiety. Child self-reported anxiety (MASC-2 total raw score) was then entered in Step 3, followed by the interaction between the two in the final step (Step 4). Our *a priori* plan called for significant ADHD × anxiety interactions to be probed with simple slope analyses to estimate the magnitude of ADHD-related executive function deficits at one standard deviation above and below mean anxiety levels. All continuous anxiety variables were mean-centered prior to analysis and analyses was conducted using SPSS Version 29. Standardized β-weights for pathways with ADHD diagnostic status as the predictor were converted to Cohen's *d* effect sizes to aid interpretability (small = 0.20; medium= 0.50; large= 0.80; Cohen, 1988).

These primary models were then supplemented with a series of sensitivity analyses to probe the impact of our *a priori* decisions to (a) select child self-report as our primary indicator of anxiety symptoms, and (b) include children with autism spectrum disorders

in both the ADHD and non-ADHD groups. Two additional sensitivity analyses were added during the peer review process. These involved (a) testing the impact of our *a priori* decision to maximize control for task impurity by specifying uncorrelated working memory and inhibition components in our dimension reduction model, and (b) examining the extent to which trait anxiety's impact may be curvilinear, based on the state arousal-performance hypothesis suggesting that there may be an optimal level of arousal that facilitates performance whereas levels that are too low or too high may impair performance (e.g., Easterbrook, 1959; Yerkes & Dodson, 1908).

First, we repeated the primary models twice across different informants: once using parent report and once using teacher report of child anxiety symptoms. Although prior work suggests that child self-report is optimal for assessing internalizing symptoms such as anxiety (Cole et al., 2002), parent and teacher report may also be informative in assessing observable anxiety symptoms and anxiety symptoms that primarily manifest in school. Next, we repeated the primary models using anxiety diagnosis (yes/no) in place of dimensional measures of anxiety. While the primary analyses allowed us to assess anxiety symptoms across a continuum of severity, grouping based on anxiety diagnoses allowed us to specifically assess the role of clinically significant levels of anxiety. All anxiety diagnoses were included in the initial analyses (i.e., generalized anxiety disorder, social anxiety disorder, separation anxiety disorder, specific phobia, and other-specified anxiety disorder). We then conducted the same model, but defined a more specific anxiety group with children diagnosed with either generalized anxiety disorder (GAD) or social anxiety disorder (SAD) based on prior work finding associations between these two domains of anxiety and executive function (e.g., Hallion et al., 2017; Visu-Petra et al., 2013; Zainal & Newman, 2018). The final sensitivity analysis that was planned *a priori* involved repeating the primary analysis again, this time excluding children diagnosed with ASD based on prior work suggesting that children with ASD may underreport their anxiety symptoms (e.g., Kalvin et al., 2020).

Finally, we repeated the primary models two more times, first with oblique working memory and inhibition components, and second with an additional step that involved adding quadratic anxiety (i.e., anxiety^2) and quadratic anxiety^2 × ADHD terms to the model.

Power Analyses

A power analysis was conducted using GPower v3.1 (Faul et al., 2007) to determine our sensitivity for detecting effects. For $\alpha = .05$ and power $(1-\beta) = .80$, in the event that all 6 potential predictors were retained in the final model (age, sex, SES, ADHD status, anxiety, and ADHD status × anxiety), we would be sufficiently powered to detect $R^2 = .04$. A single predictor is expected to be significant if it explains at least 2.28% of the variance in an executive function domain. Similarly, our sample size is powered to detect significant increases in model R^2 (ΔR^2) of .02. Thus, the study is sufficiently powered to detect clinically meaningful effects.

Results

Preliminary Analyses

All raw data were screened for univariate outliers, defined as values three standard deviations above or below the mean for the ADHD and non-ADHD groups separately. Outliers were corrected to the next most extreme value in the sample (0.37% and 0.47% of data points affected for ADHD and non-ADHD groups, respectively). Missing data were imputed using expectation maximization based on all available data and were determined to be missing completely at random (Little's MCAR test: $\chi^2 = 363.82$, $p = .82$). This affected 0.95% of data points. Sample demographics are shown in Table 1. Parent and teacher ADHD ratings were significantly higher for the ADHD relative to non-ADHD group as expected (Table 1). The ADHD and non-ADHD groups did not significantly differ from one another on child and parent report of anxiety symptoms, while teachers reported the non-ADHD group to have slightly lower anxiety symptoms (M=52.65 vs. 55.80; p=.049). In addition, the non-ADHD group was slightly older (M=10.58 vs. 10.12; p=.002) and had slightly higher IQ scores (M=105.59 vs. 101.22; p=.005), but did not differ from the ADHD group in terms of SES (p=.63). Finally, the ADHD group had a greater proportion of male participants and lower proportion of Asian children than the non-ADHD group (p=.02; p=.01, respectively).

The zero-order correlation matrix is shown in Table 2. In terms of demographics, older age was significantly associated with better working memory ($r = .41$, $p < .001$) and inhibitory control ($r = -.24$, $p < .001$). Higher SES was associated with better working memory ($r = .17$, $p = .001$), and decreased parent ($r = -.15$, $p = .006$) and teacher ($r = -.14$, $p = .01$) reported anxiety. Finally, higher IQ was associated with better working memory ($r = .37$, $p < .001$), higher child-reported anxiety ($r = .14$, $p = .01$), lower teacher-reported anxiety ($r = -.13$, $p = .02$), and diagnoses of GAD and/or SAD ($r = .12$, $p = .03$). IQ was not included as a covariate based on compelling statistical, methodological, and conceptual rationale against covarying IQ when investigating cognitive processes in ADHD (Dennis et al., 2009), and because IQ appears to reflect, in part, an outcome rather than a cause of executive function/cognitive control abilities (e.g., Engle et al., 1999). In other words, covarying IQ would preclude conclusions regarding executive functioning/cognitive control by fundamentally changing our primary predictor variables, and remove significant variance associated with our predictors and outcomes of interest (Dennis et al., 2009).

