WORKING MEMORY AND BEHAVIORAL INHIBITION IN ATTENTION-DEFICIT/HYPERACTIVITY DISORDER: A RE-EXAMINATION OF COMPETING CORE PROCESSES

By

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Abstract: Working memory and behavioral inhibition have been identified as potential underlying deficits of attention-deficit/hyperactivity disorder (ADHD) in competing models of the disorder. The stop-signal (SS) paradigm is often reified as a measure of behavioral inhibition across ADHD research. However, the choice reaction time component of the SS task likely places demands on working memory processes, consequently confounding the paradigm as a pure measure of inhibition. Therefore, the current study examined the relationship between multiple inhibition tasks to test competing models of ADHD. Forty-six boys, between 8 to 12 years old, with and without ADHD, were administered working memory (phonological and visuospatial tasks) and behavioral inhibition (go/no-go and SS) tasks. Bias-corrected bootstrapped mediation analyses indicated that working memory accounted for the relationship between group membership (ADHD and typically developing children) and both measures of inhibition. In contrast, only SS inhibition mediated the relationship between group and working memory. Overall, these findings suggest that studies that use the SS paradigm may be confounded by controlled-focused attention associated with the choice-reaction time element of the stop-signal task. Additional research that utilizes alternative measures of behavioral inhibition is needed to determine the extent of the overlap in children with the disorder.

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CHAPTER I

INTRODUCTION

Although inattention, hyperactivity, and impulsivity have traditionally been conceptualized as core features of attention-deficit/hyperactivity disorder (ADHD), more recent research suggests deficits of executive functions, such as working memory and behavioral inhibition, underlie these DSM-5-defined core symptoms of the disorder (American Psychiatric Association [APA], 2013; see Appendix A and B for a full review). Working memory is generally defined as the temporary storage, maintenance, and manipulation of information (Baddeley, 2007), while behavioral inhibition describes the process of withholding or stopping a prepotent response (Logan & Cowan, 1984). The increased interest in ADHD-related executive function deficits is reflected in competing models of ADHD that have featured working memory (Rapport, Chung, Shore, & Isaacs, 2001) and behavioral inhibition (Barkley, 1997) as central deficits of the disorder (see Appendix B for a full review). Specifically, Barkley's (1997) inhibition model of ADHD suggests behavioral disinhibition underlies deficits in working memory and other executive functions; whereas, Rapport et al.'s (2001) functional working memory model of ADHD suggests deficits in working memory are upstream of

behavioral disinhibition and other DSM-5-defined core features of the disorder (e.g., hyperactivity).

Several studies have used various methodologies to study the relationship between working memory, behavioral inhibition, and ADHD. For example, correlational studies of behavioral inhibition and working memory in children with ADHD have shown medium-magnitude associations when using the stop-signal (SS) task (Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2005; Verté, Geurts, Roeyers, Oosterlaan, & Sergeant, 2006). Findings from meta-analytic studies, however, suggest that behavioral inhibition, measured by the SS task, is downstream of basic attentional processes associated with working memory (Alderson, Rapport, & Kofler, 2007; Lijffift, Kenemans, Verbaten, & van Engeland, 2005).

More recently, Alderson, Rapport, Hudec, Sarver, & Kofler (2010) utilized bias-corrected, bootstrapped mediation analyses to directly examine competing predictions of the behavioral inhibition (Barkley, 1997) and functional working memory (Rapport et al., 2001) models of ADHD. Behavioral inhibition was measured with the SS task, while working memory was measured with analogous visuospatial and phonological working memory tasks that allowed for the creation of latent variables that reflected three components of Baddeley's (2012) multi-component model of working memory: the central executive (CE), phonological (PH) loop, and visuospatial (VS) sketchpad. Briefly, the CE is a domain-general component that is responsible for focusing attention, dividing and switching attention between storage/rehearsal systems, and blocking interference from external stimuli (Baddeley, 1996, 2007). The PH loop and VS sketchpad receive information from auditory and visual modalities, respectively, to temporarily store,

rehearse, and manipulate information (Baddeley, 2007). Collectively, Alderson and colleagues (2010) found that the CE component of working memory significantly mediated the relationship between groups (ADHD, typically developing or TD) and performance on the SS task. While inhibition significantly mediated the relationship between groups (ADHD, TD) and working memory (Alderson et al., 2010), a comparison of the magnitude of the indirect effect across models suggested that the influence of the CE on inhibition was nearly 4 times larger than that of inhibition on the CE. Consequently, these results suggested that working memory, particularly the CE, is a central deficit of ADHD that is upstream of behavioral inhibition (Alderson et al., 2010).

The first study to experimentally examine the directional relationship between behavioral inhibition and working memory in a sample of children with and without ADHD, included the SS task as the inhibition measure, an *n*-back task as the working memory measure, and a concurrent SS/*n*-back dual task (Alderson et al., in press). The authors hypothesized that performance deficits during the dual-task condition, relative to the simple inhibition and working memory conditions, would indicate overlapping or competing resource demands. Surprisingly, while both groups exhibited working memory performance declines during the dual-task condition, performance on the SS task was unaffected. In contrast to findings from previous meta-analytic (Alderson et al., 2007), experimental (Alderson, Rapport, Sarver, & Kofler, 2008), and mediation model (Alderson et al., 2010) studies, these findings appear to suggest working memory processes are either downstream or overlap demands associated with the SS task (i.e., behavioral inhibition).

Collectively, correlational studies have found medium to large associations between working memory and ADHD (Geurts et al., 2005; McNab et al., 2008; Verté et al., 2006), while meta-analytic (Alderson et al., 2007; Lijffijt et al., 2005), mediation model (Alderson et al., 2010), and experimental (Alderson et al., in press) studies have suggested that ADHD-related inhibition deficits are downstream or overlap working memory processes. However, these studies' exclusive reliance on the stop-signal task as a metric of behavioral inhibition limits interpretations about the relationship between working memory and non-SS, behavioral inhibition performance. That is, the stop-signal task places demands on higher-order cognitive processes that confound the basic, automatic inhibition processes.

Contemporary models of inhibition were derived from Gray's (1982) neurological model and early studies of reaction time (Welford, 1952). Logan and Cowan's (1984) seminal Horse-Race Model of Behavioral Inhibition suggests behavioral inhibition depends on the relative finishing times of stochastically independent go- and stop-processes that are initiated by prepotent stimuli (any stimulus that occurs before a reinforced behavior; Logan, Schachar, & Tannock, 1997; Williams, Ponesse, Schachar, Logan, & Tannock, 1999) and stop-signals, respectively. That is, inhibition occurs when the stop-process is able to overtake the go-process/prepotent response (Logan, 1982).

Early studies of inhibition relied predominantly on the go/no-go (GNG) paradigm (Donders, 1969), which requires individuals to respond to go-stimuli (e.g., letters A, B, C) via a simple reaction time task, and to withhold responses when presented with stop or no-go stimuli (e.g., the letter X; Donders, 1969; Logan, 1980). Subsequent studies have also utilized the GNG paradigm (Donders, 1969) to study automatic responses among

individuals (Logan, 1979) and stages of information processing that interact with memory demands (Logan, 1980). The SS paradigm was subsequently developed to further test Logan & Cowan's (1984) model predictions, and similar to the GNG task, requires individuals to respond to go-stimuli and withhold or discontinue responses when presented with a stop-stimulus. A relatively slow reaction time to stop-stimuli decreases the likelihood of inhibiting behavior. However, unlike the GNG task's use of a simple-reaction-time paradigm to present go-stimuli, the SS task presents go-stimuli via a choice reaction time task. Consequently, whereas the GNG task is well suited to test hypotheses of automaticity, the ability to perform an action without requiring attention for completion, and inhibition of automatic-prepotent responses (Logan, 1979), the choice reaction time component of the SS task allows for examination of behavioral inhibition within the context of more complex cognitive processes (Logan & Cowan, 1984; Verbuggen & Logan, 2009). Specifically, studies that have compared the simple and choice reaction time tasks found that choice reaction time tasks were affected by memory demands, whereas simple reaction time tasks were not (Logan, 1979, 1980). Use of the choice task also allows for examination of speed/accuracy tradeoffs and whether or not participants are attending to the go-stimuli, as well as examination of covert SSRT processes described in Logan's race model of inhibition (Verbruggen & Logan, 2009).

A practical consequence of the SS task is that associated inhibition metrics (e.g., SSRT) reflect the combined contribution of inhibition and choice-making processes, rather than a *pure* measure of inhibition. That is, go-responses (i.e., responses to the choice reaction time component) presented during a SS task place demands on decision-making processes, which in turn does not allow for automaticity of responses to develop

(Logan, 1980; Verbruggen & Logan, 2008). Even more, previous findings suggest that choice reaction time performance involves controlled-focused attention associated with the CE (Cowan, 1997; Oberauer, 2003). In contrast, go-stimuli presented in a typical GNG task place fewer demands on CE processes, since they require the use of automatic processing (Logan, 1980; Verbruggen & Logan, 2008).

To date, only one study has examined whether working memory mediates the relationship between group status (ADHD, TD) and performance on GNG tasks. Raiker, Rapport, Kofler, & Sarver (2012) examined whether working memory or SS-behavioral inhibition mediated the relationship between group status (ADHD, TD) and performance on a double-letter, high-density continuous performance test (CPT). Collectively, CE working memory processes accounted for ADHD-related impulsive behavior, and SS inhibition did not account for the ADHD-related impulsivity after controlling for CE processes. However, although CPTs are similar to GNG tasks and commonly used as measures of inhibition (Denney, Rapport, & Chung, 2005; Overtoom et al., 1998), the high-density of stop-trials (66.7%) used in Raiker et al.'s (2012) study limits generalizability to other GNG tasks that traditionally include a low-density of stop-trials (25-33%). The chosen double-letter, high-density CPT parameters are confounded by both attentional demands as well as impulsivity (Denney et al., 2005). Therefore, no study to date has compared both traditional GNG and SS inhibition paradigms in relation to working memory processes.