Primary Analyses

Inhibitory Control—Results of Step 1 indicated that both age ($\beta = -.25$, $p < .001$) and sex ($\beta = -.11$, $p = .045$) were significant predictors of inhibitory control, such that older children and female participants had better inhibitory control, while SES did not significantly predict inhibitory control ($\beta = .07$, $p = .17$; $R^2 = .07$, $p < .001$). Adding ADHD diagnosis ($\beta = .15$, $d = 0.31$, $p = .005$) to the model significantly increased the amount of explained variance ($\Delta R^2 = .02$, $p = .005$; Step 2). Individuals with ADHD exhibited small magnitude inhibitory control deficits ($d = 0.31$). In Steps 3 and 4, anxiety symptoms ($\beta = .02$, $\Delta R^2 = .00$, $p = .67$; Step 3) and the ADHD diagnosis × anxiety interaction ($\beta = .15$, $\Delta R^2 = .00$, $p = .39$; Step 4)

were not significant predictors of inhibition performance and did not significantly increase the amount of explained variance.

Working Memory—Results of Step 1 indicated that older age ($\beta = .40$, $p < .001$) and higher SES ($\beta = .16$, $p = .001$) predicted better working memory, whereas sex was not a significant predictor ($\beta = -.01$, $p = .78$; $R^2 = .19$, $p < .001$). Adding ADHD diagnosis ($\beta = -.44$, $d = -0.99$, $p < .001$) to the model significantly increased the amount of explained variance ($\Delta R^2 = .19$, $p < .001$; Step 2) and indicated that children with ADHD exhibited large magnitude deficits in working memory ($d = 0.99$). However, similar to the inhibition model, anxiety symptoms ($\beta = .07$, $\Delta R^2 = .01$, $p = .09$; Step 3) and the ADHD diagnosis × anxiety interaction ($\beta = .12$, $\Delta R^2 = .00$, $p = .42$; Step 4) were not significant predictors of inhibition performance and did not significantly increase the amount of explained variance.

Sensitivity Analyses: Informant and Anxiety Diagnoses

Overall, our primary findings indicate that ADHD is associated with large magnitude impairments in working memory ($d = 0.99$) and small magnitude impairments in inhibitory control ($d = 0.31$), but that trait anxiety neither inflates or deflates these estimates, nor is it independently associated with executive function difficulties above and beyond ADHD status. To probe the extent to which the pattern of results reported above was impacted by our *a priori* decisions to (a) operationalize anxiety as child self-report of anxiety; (b) assess anxiety dimensionally; and (c) include children with ASD in our sample, we conducted a series of sensitivity analyses examining the impact of different informants (parent, teacher) and assessing anxiety using categorical diagnostic groupings. Sensitivity analyses added during the peer review process also tested the impact of our *a priori* decision to maximally control for task impurity by specifying orthogonal working memory and inhibition components, and probed the extent to which anxiety may have a curvilinear (quadratic) impact on executive function performance. Reporting is truncated for readability.

First, we repeated the primary analyses with parent-report of anxiety instead of child self-report. The results were unchanged from the primary results. ADHD diagnostic status significantly predicted both inhibitory control ($\beta = .15$, $d = 0.31$, $p = .01$) and working memory ($\beta = -.44$, $d = -0.99$, $p < .001$), with effect sizes that were identical to the primary model, while parent-reported anxiety did not (respectively, $\beta = -.04$, $p = .50$; $\beta = .01$, $p = .79$), after controlling for age, sex, and SES (IC: $\Delta R^2 = .02$, $p = .02$; WM: $\Delta R^2 = .18$, $p < .001$; Step 2). Further, the interaction between ADHD and parent-reported anxiety did not significantly increase the amount of variance explained in either inhibitory control ($\Delta R^2 = .00$, $p = .37$; Step 3) or working memory ($\Delta R^2 = .00$, $p = .74$; Step 3).

Next, we repeated the primary analyses with teacher-report of anxiety. As with parent-report of anxiety, there were not deviations from the primary results. Above and beyond demographic variables, ADHD was a significant unique predictor of both inhibitory control ($\beta = .13$, $d = 0.27$, $p = .02$) and working memory ($\beta = -.43$, $d = -0.96$, $p < .001$), while anxiety (respectively, $\beta = .08$, $p = .13$; $\beta = -.08$, $p = .07$) and the ADHD × teacher-reported anxiety interaction were not (both $\Delta R^2 = .00$, $p > .37$).

We then probed our decision to utilize dimensional measurement of anxiety, rather than categorical anxiety diagnoses criteria. Consistent with the primary results, children with ADHD demonstrated worse inhibitory control than children without ADHD (β = .15, d = 0.31, p = .01), while anxiety diagnosis was not a significant predictor (β = −.02, d = −0.04, p = .68). The interaction between ADHD and anxiety diagnosis also did not impact our estimates of ADHD-related inhibition deficits (ΔR^2 = .007, p = .80). In contrast, both ADHD (β = −.44, d = −0.99, p < .001) and anxiety disorders (β = .09, d = 0.19, p = .04) were significant predictors of working memory, such that children with ADHD exhibited large magnitude working memory deficits (d = 0.99) whereas children with an anxiety disorder (d = 0.19) demonstrated slightly *better* working memory. However, anxiety was no longer a significant unique predictor once the interaction between ADHD and anxiety was included in the model (β = .06, d = 0.13, p = .41), despite this interaction term not explaining significant variance in working memory (ΔR^2 = .00, p = .50). Results remained unchanged when our anxiety diagnosis category was limited to generalized anxiety disorder (GAD) and/or social anxiety disorder (SAD; combined n=83).

Next, we repeated the primary model again, this time excluding children diagnosed with ASD from the analyses. Results were unchanged from the primary inhibitory control model. However, similar to the anxiety disorder models, with children with ASD excluded we found that greater anxiety symptoms were significantly associated with slightly better working memory (β = .10, d = 0.20, p = .02), but once again were no longer a significant predictor once the interaction term, also non-significant, was included in the model (ΔR^2 = .00, p = .21; Step 3). We then retested the primary models using oblique estimates of working memory and inhibition, which were significantly correlated with each other (r = −.32, p < .001) but remained statistically indistinguishable from their orthogonal counterparts (both r = .99, p < .001). The pattern and interpretation of results was unchanged (ADHD significant at p < .001, anxiety and ADHD × anxiety non-significant at p > .44), with the exception that the estimate of inhibition deficits in ADHD was somewhat larger (β = .22, d = 0.46, p < .001) without controlling for the inhibition tasks' working memory demands; the estimate of ADHD-related working memory deficits was also slightly larger (β = .46, d = 1.05, p < .001), highlighting the importance of controlling for task impurity for maximizing effect certainty.

Finally, we re-tested the primary models again, this time adding a quadratic anxiety term as well as the quadratic anxiety × ADHD interaction. For both the inhibition and working memory models, adding these terms failed to increase the explained variance (both ΔR^2 = .01, p = .14-.18), and neither anxiety^2 nor anxiety^2 × ADHD predicted inhibition (both p = .08) or working memory (both p > .28). This pattern of results was seen both when the additional terms were entered together as well as when they were entered as separate Steps.

Discussion

The current study tested conceptual model predictions suggesting that co-occurring trait anxiety may be systematically affecting our estimates of the magnitude of executive function deficits in children with ADHD, using both categorical and dimensional estimates of anxiety across multiple informants in a large and clinically evaluated sample. Contrary to our

hypotheses, we found that anxiety did not impact our estimates of ADHD-related working memory/inhibitory control deficits. Specifically, while ADHD diagnosis was robustly associated with small magnitude impairments in both inhibitory control (d = 0.31) and large magnitude impairments in working memory (d = 0.99), child anxiety symptoms assessed across multiple informants (child, parent, teacher) generally did not affect the significance or magnitude of these estimates, which were also robust to control for demographic variables (age, sex, SES). In addition, anxiety was not associated with unique difficulties in either executive function in any tested model. These results are inconsistent with previous work suggesting that anxiety may buffer or mask inhibition deficits (Bloemsma et al., 2013; Maric et al., 2018; Ursache & Raver, 2014), while producing overestimates of working memory deficits in children with ADHD (Jarrett et al., 2016; Moran, 2016; Read et al., 2020). Further, the results of the current study are also inconsistent with past studies that have found anxiety to be predictive of executive function difficulties above and beyond ADHD (Castagna et al., 2019; Read et al., 2020). In contrast, our results are more consistent with meta-analytic and other empirical studies who reported null results when examining the moderating effect of anxiety on inhibitory control or working memory in children with ADHD (Adamo et al., 2021; Read et al., 2020; van der Meer et al., 2018).

The results of the current study do not support conceptual models suggesting that anxiety may buffer or mask inhibitory control deficits in children with ADHD (Maric et al., 2018; Schatz & Rostain, 2006). While ADHD was associated with small magnitude deficits in inhibitory control (d = 0.31), this estimate was not meaningfully impacted by control for anxiety, either on its own or as a potential linear or quadratic moderator. Further, trait anxiety did not predict children's inhibitory control abilities in any tested model. Our primary analyses utilized dimensional measurement of anxiety, while many previous studies finding better inhibitory control with higher levels of anxiety evaluated differences between ADHD and ADHD plus a comorbid anxiety diagnosis groups (Maric et al., 2018; Menghini et al., 2018; Yurtbaşı et al., 2018). Indeed, previous results examining anxiety dimensionally have yielded more mixed results (Adamo et al., 2021; Read et al., 2020; Ruf et al., 2017). To address the possibility that these differing results were the result of methodological differences in assessment of anxiety and the presence of clinically significant levels of anxiety, the current study also examined anxiety as a categorical diagnosis and the results were unchanged. That is, anxiety disorders do not appear to be associated with inhibitory control difficulties above and beyond ADHD diagnosis. Further, trait anxiety did not affect the magnitude or significance of ADHD-related inhibition deficits when anxiety was operationalized based on child, parent, or teacher report of the child's anxiety symptoms, as well as when anxiety was assessed as a categorical diagnosis. In other words, the results of the current study suggest that trait anxiety is not associated with inhibitory control difficulties in clinically evaluated children when assessed using component methods that isolate reliable variance specific to inhibition based on multiple, construct valid tests. Thus, these results increase confidence that the smaller magnitude impairments in inhibition relative to working memory observed in children with ADHD are not an artifact of the lack of control for co-occurring anxiety symptoms or diagnoses in most prior studies.

Contrary to some prior work (Jarrett et al., 2016; Read et al., 2020; Tannock, 2009), higher levels of anxiety did not produce overestimates of working memory deficits in children

with ADHD. Dimensional measures of anxiety across child, parent, and teacher report did not account for significant variance in working memory abilities above and beyond ADHD diagnosis and demographic variables, nor did these measures of anxiety moderate the large magnitude working memory deficits exhibited by children with ADHD (d = 0.99). In contrast, two of our sensitivity analyses found that anxiety was associated with working memory, but in the opposite direction than expected. That is, children with an anxiety disorder (when controlling for ADHD) had slightly *better* working memory than children without an anxiety disorder (d = 0.19), and higher child self-reported anxiety was also associated with slightly better working memory when children diagnosed with ASD were excluded from analyses (d = 0.20). However, these effects were no longer significant once the non-significant interaction effects were added to the model, suggesting that children with elevated anxiety appear to have similar, or slightly better, working memory than their less anxious peers.