The Current Study

The current study is the first to examine the directional relationship between working memory and behavioral inhibition, using both SS and GNG paradigms as

measures of behavioral inhibition. As a first step, the relationship between the grouping variable and SS-behavioral inhibition performance, using working memory components as the mediator, was used in an attempt to test predictions from Rapport et al.'s (2001) functional working memory model and to replicate Alderson et al.'s (2010) previous study that found working memory mediated the relationship between group status (ADHD and TD) and inhibition. In addition, the current study tested competing predictions from Barkley's (1997) inhibition model that suggests SS-behavioral inhibition (SS task performance) would serve as a mediator of the relationship between group (ADHD, TD) and working memory performance.

Next, the competing working memory (Rapport et al., 2001) and inhibition (Barkley, 1997) model predictions were tested a second time with the GNG task performance in place of the SS task performance. The GNG task was used in this step to examine the directional relationship between non-SS inhibition (i.e., a more *pure* measure of inhibition that places fewer demands on working memory processes) and working memory. Finally, results from both approaches were compared to examine the extent to which working memory components and behavioral inhibition account for the relationship with the grouping variable, if multiple models were significant.

Collectively, working memory was predicted to be upstream of SS behavioral inhibition consistent with predictions of the functional working memory model (Rapport et al., 2001) and previous findings from Alderson et al. (2010). In addition, working memory was predicted to also be upstream of non-SS inhibition (i.e., GNG task performance), although a smaller magnitude effect was expected, relative to when the SS task was used as a measure of inhibition. This prediction was based on previous findings

that suggested the GNG task placed less demands on decision-making processes associated with the CE component of working memory (Verbruggen & Logan, 2008).

Hypotheses

Tier II: Working Memory as a Mediator between Group and Inhibition

Tier IIA: Working memory as mediator between group and SS inhibition.

The CE component of working memory was predicted to mediate the relationship between the grouping variable (TD, ADHD) and SS inhibition. This prediction was based on Alderson and colleagues' (2010) previous finding that the CE, and not PH or VS storage/rehearsal, was a significant mediator between the grouping variable and SS inhibition.

Tier IIB: Working memory as mediator between group and GNG inhibition.

The CE was predicted to mediate the relationship between the grouping variable (TD, ADHD) and GNG inhibition. This prediction was based on Raiker and colleagues' (2012) previous study that indicated the CE significantly mediated the relationship between the grouping variable and a high-density CPT task.

Tier III: Inhibition as a Mediator between Group and Working Memory

Tier IIIA: SS inhibition as mediator between group and working memory.

SS inhibition was predicted to mediate the relationship between the grouping variable and the CE component of working memory, albeit the magnitude of the indirect effect was expected to be smaller relative to when the CE was a mediator of inhibition. This prediction was based on Alderson and colleagues' (2010) previous finding that behavioral inhibition, as measured by the SS task, was a significant mediator of the

relationship between group and CE performance, but CE performance was a significantly stronger mediator of the relationship between group and inhibition.

Tier IIIB: GNG task as mediator between group and working memory. Behavioral inhibition, as measured by the GNG task, was predicted to be a significant mediator of the relationship between the grouping variable (ADHD, TD) and the CE, albeit the effect size of the indirect effect was expected to be smaller relative to when SS inhibition was used as a mediator. That is, because the choice reaction time component of the SS task is not included in the GNG task, there was less potential for shared variability between working memory and GNG inhibition (Logan, 1980; Oberauer, 2003).

CHAPTER II

METHOD

Participants

Boys with ADHD and typically developing boys (TD) between the ages of 8-12 years were recruited from flyers posted around the community in local businesses, local organizations (e.g., boy scouts), and communication with parent-teacher organizations associated with local schools. Recruitment also occurred within the Psychological Services Center (PSC), which is a university-based mental health clinic. Parent consent and child assent were obtained prior to participation. Children were grouped as typically developing (TD) or diagnosed with ADHD through a process involving several reliable and valid behavioral rating scales, cognitive and achievement assessments, and clinical interviews. Parents of all children were provided full psychoeducational reports from the children's evaluation. The Institutional Review Board (IRB) approved the study before data was collected.

Group Assignment. Inclusion in the ADHD group required: (1) a diagnosis of ADHD Combined Presentation or ADHD Predominantly Inattentive Presentation by an associate of the Center for Research of Attention and Behavior based on DSM-5 diagnostic criteria (APA, 2013), supported by information from the Kiddie-Schedule

for Affective Disorders and Schizophrenia-Present and Lifetime Version (K-SADS-PL) semi-structured clinical interview provided by the parents; (2) parent ratings at least 2 standard deviations above the mean on the Child Behavior Checklist (CBCL) or 1.5 standard deviations above the mean on the Conners-3-Parent (C3-P); and (3) teacher ratings at least 2 standard deviations above the mean on the DSM-ADHD scale on the Teacher Report Form (TRF) or 1.5 standard deviations above the mean on the Conners-3-Teacher (C3-T). All children were required to discontinue the use of medication 24 hours prior to research sessions. The ADHD group consisted of 26 children.

The TD group consisted of boys with: (1) no clinical diagnosis based on the parent and child K-SADS-PL interview and standardized rating scales (i.e., CBCL, TRF, C3-P, C3-T), and (2) normal developmental history based on information provided by the parent during a psychosocial interview. The TD group consisted of 28 children.

Children that presented with (1) gross neurological, sensory, or motor impairment, (2) psychosis, (3) history of a seizure disorder, or (4) a *Wechsler Intelligence Scale for Children-Fourth Edition* (WISC-IV) Full Scale IQ score less than 80 were excluded from the study. These factors may introduce confounds due to insufficient cognitive abilities to comprehend task instructions, or sensory or motor impairments that may limit their ability to detect or respond to stimuli. In addition, some tasks require fast, repetitive stimuli presentations that may put children at-risk for having a seizure, if they have a history of seizures.

Measures

Psychosocial and clinical interviews. Psychosocial interviews were administered to collect information about the children's prenatal, perinatal, and postnatal history, as

well as developmental, medical, educational, social, family, and cultural history. The interview provided important information to better understand the children's presenting issues, assess for impairment, screen for exclusion criteria information, and aide in differential diagnoses.

The *Kiddie-Schedule for Affective Disorders and Schizophrenia-Present and Lifetime Version* (K-SADS-PL) diagnostic semi-structured interview assessed the onset, course, frequency, duration, and severity of current and past episodes of psychopathology in children and adolescents. The K-SADS-PL was used due to its strong psychometric properties, including strong interrater reliability (98%) for present and lifetime diagnoses, and kappa coefficients ranging from good to excellent for test-retest reliability (κ = .74 - 1.00; Kaufman et al., 1997).

Behavior rating scales. Behavioral rating scales from multiple reporters assessed impairment across multiple situations (e.g., home and school), and to rule out potential other, non-ADHD diagnoses.

Child Behavior Checklist and Teacher Report Form. A parent or legal guardian completed the *Child Behavior Checklist* (CBCL; Achenbach, & Rescorla, 2001) that provides ratings of the child's emotional and behavioral functioning based on the child's age. The CBCL provides two broadband dimensions (internalizing and externalizing) and 8 narrow-band clinical domain scores (e.g., rule-breaking behavior, aggressive behavior, anxious/depressed, withdrawn/depressed, somatic complaints) that reflect emotional and behavioral functioning. The CBCL also provides clinical DSM-oriented scales that correlate with symptoms of disorders found in the *Diagnostic and Statistical Manual for Mental Disorders, 4th edition* (DSM-IV). A teacher of the participant completed the

Teacher Report Form (TRF; Achenbach, & Rescorla, 2001), which provides the same broadband dimensions, narrow-band domains, and DSM-oriented scales as the CBCL. The CBCL and TRF have strong psychometric properties, strong construct validity, and the ability to distinguish between ADHD subtypes (Ostrander, Weinfurt, Yarnold, & August, 1998; Biederman et al., 1995). The internal consistency of the scales also fell within the adequate to high range (r = .46 - .95; Sprafkin, Gadow, & Nolan, 2001).

Conners-3 Parent and Teacher Rating Scales. The *Conners 3rd Edition – Parent* (C3-P; Conners, 2008) is a 110-item measure completed by parents that assesses children's behavior during the past month and provides 6 Content Scales and 4 DSM-IV Symptom Scales. An ADHD Index Score provides a measure of how strongly a classification of ADHD is indicated, and 3 Global Index Scores summarize measures of emotional and behavioral ratings. The scale also provides validity scales that indicate whether the responses suggest a positive impression, negative impression, or inconsistent index. Teachers completed the *Conners 3rd Edition – Teacher* (C3-T; Conners, 2008), which is a 115-item measure with the same scales as the C3-P. The C3-P and C3-T have strong psychometric properties including strong internal consistency (α = .77 - .97) and test-retest reliability (r = .71 - .98; Conners, 2008).

Additional scales were used to determine the emotional functioning of participants and rule-out competing diagnoses.

Children's Depression Inventory. Children completed the *Children's Depression Inventory* (CDI; Kovacs, 2003), which is a 27-item self-report measure of depression-related symptoms in children and adolescents that occurred during the two weeks prior to administration. The CDI assesses five areas of functioning: negative mood, interpersonal

problems, ineffectiveness, anhedonia, and negative self-esteem, and has high reliability (all $\alpha > .79$; Kovacs, 2003).

Revised Children's Manifest Anxiety Scale-II. Children completed the *Revised Children's Manifest Anxiety Scale-II* (RCMAS-2; Reynolds, & Richmond, 2008), which is a 49-item self-report measure of anxiety-related symptoms for children and adolescents. The RCMAS-2 measures three areas of functioning: physiological anxiety, worry, and social anxiety, along with a measure of social desirability (defensiveness scale) and a measure of validity and biased responding (inconsistent responding index). The RCMAS-2 has high reliability for each scale ($\alpha = .75$ - $.92$; Reynolds & Richmond, 2008).

Intellectual and Academic Functioning

Wechsler Intelligence Scale for Children-Fourth Edition. All children were administered the *Wechsler Intelligence Scale for Children-Fourth Edition* (Wechsler, 2003) to assess their current level of intellectual functioning. The psychometric properties of the WISC-IV indicate strong internal consistency (all $\alpha > .79$) and test-retest reliability (all $r > .71$; Wechsler, 2003; Williams, Weiss, & Rolfhus, 2003). The WISC-IV was used to determine group inclusion (FSIQ > 80).