Assessment of working memory and inhibitory control may play a role in differences in results across studies. For example, past studies have often used assessments of working memory that have been shown to be reflective of short-term memory or general cognitive abilities, rather than working memory specifically (for reviews, see Canivez et al., 2016; Rapport et al., 2013; Snyder et al., 2015). The current study used multiple, well-validated tests of both working memory and inhibitory control, as well as factor analytic procedures to derive best estimates of the two executive functions that minimized task-specific and error variance. Thus, it is possible that the difference between the current and some previous studies suggests that anxiety may exert an effect on short-term memory or general cognitive functioning, but not working memory processes specifically. That is, working memory-specific cognitive processes – such as reordering, updating, and dual-processing/interference control (Fosco et al., 2020) – may be less influenced by anxiety than more general abilities or short-term memory that may be more negatively affected by reduced filtering efficiency. Similarly, while previous studies have found interactions between anxiety and ADHD in predicting individual inhibitory control performance metrics on single tests (Menghini et al., 2018; Ruf et al., 2017), the current study found no associations when examining component estimates of inhibitory control that are less likely to be confounded by the non-inhibition processes (e.g., working memory; Kofler et al., 2023) required for performance on any given inhibition test. This contrast may be suggestive of task-specific rather than construct-level effects, and/or differential associations based on the multi-component nature of inhibitory control (e.g., Alderson et al., 2008).

Limitations and Future Directions

Finally, while the primary goal of the current study was to evaluate the extent to which anxiety systematically inflates or blunts/masks estimates of the magnitude of executive function deficits in children with ADHD, other work has taken a more dimensional approach to ADHD (e.g., Castagna et al., 2019; Read et al., 2020). Thus, while the current study indicates that trait anxiety does not affect estimates of working memory or inhibitory control deficits in children diagnosed with ADHD, it remains possible that anxiety may affect the strength of associations between these executive functions and specific ADHD symptom clusters/symptoms (e.g., Jarrett & Ollendick, 2008; Schatz & Rostain, 2006). Further, prior

work suggests that deficits in executive function contribute functionally if not causally to behavioral manifestations of ADHD symptoms (Barkley, 1997; Kofler et al., 2010, 2020; Rapport et al., 2001, 2009), whereas causal conclusions cannot be drawn from the current study given the cross-sectional design. Further research in this area is of particular clinical relevance given the complex associations between anxiety and ADHD across the lifespan that continue to be debated in the literature (for review, see Koyuncu et al., 2022). Longitudinal and experimental work is necessary to further elucidate the development and directionality between ADHD and anxiety symptoms, and executive functioning, given the differential role that executive function may play in both the development and maintenance of each of these symptom domains (e.g., Eysenck et al., 2007; Ferreri et al., 2011; Rapport et al., 2001).

Additionally, influential conceptual models have suggested several mechanisms by which anxiety may exert its effect on executive function, as well as the relation between ADHD and executive function. For example, trait anxiety may increase the likelihood of state anxiety that in turn increases motivation and effort (Tannock, 2009) or cortical arousal (Arnsten, 2009), which could augment performance on cognitive tasks, or this anxiety may exacerbate difficulties during cognitive tasks as a result of increased worries about competence (Schatz & Rostain, 2006). The current study did not assess the extent to which these mechanisms may have contributed to the pattern of results seen. Similarly, the current study assessed anxiety at a trait level, rather than measuring individual differences in the level of anxiety that children may have experienced during the cognitive tasks themselves (i.e., state anxiety). Thus, the current study does not rule out the possibility that state anxiety may impact executive function test performance. Interestingly, however, while some prior work has suggested that state and trait anxiety have differential effects on executive functions such as working memory and inhibitory control (Ursache & Raver, 2014; Visu-Petra et al., 2013), meta-analytic evidence indicates that relations between anxiety and executive functions do not differ significantly when based on state (induced) versus trait anxiety (Moran et al., 2016; Shi et al., 2019). Future work should explore the distinction between trait and state anxiety in these relations, particularly to evaluate specific theories about the mechanisms by which anxiety can exert an effect on executive functioning.

Finally, the clinical nature of a subset of our Non-ADHD comparison group should be acknowledged. Thus, although the current study suggests strongly that trait anxiety does not impact estimates of executive function impairments in ADHD, it remains possible that these estimates would have been larger if the ADHD group was compared to a fully neurotypical/healthy control group. Future studies are needed to determine the extent to which other commonly co-occurring conditions may impact executive functioning for children with ADHD.

Conclusion

Taken together, the current study found that trait anxiety, assessed both dimensionally across multiple informants and as categorical diagnoses, does not appear to significantly affect our estimates of the magnitude of executive function deficits observed in children with ADHD. That is, trait anxiety was not uniquely predictive of working memory or inhibitory control

difficulties, nor did it differentially produce over- or under-estimates of ADHD-related difficulties in working memory or inhibition. These findings do not support theoretical models suggesting that anxiety may buffer against/mask inhibitory control difficulties while exacerbating working memory problems in children with ADHD (Schatz & Rostain, 2006; Tannock, 2009). Indeed, consistent with a recent meta-analysis (Majeed et al., 2023), results from the current study suggest that greater anxiety may actually be associated with slightly *better* working memory accuracy for clinically evaluated children broadly. However, it remains possible that trait anxiety may impact executive functioning for a subset of children with ADHD given the disorder's well documented neurocognitive heterogeneity (e.g., Karalunas et al., 2017; Kofler et al., 2019) as well as because group level findings likely do not translate perfectly into individual level functioning (i.e., ergodicity). Future research should further probe the extent to which anxiety may interact to affect the strength of association between executive functioning abilities and specific ADHD symptoms, as well as the mechanisms that may underlie these effects if detected.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

Adamo N, Michelini G, Cheung CHM, Buitelaar JK, Asherson P, Rijsdijk F, & Kuntsi J (2021). Does co-occurring anxiety modulate ADHD-related cognitive and neurophysiological impairments? Journal of Attention Disorders, 25(8), 1135–1145.