Kaufman Test of Educational Achievement – Second Edition. All child participants were administered the *Kaufman Test of Educational Achievement – Second Edition* (KTEA-II; Kaufman, & Kaufman, 2004) as a measure of school-based academic achievement. The psychometric properties of the KTEA-II are indicative of strong internal consistency (all $\alpha > .85$), inter-rater reliability, and validity (Kaufman, & Kaufman, 2004). Assessing participant achievement was used to inform if the children

are able to comprehend task procedures, such as meeting the minimum reading-level requirements for tasks like the PH working memory task described below.

Working Memory Experimental Tasks

Phonological (PH) working memory task. Modified from the task developed by Rapport and colleagues (2008), the PH Working Memory Task measured phonological working memory as described by Baddeley's (2007) model. Participants used a touch-screen computer (37 x 30 cm monitor screen) to complete the task that was programmed using SuperLab Pro 4.0 (Cedrus, San Pedro, CA) computer programming software. The PH task was split into four blocks of varying set sizes (3, 4, 5, and 6) that corresponded to the number of stimuli, and were presented in a counter-balanced order to control for any order effects. Each set-size block consisted of 24 consecutive trials.

Similar to the WISC-IV's Letter-Number Sequencing task (Wechsler, 2003), the PH task presented a series of shuffled numbers ranging from zero to nine (0-9) and one letter (e.g., T, G, H) for each set size (3, 4, 5, and 6). The stimuli were delivered at a comfortable volume through the computer's speakers. The stimuli were not presented twice in the same trials and a 200 ms inter-stimulus interval occurred after each number or letter is presented. Following each trial and stimulus presentation, an auditory "click" played and a green traffic light appeared on the screen prompting children to make a verbal response. The children were instructed to rearrange and say the numbers in order from least to greatest and say the letter last (see Figure 1). Following verbal responses, children would touch the screen to advance to the next trial. Children are allowed a maximum of 10,000 ms per stimulus to respond (i.e., 40,000 ms for set size 4) before the next trial starts. Verbal responses were independently recorded by two coders situated

behind a one-way mirror in order to reliably record the children's responses. Coders' responses were checked for inter-rater agreement. When there were discrepancies between coders, the responses were checked using video and audio recordings of the task.

To ensure the children understood the instructions, a block of five practice trials were given before set size 3 and again before set sizes 4, 5, or 6 (depending on the counter-balanced order). For set size 3, the letter always appeared second in each series. In set sizes 4 through 6, the letter was counter balanced between the first and last stimuli. An 80% or higher success rate was required during practice trials before beginning the experimental trials. The dependent variable for the PH task is the average number of stimuli correct per trial for each of the four stimulus set sizes (3, 4, 5, 6).

Visuospatial (VS) working memory task. The VS working memory task was based on Baddeley's (2007) model of working memory and was a modified version of the task by Rapport and colleagues (2008). Participants used a touch-screen computer to complete the task that was programmed using SuperLab Pro 4.0 (Cedrus, San Pedro, CA) computer programming software. The VS task was split into four blocks of varying set sizes (3, 4, 5, and 6) that correspond to the number of stimuli. The blocks were presented in a counter-balanced order to control for any order effects. Each set-size block consisted of 24 consecutive trials.

Three vertical columns appeared on the screen with three identical boxes (measuring 2.85 x 2.85 cm) in each column. The three columns were offset from the typical 3 x 3 grid to decrease the likelihood of PH coding of stimuli. In each trial, a series of black dots and one red dot were presented, measuring 2.22 cm in diameter. Each dot appeared one at a time for 800 ms with a 200 ms inter-stimulus interval. Following each

trial of stimuli presentation, a blank grid (see Figure 2) appeared for children to respond. Children were instructed to touch the boxes that the black dots appeared, in the same order that the black dots appeared, and to touch the box that the red dot appeared in last (see Figure 3). Children were allowed a maximum of 10,000 ms to respond to each stimulus (i.e., 10,000 ms for each dot). However, if a child responded to the first stimulus in 4,000 ms, then the child had 10,000 ms to respond where the second stimuli presented. There is a 1,000 ms inter-trial interval after the children responded or the response time was exceeded. Finally, the computer played a click sound to indicate a new trial would be presented after an additional 1,000 ms.

To ensure the children understood the instructions, a block of five practice trials were administered before set size 3 and again before set sizes 4, 5, or 6 (depending on the counter-balanced order). For set size 3, the red dot always appeared second in the stimuli presentation. In set sizes 4 through 6, the red dot was counter balanced between the first and last stimuli. An 80% or higher success rate was required during practice trials before beginning the experimental trials. The dependent variable is the average number of stimuli correct per trial for each of the four stimulus set sizes (3, 4, 5, 6).

Working memory components. Three latent variables that reflect working memory component processes (i.e., PH, VS, CE) of Baddeley's model were estimated and analyzed throughout the paper to better understand which components are contributing to the deficits associated with ADHD. To create these variables, the average number of stimuli recalled correctly for each set-size represented the children's performance from the PH and VS working memory task, which each include the storage/rehearsal and CE components (see Figure 4).

Rationale for estimating latent scores that reflect components of working memory was based on previous findings that suggested PH and VS storage/rehearsal processes are anatomically and functionally independent, while the CE is domain-general and shared by the two subsystems (i.e., PH and VS; Baddeley, 2007). The latent variable approach described by Rapport et al. (2008) was used to compute the individual variables for the independent CE, PH storage/rehearsal, and VS storage/rehearsal components. Shared variance between the PH and VS variables represented the domain-general CE, whereas unique (residual) variance represented the PH or VS storage/rehearsal components. First, the PH scores were regressed onto VS scores for each set-size to remove the shared variance associated with the CE. The four VS residual scores from each set size were averaged to represent the overall VS storage/rehearsal component of working memory. Next, the VS scores were regressed onto the PH scores at each set-size to remove the shared variance associated with the CE. The four PH residual scores from each set size were averaged to represent the overall PH storage/rehearsal component of working memory without the influence of the CE. The shared variance from both regressions were averaged to represent the CE component of working memory without influence from the VS or PH storage/rehearsal components.

Behavioral Inhibition Experimental Tasks

Stop-signal (SS) behavioral inhibition task. The SS task and administration instructions were identical to those described in Schachar, Mota, Logan, Tannock, and Klim (2000) and many previous studies that examined behavioral inhibition (Alderson, Rapport, Sarver, & Kofler, 2008; Miller, Galanter, & Pribram, 2013; Overtoom et al., 2002; Senderecka, Grabowska, Szewczyk, Gerc, & Chmylak, 2012). Go-stimuli were

displayed for 1000 ms as uppercase letters X and O that were positioned in the center of a 37 x 30 cm computer screen. The Xs and Os appeared with equal frequency throughout the experimental blocks. A wireless response pad was used wherein the left button was used to respond to the letter X, and the right button was used to respond to the letter O. Each go-stimulus was preceded by a dot (i.e., fixation point) displayed in the center of the screen for 500 ms. The fixation point served as an indicator that a go-stimulus was about to appear. A 1000 Hz auditory tone (i.e., stop-signal) was generated by the computer and delivered through speakers, which was presented randomly on 25% of the experimental trials. Stop-signal delays (SSD) – the latency between presentation of go and stop-stimuli – was initially set at 250 ms, but dynamically adjusted ± 50 ms contingent on a child's performance on the previous stop-trial. Successfully inhibited stop-trials were followed by a 50 ms increase in SSD, and unsuccessfully inhibited stop-trials were followed by a 50 ms decrease in SSD. The algorithm was designed to approximate successful inhibition on 50% of the stop-trials. All children completed two practice blocks before beginning eight consecutive experimental blocks of 32 trials (24 go-trials, 8 stop-trials). The first five blocks of the SS task were utilized in the current study to calculate dependent variables in order to reduce the effects of fatigue.

Several variables were derived from the SS task. Mean reaction time (MRT) was the average reaction time to go-stimuli. MRT variability was a measure of how much go-reaction times varied. SSD was the latency between presentation of the go-stimulus and stop-signal. Stop-signal reaction time (SSRT) was the time interval between the onset of the stop-signal and inhibition. SSRT is a covert construct that was calculated using the subtraction method (i.e., subtracting SSD from MRT; Logan et al., 1997). Consistent with

the recommendation of Alderson et al. (2007), it was determined that SSRT would only be used as a metric of inhibition if SSD was significant, otherwise SSD would be used. Finally, percent of inhibition referred to the overall percentage of the frequency of inhibiting a response. Percent of inhibition should be approximately 50% to assure that the SS task's tracking algorithm was successful, and consequently, to justify using the subtraction method to calculate SSRT (Band, van der Molen, & Logan, 2003). If percent of inhibition was not equal to 50%, SSD would be used as the measure of inhibition. MRT, MRT variability, SSD, SSRT, and percent of inhibition served as dependent variables. Figure 5 displays a visual schematic of the SS task.

Go-NoGo (GNG) behavioral inhibition task. The GNG task was developed and administered with similar parameters to the SS task. The task was created using SuperLab Pro 4.0 (Cedrus, San Pedro, CA) computer programming software. Letters were presented one at a time at the center of the screen for a duration of 1000 ms. The presented letters were 4.0 cm in height and bold, Times New Roman font. A 1000 ms inter-stimulus interval separated each letter stimulus. Children were instructed to respond as quickly as they were able each time they saw a letter or go-stimuli (e.g., A, B, C), and to not respond when a "Y" appeared (i.e., no-go stimuli). Children responded by clicking the left-mouse button. The task consisted of one practice block and three consecutive experimental blocks. Each block contained 32 trials, with 8 no-go trials. Figure 6 displays a visual schematic of the GNG task. The number of commission errors served as the dependent variable.