Alderson RM, Rapport MD, & Kofler MJ (2007). Attention-deficit/hyperactivity disorder and behavioral inhibition: A meta-analytic review of the stop-signal paradigm. Journal of Abnormal Child Psychology, 35(5), 745–758.

Alderson RM, Rapport MD, Sarver DE, & Kofler MJ (2008). ADHD and behavioral inhibition: A re-examination of the stop-signal task. Journal of Abnormal Child Psychology, 36(7), 989–998.

Alfonso SV, & Lonigan CJ (2021). Trait anxiety and adolescent's academic achievement: The role of executive function. Learning and Individual Differences, 85, 101941.

Arnsten AFT (2009). Toward a new understanding of attention-deficit hyperactivity disorder pathophysiology: An important role for prefrontal cortex dysfunction. CNS Drugs, 23, 33–41.

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). American Psychiatric Publishing.th

Barkley RA (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. Psychological Bulletin, 121(1), 65–94.

Bishop C, Mulraney M, Rinehart N, & Sciberras E (2019). An examination of the association between anxiety and social functioning in youth with ADHD: A systematic review. Psychiatry Research, 273, 402–421.

Bloemsma JM, Boer F, Arnold R, Banaschewski T, Faraone SV, Buitelaar JK, Sergeant JA, Rommelse N, & Oosterlaan J (2013). Comorbid anxiety and neurocognitive dysfunctions in children with ADHD. European Child & Adolescent Psychiatry, 22(4), 225–234.

Castagna PJ, Calamia M, Roye S, Greening SG, & Davis TE (2019). The effects of childhood inattention and anxiety on executive functioning: Inhibition, updating, and shifting. Attention Deficit and Hyperactivity Disorders, 11(4), 423–432.

Chan ES, Macias M, & Kofler MJ (2022). Does Child Anxiety Exacerbate or Protect Against Parent–Child Relationship Difficulties in Children with Elevated ADHD Symptoms?. Journal of Psychopathology and Behavioral Assessment, 1–13.

Chan ESM, Groves NB, Marsh CL, Miller CE, Richmond KP, & Kofler MJ (2022). Are there resilient children with ADHD? Journal of Attention Disorders, 26(5), 643–655.

Cirino PT, Chin CE, Sevcik RA, Wolf M, Lovett M, & Morris RD (2002). Measuring socioeconomic status: Reliability and preliminary validity for different approaches. Assessment, 9(2), 145–155.

Cohen J (1988). Statistical power analysis for the behavioural sciences. Lawrence Erlbaum Associates.

Cole DA, Tram JM, Martin JM, Hoffman KB, Ruiz MD, Jacquez FM, & Maschman TL (2002). Individual differences in the emergence of depressive symptoms in children and adolescents: A longitudinal investigation of parent and child reports. Journal of Abnormal Psychology, 111(1), 156–165.

Conway ARA, Kane MJ, Bunting MF, Hambrick DZ, Wilhelm O, & Engle RW (2005). Working memory span tasks: A methodological review and user's guide. Psychonomic Bulletin & Review, 12(5), 769–786.

Dennis M, Francis DJ, Cirino PT, Schachar R, Barnes MA, & Fletcher JM (2009). Why IQ is not a covariate in cognitive studies of neurodevelopmental disorders. Journal of the International Neuropsychological Society: JINS, 15(3), 331–343.

Distefano C, Zhu M, & Mîndrilă D (2009). Understanding and using factor scores: Considerations for the applied researcher. Practical Assessment, Research, and Application, 14(20), 1–11.

DuPaul GJ, Power TJ, Anastopoulos AD, & Reid R (2016). ADHD Rating Scale-5 for children and adolescents: Checklists, norms, and clinical interpretation. Guilford Press.

Engle RW, Tuholski SW, Laughlin JE, & Conway ARA (1999). Working memory, short-term memory, and general fluid intelligence: A latent-variable approach. Journal of Experimental Psychology: General, 128(3), 309–331.

Eysenck MW, Derakshan N, Santos R, & Calvo MG (2007). Anxiety and cognitive performance: Attentional control theory. Emotion, 7(2), 336–353.

Faul F, Erdfelder E, Lang A-G, & Buchner A (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behavior Research Methods, 39(2), 175–191.

Ferreri F, Lapp LK, & Peretti C-S (2011). Current research on cognitive aspects of anxiety disorders. Current Opinion in Psychiatry, 24(1), 49–54.

Fosco WD, Kofler MJ, Groves NB, Chan ESM, & Raiker JS (2020). Which "working" components of working memory aren't working in youth with ADHD? Journal of Abnormal Child Psychology, 48(5), 647–660.

Gomez R, & Sanson AV (1994). Effects of experimenter and mother presence on attentional performance and activity of hyperactive boys. Journal of Abnormal Child Psychology, 22(5), 517–529.

Halperin JM, & Schulz KP (2006). Revisiting the role of the prefrontal cortex in the pathophysiology of attention-deficit/hyperactivity disorder. Psychological Bulletin, 132(4), 560–581.