Procedure

All children participated in two clinical sessions where they were administered the psychosocial interview, clinical interview, and assessments of intellectual functioning and academic achievement. After the clinical assessment was completed, children participated in 3 to 4 research sessions to complete the working memory and inhibition tasks. Each clinical and research session lasted approximately three hours. Clinical sessions were scheduled for weekday mornings to minimize potential fatigue that may affect the children's performance. After obtaining consent/assent in the first clinical session, one associate completed the psychosocial interview with the parents, while a second associate administered the WISC-IV with the child. In the second session, one associate completed the K-SADS-PL interview with the parents, while a second associate administered the KTEA-II with the child. Behavioral rating scales from the parent and teacher were obtained before the first clinical session. Research sessions were scheduled on Saturday mornings and/or early afternoons to minimize the number of school absences. The VS, PH, GNG, and SS tasks were completed as a part of a larger battery of experimental tasks that were counterbalanced across research sessions. Graduate assistants administered the research tasks within the CRAB laboratory. Frequent breaks were taken after every two to three tasks to help reduce fatigue. After completing the clinical and research sessions, parents were given a copy of a comprehensive psychoeducational report and feedback to explain the results of their child's assessment.

CHAPTER III

RESULTS

Outliers

Independent and dependent variables were screened for univariate outliers prior to running analyses. The analyses examined groups (ADHD, TD) for outliers independently. Outliers were defined as values at least 3.29 standard deviations greater than or less than the mean for each group (Tabachnick & Fidell, 2001). This value corresponds with the *p*-value of .001. No outliers were identified.

Preliminary Analyses

Stop-Signal Task Validity. The validity of the SS task was analyzed to determine if the subtraction method could be applied to calculate SSRT (Band et al., 2003). First, chi square analyses were conducted for each child to determine if their percentage of inhibition, at an individual level, significantly differed from 50%. Scores that differed from 50% would lead to an incorrect calculation of SSRT (Logan et al., 1997). Therefore, scores with chi square values greater than 3.841 were excluded from the analyses. Eight children ($n_{ADHD} = 2$; $n_{TD} = 6$) were excluded based on the chi-square analyses. This resulted in a final sample of 46 children with 24 children included in the ADHD group, and 22 children included in the TD group. Next, an independent samples

t-test indicated that the ADHD group (48.0%) and TD group (52.6%) were not significantly different with respect to their percentage of inhibition, $t(44) = 1.93$, $p = .060$. Finally, a subsequent independent samples *t*-test indicated a significant between-group effect for SSD, $t(44) = 2.61$, $p = .012$, suggesting children with ADHD were less successful at inhibiting on average compared to TD children. Therefore, SSRT was calculated and used as the measurement of SS inhibition in subsequent analyses.

Sample and Demographic Information. The final sample excluding outliers from the SS task validity analyses consisted of 80% Caucasian, 9% Native American, 4% Biracial, 4% Hispanic, and 2% Asian children. Demographic data were examined for between-group differences using independent samples *t*-tests (age, FSIQ, SES) and Pearson's chi squared tests (ethnicity) to determine if covariate analyses were warranted. Groups did not significantly differ in age, $t(44) = -.04$, $p = .968$, ethnicity, $\chi^2(4) = 1.94$, $p = .746$, Hollingshead SES ratings[1], $t(41) = 1.43$, $p = .160$, and FSIQ, $t(44) = 1.44$, $p = .156$. In addition, between-group differences were examined using independent samples *t*-tests for ratings of emotional and behavioral functioning (CBCL, TRF, C3-P, C3-T, CDI, and RCMAS). Children with ADHD had significantly higher ratings on all emotional and behavioral measures compared to TD children. Of the 24 children included in the ADHD group, 15 met for a comorbid diagnoses: specific learning disorder ($n = 3$), oppositional defiant disorder ($n = 8$), conduct disorder ($n = 1$), specific phobia ($n = 1$), enuresis ($n = 4$), encopresis ($n = 2$), and persistent depressive disorder ($n = 1$). This comorbidity is consisted with previous epidemiological studies (Busch et al., 2002; Wilens, Biederman, & Spencer, 2002). Of the 22 children included in the TD group, 6 had clinical elevations on teacher or parent ratings; however, clinical interview with the

[1] SES data was not available for three participants due to insufficient information needed for calculation.

parents indicated that these elevations reflected isolated events and/or were not interfering with the children's functioning. Table 1 displays a summary of the sample's demographic and rating scale information.

Mediation Analyses

All mediation analyses were completed using Shrout and Bolger's (2002) bootstrapping procedure. This resampling with replacement procedure used the original data to derive 1000 samples as recommended by Shrout & Bolger (2002). Significant indirect effects ($p < .05$) were indicated if the 95% confidence intervals did not include zero. Bias-corrected bootstrap mediation analyses are recommended for sample sizes as small as 20 participants to reduce the likelihood of Type II error (Efron & Tibshirani, 1993; Preacher & Hayes, 2004). Our final sample included 46 children.

Tier I: Intercorrelations

Prior to running mediation analyses, intercorrelations using Pearson's *r* statistics were calculated between the grouping variable (ADHD, TD), PH storage/rehearsal, VS storage/rehearsal, CE, SS inhibition performance (SSRT), and GNG inhibition performance (commission errors). The grouping variable (dummy coded as TD = 0, ADHD = 1) significantly correlated with SS inhibition, $r = .29$, $p = .026$, and CE, $r = -.394$, $p = .003$, with children with ADHD being associated with worse SS inhibition and CE performance. SS inhibition was significantly associated with VS storage/rehearsal and CE, and GNG inhibition was significantly associated with PH storage/rehearsal, VS storage/rehearsal, and CE components of working memory (see Table 2). All planned mediation models were tested since all *a* and *b* paths were non-zero, suggesting the

product of *ab* is not zero and could potentially still influence the indirect effect (Preacher & Hayes, 2002, 2008).

Tier II: Working Memory as a Mediator between Group (ADHD and TD) and Inhibition

Tier IIA: Working memory as mediator between group and SS inhibition. Similar to Alderson et al. (2010), mediation analyses examined whether working memory mediates the relationship between the grouping variable (TD, ADHD) and SS inhibition (see Figure 7). CE was found to have an overall significant indirect effect on the relationship between grouping variable and SS inhibition, $\beta = .20$, $SE = .08$, 95% confidence interval = .06 - .39, $\kappa^2 = .20$, 95% confidence interval = .06 - .39. Neither PH storage/rehearsal ($\beta = .02$, $SE = .04$, 95% confidence interval = -.05 - .14, $\kappa^2 = .02$, 95% confidence interval = .00 - .10) nor VS storage/rehearsal ($\beta = .04$, $SE = .06$, 95% confidence interval = -.03 - .21, $\kappa^2 = .04$, 95% confidence interval = .00 - .21) were significant mediators of the relationship between the grouping variable and SS inhibition.

Tier IIB: Working memory as mediator between group and GNG inhibition. Similar to Tier IIA, Tier IIB consisted of a mediation analysis to determine if working memory mediated the relationship between the grouping variable (TD, ADHD) and GNG inhibition (see Figure 8). CE was found to have an overall significant indirect effect on the relationship between group and GNG inhibition, $\beta = .10$, $SE = .07$, 95% confidence interval = .004 - .31, $\kappa^2 = .10$, 95% confidence interval = .01 - .26. Neither PH storage/rehearsal, $\beta = -.06$, $SE = .06$, 95% confidence interval = -.25 - .01, $\kappa^2 = .07$, 95% confidence interval = .00 - .23, nor VS storage/rehearsal, $\beta = .07$, $SE = .07$, 95%

confidence interval = -.03 - .28, κ^2 = .07, 95% confidence interval = .01 - .25, were significant mediators.

Tier III: Inhibition as a Mediator between Group and Working Memory

Tier IIIA: SS inhibition as mediator between group and working memory. Tier IIIA examined whether SS inhibition mediated the relationship between the grouping variable and working memory performance. There was a significant indirect effect for SS inhibition as the mediator of the relationship between the grouping variable and CE (β = -.13, *SE* = .13, 95% confidence interval = -.56 - -.04, κ^2 = .14, 95% confidence interval = .02 - .30), and the grouping variable and VS storage/rehearsal (β = -.09, *SE* = .06, 95% confidence interval = -.27 - -.001, κ^2 = .09, 95% confidence interval = .006 - .25). However, SS inhibition did not significantly mediate the relationship between the grouping variable and PH storage/rehearsal (β = -.02, *SE* = .05, 95% confidence interval = -.13 - .07, κ^2 = .02, 95% confidence interval = .00 - .10). Figure 9 displays a visual representation of the analyses in Tier IIIA.

Tier IIIB. GNG inhibition as mediator between group and working memory. Tier IIIB analyzed whether GNG inhibition processes accounted for the relationship between the grouping variable and working memory performance (see Figure 10). GNG inhibition did not significantly mediation the relationship between grouping variable and the CE (β = -.02, *SE* = .04, 95% confidence interval = -.14 - .04, κ^2 = .03, 95% confidence interval = .001 - .11), PH storage/rehearsal (β = .03, *SE* = .05, 95% confidence interval = -.04 - .17, κ^2 = .03, 95% confidence interval = .002 - .13), or VS storage/rehearsal (β = -.05, *SE* = .07, 95% confidence interval = -.18 - .09, κ^2 = .05, 95% confidence interval = .002 - .15).

CHAPTER IV

DISCUSSION

The current study is the first to examine the directional relationship between the proposed core deficits of ADHD, including working memory (Rapport et al., 2001) and behavioral inhibition (Barkley, 1997), using both SS and GNG paradigms as measures of behavioral inhibition. Previous studies have largely relied on the SS inhibition as the measure of inhibition; however, the SS task may place greater demands on controlled-focus attention that is associated with the CE component of working memory, compared to the GNG task (Cowan, 1997; Oberauer, 2003; Verbruggen & Logan, 2008). To that end, the association between the SS task and CE may account for equivocal findings across previous studies that appear to provide support both working memory and inhibition models of ADHD (e.g., Alderson et al., 2010; Clark et al., 2007; Brocki, Nyberg, Thorell, & Bohlin, 2007; Kerns, McInerney, & Wilde, 2001; Raiker et al., 2012).

Intercorrelations between the grouping variable, working memory components, and behavioral inhibition performance were examined as a first step. Overall, findings indicated that ADHD was associated with slower SSRT, consistent with previous meta-analytic findings (Alderson et al., 2007; Lijffijt et al., 2005; Oosterlaan, Logan, &

Sergeant, 1998). In addition, the ADHD group was associated with a shorter SSD. Collectively, these findings indicate that children in the ADHD group, relative to the TD group, were associated with deficits of SS inhibition.

Surprisingly, SS inhibition was not significantly associated with GNG performance, suggesting the SS and GNG tasks may measure different constructs of inhibition. This finding differs from previous neuroimaging studies that found overlap between GNG and SS task performance, as both require decisions to respond or not respond (e.g., Rubia et al., 2001), but emphasizes the distinct neuroanatomical regions, neuropharmacological, and neuroimaging differences found while completing SS or GNG inhibition tasks (Eagle, Bari, & Robbins, 2008; Rubia et al., 2001). The differences may also be due to greater demands on decision making processes (Verbruggen & Logan, 2009; Eagle et al., 2008) and multiple input modalities (i.e., auditory and visual processing; Hirose et al., 2012) associated with the SS task, compared to the GNG task. Moreover, a factor analytic study found differences between the commonly used measurements (e.g., reaction time and error rates) that load onto separate factors, such that reaction time loaded onto a cognitive efficiency/speed faction and error rates loaded onto a inhibition control factor (Vuontela et al., 2013).

Not surprisingly, ADHD had large associations with deficient central executive processes (d = .85[2]), small associations with VS storage/rehearsal processes (d = .28), and medium associations with PH storage/rehearsal processes (d = .45). These associations were relatively smaller than associations reported in Alderson et al. (2010) and Raiker et al. (2012), which may be due to the current study's inclusion of multiple ADHD presentations (e.g., combined and inattentive type). The VS storage/rehearsal effect size

[2] Effect size converted into Cohen's d for comparisons across studies.

was also relatively smaller than those found in previous meta-analytic findings (Kasper, Alderson, & Hudec, 2012; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005), while the PH storage/rehearsal findings were relatively smaller compared to Kasper et al. (2012) and Willcutt et al. (2005) reviews, but similar to Martinussen et al. (2005). However, the overall pattern of findings is consistent with previous research, such that the CE had larger associations with group membership, compared to VS and PH storage/rehearsal components.

Similar to findings from previous studies (i.e., CE and VS storage/rehearsal; Dalen, Sonuga-Barke, Hall, & Remington, 2004; Raiker et al., 2012; Solanto et al., 2001; Stevens, Quittner, Zuckerman, & Moore, 2002), SS and GNG inhibition had small to large magnitude associations with CE and VS storage/rehearsal components of working memory. PH storage/rehearsal performance, however, was only associated with errors made during the GNG task. While both SS and GNG paradigms presented text-based go-stimuli, these findings appear to suggest that the children did not rely on orthographic conversion processes (Baddeley, 2007), but rather, simply evaluated the text and made decisions based on the visual presentation.

Bootstrapped mediation analyses were subsequently used to determine if CE processes accounted for the relationship between group and behavioral inhibition (i.e., SS and GNG performance). Consistent with the findings of Alderson et al. (2010), CE processes, but not VS or PH storage/rehearsal processes, significantly mediated the relationship between ADHD and SS performance deficits. Moreover, the CE also mediated the relationship between group membership and GNG performance, consistent with the findings from Raiker et al. (2012). Comparison of the magnitude of the indirect

effects across mediation models revealed that, while both indirect effects were medium magnitude (Cohen, 1988; Preacher & Kelley, 2011), the magnitude of the indirect effect of group on SS performance through the CE (κ^2 = .20) was twice as large compared to when it was a mediator of group on GNG performance (κ^2 = .10). Moreover, SS inhibition was found to mediate the relationship between the grouping variable and VS storage/rehearsal (κ^2 = .09) as well as CE (κ^2 = .14). The effect sizes suggest small to medium effects, respectively, with the CE having greater influence on SS inhibition compared to VS storage/rehearsal. Additionally, GNG inhibition was not found to mediate the grouping variable and CE, VS storage/rehearsal, or PH storage/rehearsal. Collectively, these findings are consistent with our a priori predictions and previous studies (Verbruggen & Logan, 2008, 2009) that suggest the SS task, relative to the GNG task, places greater demands on controlled-focused attention associated with the CE (Cowan, 1997, 1988).

Finally, to further understand ADHD-related deficits in relation to competing models of the disorder, comparisons were made across tiers. The indirect effect of group on SS inhibition through the CE (κ^2 = .20) was relatively larger than the indirect effect of group on CE (κ^2 = .14) through SS inhibition. While the general pattern of the effects are similar to those reported by Alderson et al. (2010), the current study had a smaller difference between effect sizes. That is, Alderson and colleagues found the magnitude of the indirect effect of group on SS inhibition through the CE to be approximately four times the size of the indirect effect of group on CE through SS inhibition. Smaller effect sizes were also found for the indirect effect of group on GNG through CE (κ^2 = .10) and the indirect effect group on VS storage/rehearsal through SS inhibition (κ^2 = .09).

Moreover, the effect for the indirect effect of group and SS inhibition through CE was double the indirect effect of group and GNG through CE. Collectively, these findings further suggest that performance on the SS task, relative to the GNG task, is more vulnerable to working memory and other cognitive processes as the task requires decision-making processes associated with the choice-reaction time component, and does not allow for the development of automaticity (Cowan, 1997; Oberauer, 2003; Verbruggen & Logan, 2008). Furthermore, these results may give insight to the mixed findings supporting both competing models of ADHD. That is, the findings supporting Barkley's (1997) inhibition model of ADHD may be confounded by the use of the SS task, which is susceptible to working memory processes. Additionally, the larger effect sizes when CE is a mediator, and the lack of significant findings when GNG was the mediator of group and working memory, suggests behavioral inhibition processes are likely downstream of working memory, consistent with predictions of Rapport et al.'s (2008) functional working memory model of ADHD.

The current study replicated previous findings from Alderson et al. (2010) and further examined competing predictions between working memory and behavioral inhibition deficits utilizing GNG paradigms among children with ADHD and typically developing peers. Nevertheless, some limitations should be noted about the current study. The sample included boys with ADHD who also met diagnostic criteria for other disorders. Inclusion of comorbid disorders may confound the current study's estimates of ADHD-related executive function deficits, since previous findings suggest that executive function deficits are associated with other psychopathology (e.g., Pennington & Ozonoff, 1996). However, this comorbidity is expected based on past epidemiological findings

(Busch et al., 2002; Wilens et al., 2002), suggesting the sample is likely generalizable to typical children with ADHD. Additionally, the current study included a relatively small sample size, which may increase the risk for Type II errors. However, bias-corrected bootstrapping procedures were utilized to decrease the likelihood of Type II errors (Shrout & Bolger, 2002), and the study is expected to have sufficient power (Efron & Tibshirani, 1993; Preacher & Hayes, 2004). Nevertheless, future studies are recommended to replicate these findings with larger sample sizes and samples including girls in order to promote the generalization of the current study.

The current study was the first to test competing models of ADHD with using both SS and GNG paradigms. Findings from this study suggest performance on the SS task, compared to the GNG task, may be vulnerable to working memory processes as the CE had the largest indirect effect between group and inhibition. These findings also suggest that working memory processes overlap or are upstream of behavioral inhibition deficits among children with ADHD. Future research is needed to determine the nature of this overlap with the use of inhibition and working memory tasks that segregate the different components of each executive function to determine where the deficits occur among children with the disorder. Identifying the core deficits of ADHD can aid in developing treatments and interventions that underlie the disorder rather than treating the symptoms. Additionally, these findings can aid teachers and medical professionals in understanding how to adjust the environment for children with ADHD to help them succeed.

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TABLES AND FIGURES

Table 1. Sample and demographic variables

	ADHD (n = 24) *M (SD)*	TD (n = 22) *M (SD)*	χ^2	*t*
Ethnic Composition			1.94	
Age	9.98 (1.53)	9.96 (1.54)		-.04
FSIQ	99.79 (9.74)	104.45 (12.15)		1.44
SES[a]	46.14 (8.75)	50.48 (11.04)		1.43
CBCL DSM-ADHD	66.54 (7.42)	52.91 (4.67)		-7.52***
TRF DSM-ADHD	64.13 (7.54)	52.59 (4.50)		-6.36***
C3-P ADHD-I	75.63 (8.86)	51.45 (10.09)		-8.65***
C3-P ADHD-HI	72.46 (13.86)	49.56 (9.28)		-6.51***
C3-T ADHD-I	72.38 (9.53)	48.18 (8.42)		-9.09***
C3-T ADHD-HI	65.67 (16.47)	49.82 (13.40)		-3.56**
RCMAS-2	49.88 (10.14)	39.05 (7.24)		-4.07***
CDI	49.50 (7.42)	41.82 (6.77)		-3.66**

Note. ADHD = Attention-Deficit/Hyperactivity Disorder; TD = Typically Developing; M = Mean; SD = Standard Deviation; FSIQ = Full Scale Intelligence Quotient; CBCL = Child Behavior Checklist; TRF = Teacher Report Form; C3-P = Conners-3 Parent Rating Scale; C3-T = Conners-3 Teacher Rating Scale; DSM-ADHD = Attention-Deficit/Hyperactivity Problems Scale; ADHD-I = DSM ADHD Inattention Subscale; ADHD-HI = DSM ADHD Hyperactive/Impulsive Subscale.
*$p < .05$, **$p < .01$, ***$p < .001$
[a] SES data was not available for 3 participants due to insufficient information.

Table 2. Intercorrelations.