Hirsch CR, & Mathews A (2012). A cognitive model of pathological worry. Behaviour Research and Therapy, 50(10), 636–646.

Huizinga M, Dolan CV, & van der Molen MW (2006). Age-related change in executive function: Developmental trends and a latent variable analysis. Neuropsychologia, 44(11), 2017–2036.

Irwin Harper LN, Groves NB, Marsh CL, Cole AM, & Kofler MJ (2022). Does training working memory or inhibitory control produce far-transfer improvements in set shifting for children with ADHD? A randomized controlled trial. Child Neuropsychology, 1–21.

Irwin LN, Kofler MJ, Soto EF, & Groves NB (2019). Do children with attention deficit/hyperactivity disorder (ADHD) have set shifting deficits? Neuropsychology, 33(4), 470–481.

Jarrett MA, & Ollendick TH (2008). A conceptual review of the comorbidity of attention deficit/hyperactivity disorder and anxiety: Implications for future research and practice. Clinical Psychology Review, 28(7), 1266–1280.

Jarrett MA, Wolff JC, Davis TE, Cowart MJ, & Ollendick TH (2016). Characteristics of children with ADHD and comorbid anxiety. Journal of Attention Disorders, 20(7), 636–644.

Kalvin CB, Marsh CL, Ibrahim K, Gladstone TR, Woodward D, Grantz H, Ventola P, & Sukhodolsky DG (2020). Discrepancies between parent and child ratings of anxiety in children with autism spectrum disorder. Autism Research, 13(1), 93–103.

Kane MJ, & Engle RW (2003). Working-memory capacity and the control of attention: The contributions of goal neglect, response competition, and task set to Stroop interference. Journal of Experimental Psychology: General, 132(1), 47–70.

Karalunas SL, Gustafsson HC, Dieckmann NF, Tipsord J, Mitchell SH, & Nigg JT (2017). Heterogeneity in development of aspects of working memory predicts longitudinal attention deficit hyperactivity disorder symptom change. Journal of Abnormal Psychology, 126(6), 774–792.

Karr JE, Areshenkoff CN, Rast P, Hofer SM, Iverson GL, & Garcia-Barrera MA (2018). The unity and diversity of executive functions: A systematic review and re-analysis of latent variable studies. Psychological Bulletin, 144(11), 1147–1185.

Killeen PR, Russell VA, & Sergeant JA (2013). A behavioral neuroenergetics theory of ADHD. Neuroscience and Biobehavioral Reviews, 37(4), 625–657.

Kofler MJ, Irwin LN, Soto EF, Groves NB, Harmon SL, & Sarver DE (2019). Executive functioning heterogeneity in pediatric ADHD. Journal of Abnormal Child Psychology, 47(2), 273–286.

Kofler MJ, Rapport MD, Bolden J, Sarver DE, & Raiker JS (2010). ADHD and working memory: The impact of central executive deficits and exceeding storage/rehearsal capacity on observed inattentive behavior. Journal of Abnormal Child Psychology, 38(2), 149–161.

Kofler MJ, Sarver DE, Harmon SL, Moltisanti A, Aduen PA, Soto EF, & Ferretti N (2018). Working memory and organizational skills problems in ADHD. Journal of Child Psychology and Psychiatry, and Allied Disciplines, 59(1), 57–67.

Kofler MJ, Sarver DE, Spiegel JA, Day TN, Harmon SL, & Wells EL (2017). Heterogeneity in ADHD: Neurocognitive predictors of peer, family, and academic functioning. Child Neuropsychology, 23(6), 733–759.

Kofler MJ, Sarver DE, & Wells EL (2020). Working memory and increased activity level (hyperactivity) in ADHD: Experimental evidence for a functional relation. Journal of Attention Disorders, 24(9), 1330–1344.

Koyuncu A, Ayan T, Ince Guliyev E, Erbilgin S, & Deveci E (2022). ADHD and anxiety disorder comorbidity in children and adults: Diagnostic and therapeutic challenges. Current Psychiatry Reports, 24(2), 129–140.

Larson K, Russ SA, Kahn RS, & Halfon N (2011). Patterns of comorbidity, functioning, and service use for US children with ADHD, 2007. Pediatrics, 127(3), 462–470.

Lipszyc J, & Schachar R (2010). Inhibitory control and psychopathology: A meta-analysis of studies using the stop signal task. Journal of the International Neuropsychological Society, 16(6), 1064–1076.

Maric M, Bexkens A, & Bögels SM (2018). Is clinical anxiety a risk or a protective factor for executive functioning in youth with ADHD? A Meta-regression analysis. Clinical Child and Family Psychology Review, 21(3), 340–353.

Menghini D, Armando M, Calcagni M, Napolitano C, Pasqualetti P, Sergeant JA, Pani P, & Vicari S (2018). The influence of generalized anxiety disorder on executive functions in children with ADHD. European Archives of Psychiatry and Clinical Neuroscience, 268(4), 349–357.

Miyake A, Friedman NP, Emerson MJ ., Witzki AH., Howerter A., & Wager TD. (2000). The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: A latent variable analysis. Cognitive Psychology, 41(1), 49–100.

Moran TP (2016). Anxiety and working memory capacity: A meta-analysis and narrative review. Psychological Bulletin, 142(8), 831–864.

Overgaard KR, Aase H, Torgersen S, & Zeiner P (2016). Co-occurrence of ADHD and anxiety in preschool children. Journal of Attention Disorders, 20(7), 573–580.

Polanczyk GV, Willcutt EG, Salum GA, Kieling C, & Rohde LA (2014). ADHD prevalence estimates across three decades: An updated systematic review and meta-regression analysis. International Journal of Epidemiology, 43(2), 434–442.