	1	2	3	4	5	6
1. Group	1.00					
2. SSRT	.29*	1.00				
3. GNG	.11	-.16	1.00			
4. PH S/R	-.22	-.14	.25*	1.00		
5. VS S/R	-.14	-.33*	-.48**	-.60***	1.00	
6. CE	-.39**	-.54***	-.26*	.44**	.45**	1.00

Note. One-tailed Pearson's *r* correlations are presented in Table 2. Group = Grouping variable; SSRT = Stop-signal reaction time; GNG = Go/no-go commission errors; PH S/R = Phonological storage/rehearsal; VS S/R = Visuospatial storage/rehearsal; CE = Central executive.
*$p < .05$, **$p < .01$, ***$p < .001$

Table 3. Summary of Experimental Tasks

	ADHD (*n* = 24) *M (SD)*	TD (*n* = 22) *M (SD)*
SSRT	352.13 (68.74)	318.73 (39.96)
SSD	248.54 (34.85)	273.98 (30.94)
SS MRT	600.68 (67.79)	592.71 (52.66)
GNG	4.71 (2.82)	3.95 (4.13)
PH S/R	-.15 (.65)	.12 (.54)
VS S/R	-.07 (.56)	.09 (.58)
CE	2.74 (.51)	3.15 (.45)

Note. ADHD = Attention-deficit/hyperactivity disorder group; TD = Typically developing group; M = Mean; SD = Standard deviation; SSRT = Stop-signal reaction time; SSD = Stop-signal delay; SS MRT = Stop-signal mean reaction time; GNG = Go/no-go commission errors; PH S/R = Phonological storage/rehearsal; VS S/R = Visuospatial storage/rehearsal; CE = Central Executive.

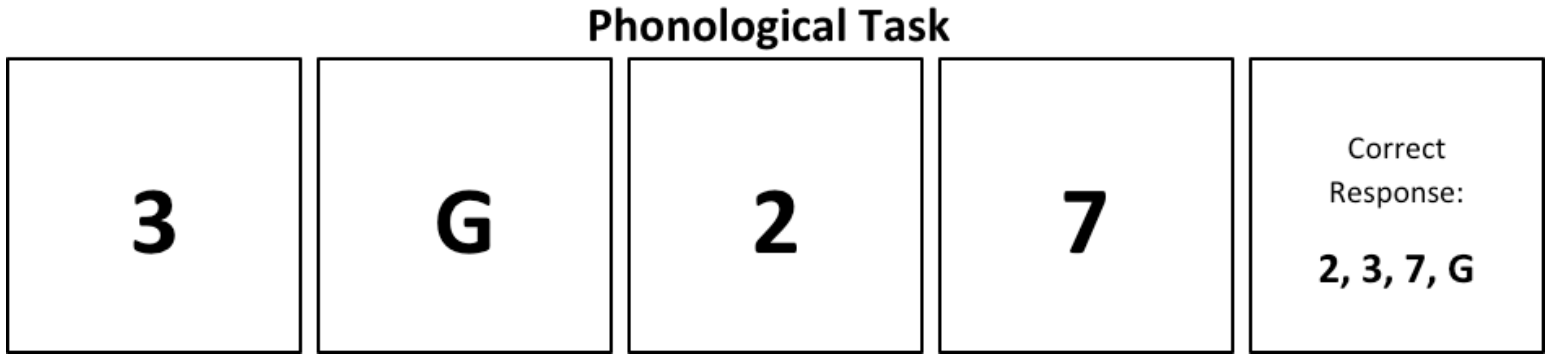

Figure 1. Visual representation of the phonological working memory task.

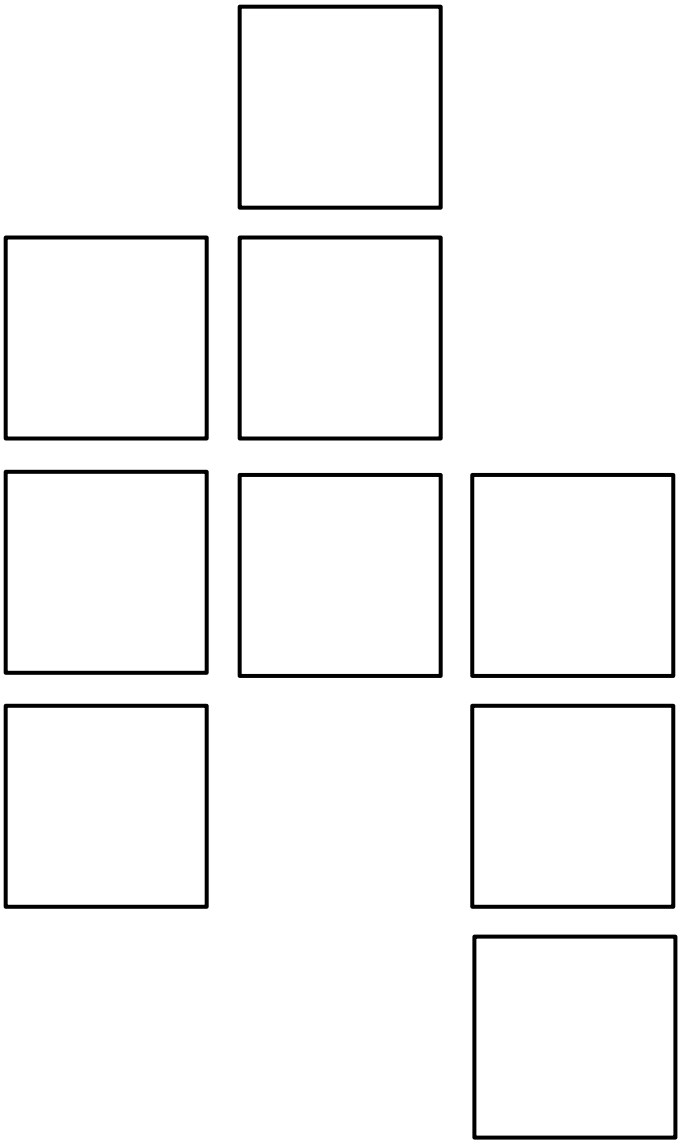

Figure 2. Representation of the blank grid presented during the visuospatial working memory task.

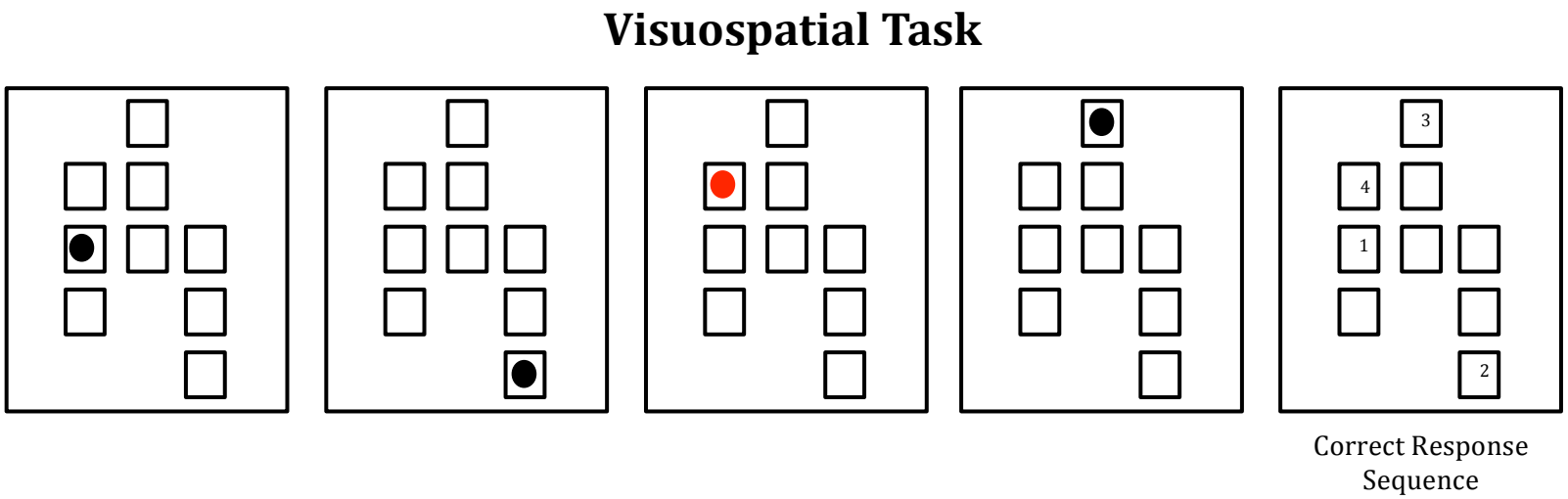

Figure 3. Visual representation of the visuospatial working memory task.

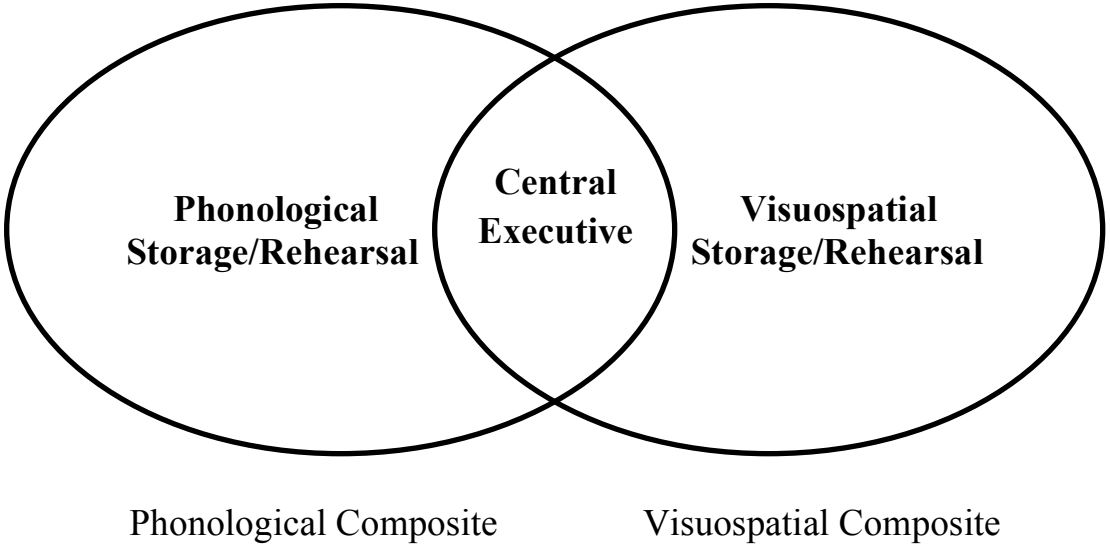

Figure 4. Diagram of Baddeley's working memory components.