Rapport MD, Bolden J, Kofler MJ, Sarver DE, Raiker JS, & Alderson RM (2009).Hyperactivity in boys with attention-deficit/hyperactivity disorder (ADHD): A ubiquitous core symptom or manifestation of working memory deficits? Journal of Abnormal Child Psychology, 37(4), 521–534.

Rapport MD, Chung K-M, Shore G, & Isaacs P (2001). A conceptual model of child psychopathology: Implications for understanding attention deficit hyperactivity disorder and treatment efficacy. Journal of Clinical Child & Adolescent Psychology, 30(1), 48–58.

Rapport MD, Orban SA, Kofler MJ, & Friedman LM (2013). Do programs designed to train working memory, other executive functions, and attention benefit children with ADHD? A meta-analytic review of cognitive, academic, and behavioral outcomes. Clinical Psychology Review, 33(8), 1237–1252.

Read N, Mulraney M, McGillivray J, & Sciberras E (2020). Comorbid anxiety and irritability symptoms and their association with cognitive functioning in children with ADHD. Journal of Abnormal Child Psychology, 48(8), 1035–1046.

Ruf BM, Bessette KL, Pearlson GD, & Stevens MC (2017). Effect of trait anxiety on cognitive test performance in adolescents with and without attention-deficit/hyperactivity disorder. Journal of Clinical and Experimental Neuropsychology, 39(5), 434–448.

Sarver DE, Rapport MD, Kofler MJ, Raiker JS, & Friedman LM (2015). Hyperactivity in attention-deficit/hyperactivity disorder (ADHD): Impairing deficit or compensatory behavior? Journal of Abnormal Child Psychology, 43(7), 1219–1232.

Schatz DB, & Rostain AL (2006). ADHD with comorbid anxiety: A review of the current literature. Journal of Attention Disorders, 10(2), 141–149.

Shaw P, Eckstrand K, Sharp W, Blumenthal J, Lerch JP, Greenstein D, Clasen L, Evans A, Giedd J, & Rapoport JL (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. Proceedings of the National Academy of Sciences, 104(49), 19649–19654.

Snyder HR, Miyake A, & Hankin BL (2015). Advancing understanding of executive function impairments and psychopathology: Bridging the gap between clinical and cognitive approaches. Frontiers in Psychology, 6(328), 1–24.

Soreni N, Crosbie J, Ickowicz A, & Schachar R (2009). Stop signal and Conners' continuous performance tasks: Test--retest reliability of two inhibition measures in ADHD children. Journal of Attention Disorders, 13(2), 137–143.

St Clair-Thompson HL, & Gathercole SE (2006). Executive functions and achievements in school: Shifting, updating, inhibition, and working memory. Quarterly Journal of Experimental Psychology, 59(4), 745–759.

Tannock R (2009). ADHD with anxiety disorders. In Brown TE (ed.), ADHD comorbidities: Handbook for ADHD complications in children and adults (pp. 131–155). American Psychiatric Publishing.

Ursache A, & Raver CC (2014). Trait and state anxiety: Relations to executive functioning in an at-risk sample. Cognition & Emotion, 28(5), 845–855.

van der Meer D, Hoekstra PJ, van Rooij D, Winkler AM, van Ewijk H, Heslenfeld DJ, Oosterlaan J, Faraone SV, Franke B, Buitelaar JK, & Hartman CA (2018). Anxiety modulates the relation between attention-deficit/hyperactivity disorder severity and working memory-related brain activity. World Journal of Biological Psychiatry, 19(6), 450–460.

van Lieshout M, Luman M, Buitelaar J, Rommelse NNJ, & Oosterlaan J (2013). Does neurocognitive functioning predict future or persistence of ADHD? A systematic review. Clinical Psychology Review, 33(4), 539–560.

Verbruggen F, Chambers CD, & Logan GD (2013). Fictitious inhibitory differences: How skewness and slowing distort the estimation of stopping latencies. Psychological Science, 24(3), 352–362.

Visu-Petra L, Miclea M, & Visu-Petra G (2013). Individual differences in anxiety and executive functioning: A multidimensional view. International Journal of Psychology, 48(4), 649–659.

Wells EL, Kofler MJ, Soto EF, Schaefer HS, & Sarver DE (2018). Assessing working memory in children with ADHD: Minor administration and scoring changes may improve digit span backward's construct validity. Research in Developmental Disabilities, 72, 166–178.

Wilens TE, Biederman J, Brown S, Tanguay S, Monuteaux MC, Blake C, & Spencer TJ (2002). Psychiatric comorbidity and functioning in clinically referred preschool children and school-age youths with ADHD. Journal of the American Academy of Child and Adolescent Psychiatry, 41(3), 262–268.

Willcutt EG, Doyle AE, Nigg JT, Faraone SV, & Pennington BF (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. Biological Psychiatry, 57(11), 1336–1346.

Willcutt EG, Nigg JT, Pennington BF, Solanto MV, Rohde LA, Tannock R, Loo SK, Carlson CL, McBurnett K, & Lahey BB (2012). Validity of DSM-IV attention deficit/hyperactivity disorder symptom dimensions and subtypes. Journal of Abnormal Psychology, 121(4), 991–1010.

Willoughby MT, Blair CB, & Family Life Project Investigators. (2016). Measuring executive function in early childhood: A case for formative measurement. Psychological Assessment, 28(3), 319–330.

Wright L, Lipszyc J, Dupuis A, Thayapararajah SW, & Schachar R (2014). Response inhibition and psychopathology: A meta-analysis of go/no-go task performance. Journal of Abnormal Psychology, 123(2), 429–439.

Yurtbaşı P, Aldemir S, Teksin Bakır MG, Aktaş Ş, Ayvaz FB, Piştav Satılmış Ş, & Münir K (2018). Comparison of neurological and cognitive deficits in children with ADHD and anxiety disorders. Journal of Attention Disorders, 22(5), 472–485.

Zainal NH, & Newman MG (2018). Executive function and other cognitive deficits are distal risk factors of generalized anxiety disorder 9 years later. Psychological Medicine, 48(12), 2045–2053.

Table 1.

Sample and demographic variables

Variable	ADHD (N=197)		Non-ADHD (N=142)		Cohen's *d*	*p*	Possible Range	Obtained Range		Skewness		Kurtosis	
	M	*SD*	*M*	*SD*				ADHD	Non-ADHD	ADHD	Non-ADHD	ADHD	Non-ADHD
Sex (%Male/Female)	63/37		50/50		--	.02	--	--	--	--	--	--	--
Ethnicity (% B/A/W/H/M)	16/0/69/6/9		10/4/65/8/13		--	.03	--	--	--	--	--	--	--
Age	10.12	1.39	10.58	1.34	.34	.002	8.00–13.37	8.10–13.34	8.29–13.37	0.58	0.35	−0.75	−0.84
SES	47.53	10.16	46.95	12.09	−.05	.63	11–66	21–66	11–66	−0.50	−0.61	−0.33	0.07
IQ	101.22	14.81	105.59	13.18	.31	.005	45–155	73–138	73–151	0.07	0.03	−0.67	0.51
ADHD Symptoms													
BASC-2/3 Parent ATT T-Score	68.53	7.10	57.53	10.30	−1.28	<.001	10–120	46–86	36–81	−0.53	0.16	0.95	−0.77
BASC-2/3 Parent HYP T-Score	69.45	12.61	55.13	12.18	−1.15	<.001	10–120	42–102	36–93	0.00	0.91	−0.37	0.64
Anxiety													
Diagnosis (%Yes/No)	33/67		31/69		--	.63	--	--	--	--	--	--	--
MASC-2 Self-Report Total Raw Score	63.51	23.00	60.96	20.92	−.12	.30	0–150	6–126	14–120	0.12	0.02	−0.33	−0.26
MASC-2 Self-Report Total T-Score	55.85	10.66	54.57	9.61	−.13	.25	40–90	40–89	40–81	0.45	0.22	−0.21	−0.64
BASC-2/3 Parent Anxiety Scale T-Score	55.97	11.86	55.88	11.91	−.01	.94	10–120	30–89	31–89	0.38	0.44	−0.41	−0.26
BASC-2/3 Teacher Anxiety Scale T-Score	55.80	13.50	52.65	12.95	−.24	.049	10–120	38–108	38–99	1.13	1.25	1.49	1.34
Executive Function													
IC Component Score	0.17	1.09	−0.24	0.80	−.41	<.001	−1.94–4.18	−1.94–4.18	−1.82–2.50	0.82	0.94	0.86	1.45
WM Component Score	−0.40	0.97	0.56	0.74	1.10	<.001	−3.29–1.99	−3.29–1.73	−1.87–1.99	−0.44	−0.60	−0.07	0.74

Note: A = Asian; ATT = Attention Problems; B = Black or African American; BASC-2/3 = Behavior Assessment Scale for Children, 2nd or 3rd edition; H = Hispanic or Latino; HYP= Hyperactivity; IC = Inhibitory Control; IQ = WISC-V short-form IQ score, fluid reasoning index score, or one-subtest screener; MASC-2 = Multidimensional Anxiety Scale for Children, 2nd edition; MR = Multiracial; SES = Hollingshead SES total score; W = White Not Hispanic or Latino; WM = Working Memory. Executive function component scores are z-scores relative to the current sample. Cohen's d effect sizes are interpreted as small = 0.20; medium = 0.50; large = 0.80.

Table 2.

Zero-order correlations (N=339)

	Child-rated Anxiety	Parent-rated Anxiety	Teacher-rated Anxiety	WM	IC	ADHD Diagnosis	Anxiety Diagnosis	GAD SAD Diagnoses	Sex	Age	SES
Child-rated Anxiety	-----										
Parent-rated Anxiety	.13*	-----									
Teacher-rated Anxiety	.14*	.30**	-----								
WM	.03	−.03	−.15**	-----							
IC	.05	−.04	.07	.00	-----						
ADHD Diagnosis	.06	.00	.11*	−.48***	.20***	-----					
Anxiety Diagnosis	.24***	.34***	.17**	.07	−.03	.03	-----				
GAD SAD Diagnoses	.25***	.33***	.19**	.13*	−.03	.01	.82***	-----			
Sex	.01	.02	.09	−.02	−.10	−.13*	.07	.01	-----		
Age	−.06	−.03	.02	.41**	−.24**	−.17**	.00	.04	−.03	-----	
SES	.03	−.15**	−.14*	.18**	.06	.03	−.02	.01	.02	.04	-----
IQ	.14*	−.04	−.13*	.37**	.06	−.15**	.07	.12*	.07	−.05	.30**

Note. Child-rated Anxiety = MASC-2 Self-Report Total Raw Score; GAD = Generalized Anxiety Disorder; IC: inhibitory control component score; IQ = WISC-V short-form IQ score, fluid reasoning index score, or one-subtest screener; Parent-rated Anxiety = BASC-2/3 Parent Anxiety Scale T-Score; SAD = Social Anxiety Disorder; SES = Hollingshead socioeconomic status Total Score; Teacher-rated Anxiety = BASC-2/3 Teacher Anxiety Scale T-Score; WM: working memory component score.

* = $p < .05$

** = $p < .01$

*** = $p < .001$