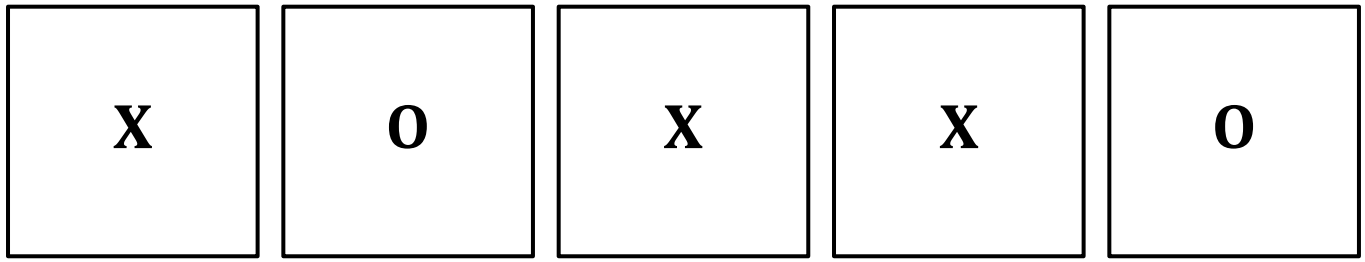

Figure 5. Visual representation of the SS Task. "Stop-Signal" refers to the auditory tone heard while completing the task that indicates a button should not be pressed.

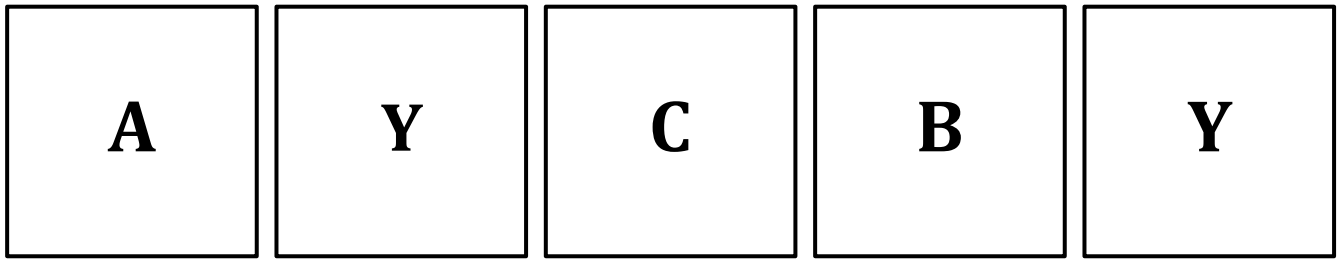

Figure 6. Visual representation of the GNG task. Go responses indicate a response (i.e., a button press) is correct. No go responses indicate that no response is correct.

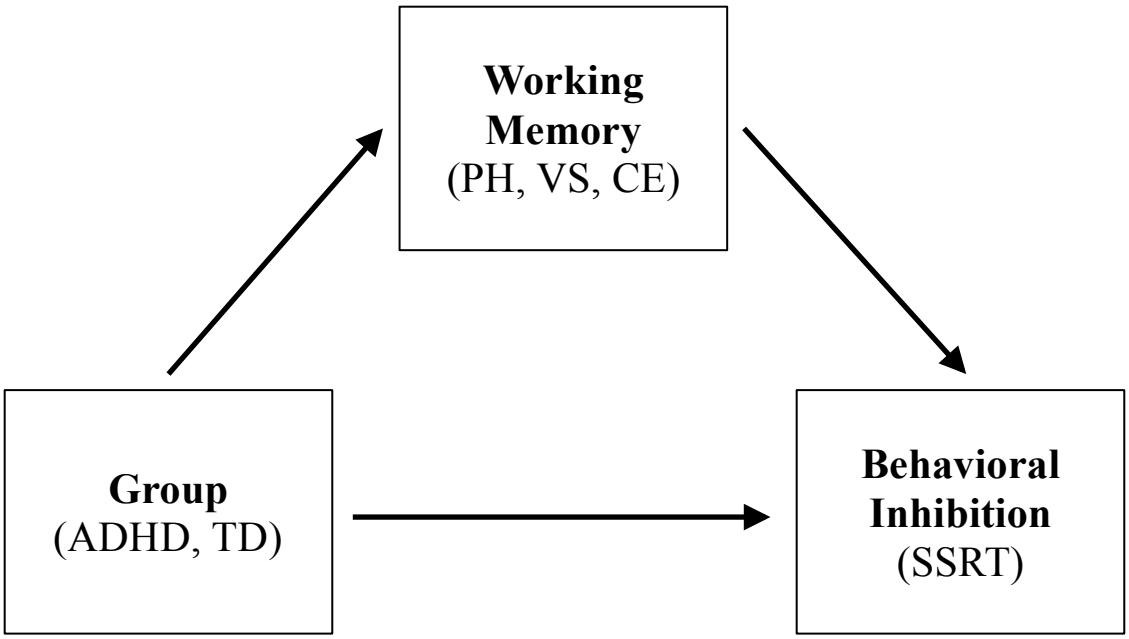

Figure 7. Visual schematic of the mediation model for Tier IIA. ADHD = Attention-deficit/hyperactivity disorder; TD = Typically developing; PH = Phonological storage/rehearsal; VS = Visuospatial storage/rehearsal; CE = Central executive; SSRT = Stop-signal reaction time.

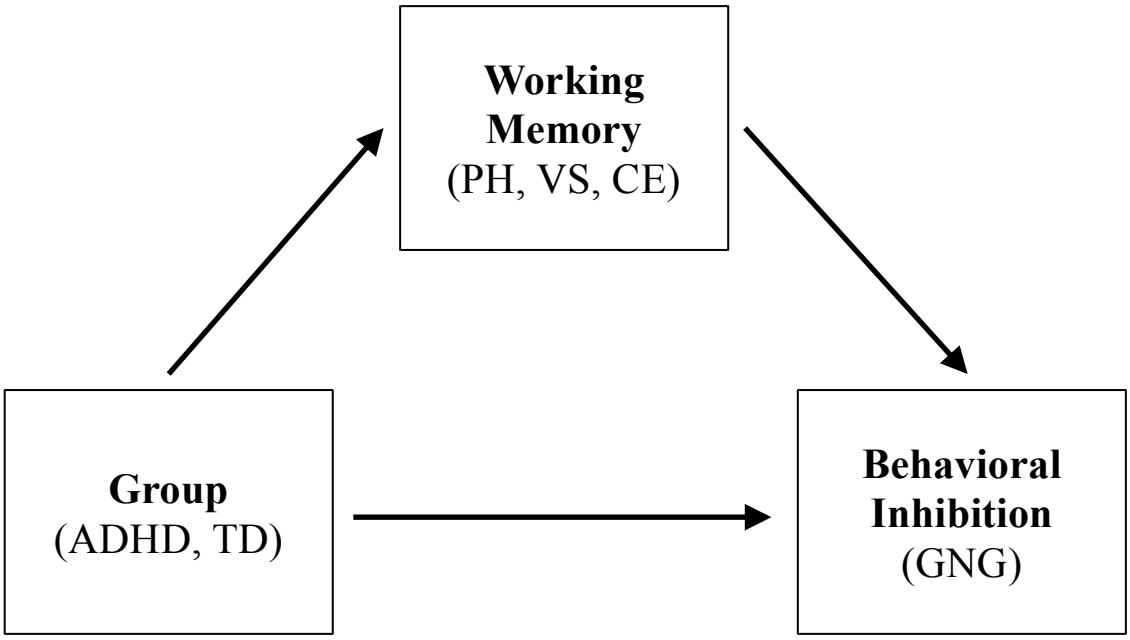

Figure 8. Visual schematic of the mediation model for Tier IIB. ADHD = Attention-deficit/hyperactivity disorder; TD = Typically developing; PH = Phonological storage/rehearsal; VS = Visuospatial storage/rehearsal; CE = Central executive; GNG = Go/no-go.

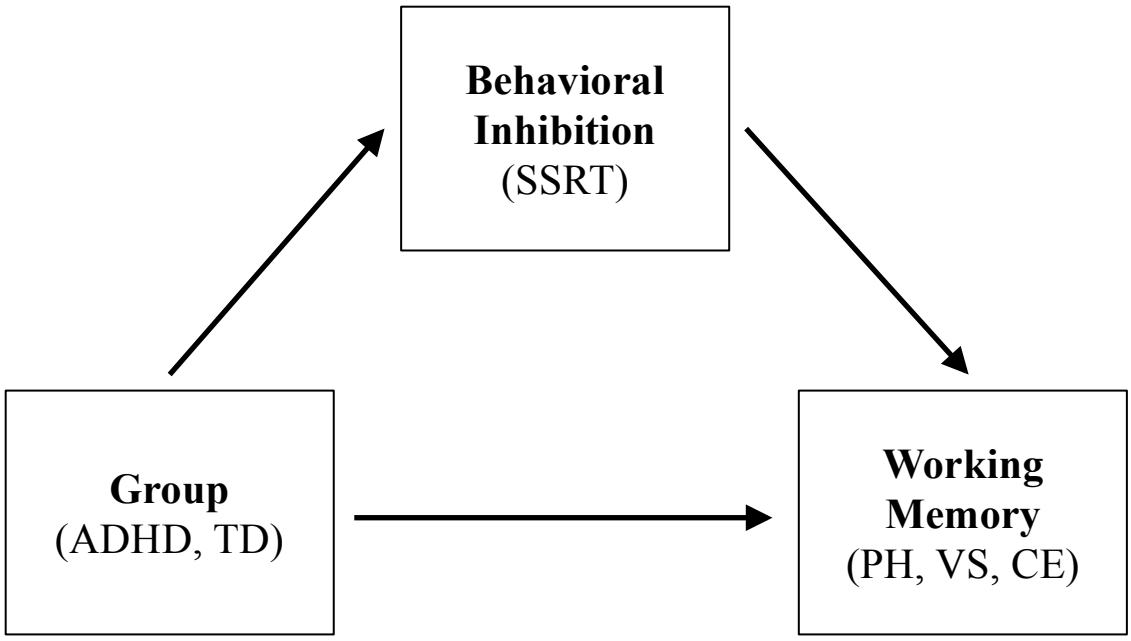

Figure 9. Visual schematic of the mediation model for Tier IIIA. ADHD = Attention-deficit/hyperactivity disorder; TD = Typically developing; PH = Phonological storage/rehearsal; VS = Visuospatial storage/rehearsal; CE = Central executive; SSRT = Stop-signal reaction time.

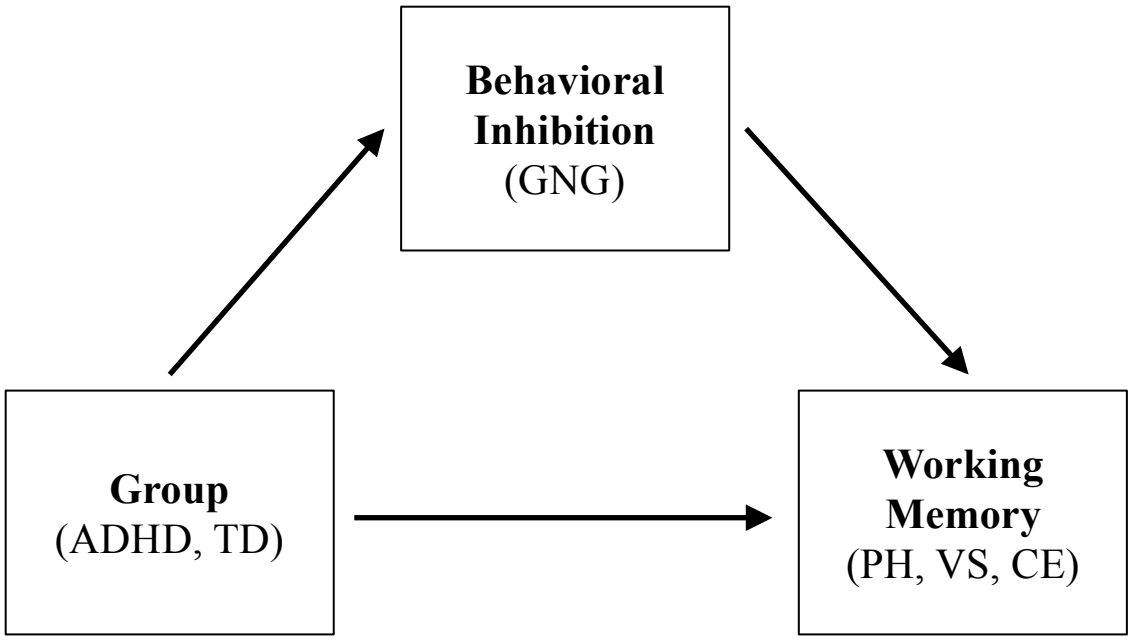

Figure 10. Visual schematic of the mediation model for Tier IIIB. ADHD = Attention-deficit/hyperactivity disorder; TD = Typically developing; PH = Phonological storage/rehearsal; VS = Visuospatial storage/rehearsal; CE = Central executive; GNG = Go/no-go.

APPENDIX A

OVERVIEW OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

Attention-deficit/hyperactivity disorder (ADHD) is a complex, highly heritable disorder (Barkley, 2006; Biederman, 2004) that is characterized by symptoms of inattention (Douglas, 1972; Burgess et al., 2010), impulsivity (Douglas, 1972; Raiker et al., 2012), and hyperactivity (Frick & Lahey, 1991; Rapport et al., 2009). Symptoms of ADHD appear before the age of 12 years and typically persist throughout the lifespan (APA, 2013; Okie, 2006). Factor analytic (DuPaul et al., 1998), structural equation model (Larsson, Lichtenstein, & Larsson, 2006), and meta-analytic (Willcutt et al., 2012) studies reveal two factors, inattention and hyperactivity-impulsivity, which are reflected in the DSM-5 diagnostic presentations (APA, 2013).

Currently, the prevalence of ADHD in the United States falls between 4% and 9% of children (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007), and about 3% to 4.4% of adults (Faraone & Biederman, 2005; Kessler et al., 2006; Polanczyk & Jensen, 2008). The worldwide prevalence rate is approximately 5.23% for children and adolescents (Polanczyk & Jensen, 2008). In school-aged children, ADHD-Predominantly Inattentive Presentation (3.9-4.5%) is the most prevalent subtype followed by the

ADHD-Predominantly Hyperactive/Impulsive Presentation (1.7-1.9%) and ADHD-Combined Presentation (1.9%; Gaub & Carlson, 1997; Graetz, Sawyer, Hazell, Arney, & Baghurst, 2001); however, ADHD-Combined Presentation is the most commonly referred subtype in the clinical setting (Gaub & Carlson, 1997). Early theories believed that ADHD was a childhood disorder that remitted upon maturation into adulthood, such that less than 1% of adults over the age of 20 years old met criteria for ADHD (Hill & Schoener, 1996; Polanczyk & Jensen, 2008). However, early estimates of adulthood prevalence rates were biased by a lack of epidemiological studies for certain populations (i.e., adults with ADHD; Polanczyk & Jensen, 2008) and methodological procedures that varied across samples of different age groups (i.e., self-report in adults versus parent-report in children; Barkley, Fischer, Smallish, & Fletcher, 2002; Polanczyk & Jensen, 2008).

Gender differences have emerged across the different presentations of ADHD. Girls with ADHD typically show fewer externalizing and hyperactive symptoms and greater inattentive and internalizing symptoms, compared to boys with the disorder (Gershon, 2002). Several studies suggest that teachers perceive boys with ADHD to have greater symptom severity than girls, leading teachers to have an increased sensitivity to identify boys with ADHD (Gershon, 2002; Reid et al., 2000). These studies suggest there is a difference between inattentive and hyperactivity-impulsivity symptoms and the identification of ADHD across genders, such that girls who display more inattentive symptoms are not as likely to be diagnosed as boys with hyperactivity-impulsivity symptom presentations (Gershon, 2002). Further, evidence for gender differences in ADHD-related executive function deficits has emerged, albeit findings across studies are

relatively equivocal (Berlin, Bohlin, & Rydell, 2004; Houghton et al., 1999; Seidman et al., 2010; Thorell & Wåhlstedt, 2006). For example, Wodka and colleagues (2008) found that girls with the predominantly hyperactive/impulsive presentation exhibited relatively greater executive function deficits compared to boys or girls with the predominantly inattentive presentation, whereas boys with the predominantly inattentive presentation exhibited relatively greater executive function deficits compared to girls or boys with the predominantly hyperactive/impulsive presentation. Therefore, ADHD presentations that were not common for the children's gender showed relatively greater executive function deficits. In contrast, studies have also shown general executive function deficits in children with ADHD with no differences in executive functions between genders (Thorell & Wåhlstedt, 2006).

Historical Perspective of ADHD

Attention-deficit/hyperactivity disorder developed from diagnostic criteria dating back to the 1800s (Lange, Reichl, Lange, Tucha, & Tucha, 2010; Rafalovich, 2001). In the early 1900s, doctors distinguished brain dysfunction that differed from individuals with mental retardation (Rafalovich, 2001). For example, inattention, hyperactivity, scholastic underachievement, and behavior problems were observed in children with encephalitis (Ebaugh, 1923), severe head injury, and other diseases (Laufer, Denhoff, & Solomons, 1956). These advancements lead to the diagnostic moniker of *hyperkinetic impulse disorder* that described the symptom of restlessness across multiple settings, with similar criteria to the hyperactivity component of the current ADHD diagnostic criteria (Lange et al., 2010). The DSM-II (American Psychiatric Association, 1968) diagnostic criteria for *hyperkinetic reaction of childhood* deemphasized brain dysfunction as the

characteristic symptom, leading to the recognition and development of the term hyperactivity as a primary symptom (Barkley, 2006; Lange et al., 2010).

Conceptualization of the disorder shifted again during the 1970s from an emphasis on hyperactivity to one of attention, when findings of sustained attention and impulse control deficits began to emerge (Barkley, 2006; Douglas, 1972; Lange et al., 2010). These findings were reflected in the DSM-III (American Psychiatric Association, 1980), which updated the diagnostic criteria for *attention deficit disorder (ADD)*. The criteria for ADD specified whether the diagnosis was with or without hyperactivity and contained three categories of symptoms: inattention, impulsivity, and hyperactivity (Lange et al., 2010). Transitioning to the DSM-IV's (American Psychiatric Association [APA], 1994) *attention-deficit/hyperactivity disorder (ADHD)* reframed diagnostic criteria into inattentive, hyperactivity-impulsivity, and combined subtypes in order to reflect current empirical research on the subtypes and increase the diagnostic criteria's validity and reliability (Biederman et al., 1997; Lahey et al., 1994). The restructuring improved diagnostic sensitivity for girls who presented with symptoms of the inattentive subtype, preschoolers who typically met criteria for the hyperactivity-impulsivity subtype, and adults due to the inclusion of occupational-related symptom descriptions (APA, 1994; Lahey et al., 1994; Lange et al., 2010). Although the behavioral symptoms did not change from the DSM-IV (APA, 1994) to the DSM-5 (APA, 2013), the nomenclature of "subtypes" in the DSM-IV was changed to "presentations." The DSM-5 (APA, 2013) also broadened the application of ADHD to better incorporate diagnoses through adulthood by expanding the criteria across multiple settings and shifting the symptom onset from 7 years to 12 years.

ADHD and Comorbid Disorders

Several psychological disorders, such as depression and anxiety, and medical conditions, such as allergies and asthma, are highly comorbid with ADHD (Biederman, Newcorn, & Sprich, 1991; Jensen, Martin, & Cantwell, 1997). Two highly comorbid disorders with ADHD are oppositional defiant disorder (ODD) and conduct disorder (CD), which may lead to higher impulsivity symptoms in boys (Newcorn et al., 2001). Comorbidity of ADHD and CD is also associated with an earlier age of onset and a decreased likelihood of remission, whereas the comorbidity of ADHD and anxiety is associated with less severe behavioral symptoms and fewer impulsivity symptoms (Jensen et al., 1997). Diagnoses of ADHD and CD are also thought to have high global burden ratings, due to having an earlier childhood onset compared to other diseases (Erskine et al., 2014). Children with ADHD and a comorbid disorder have different presentations between genders, such that boys with ADHD and ODD experience greater symptom severity, and girls with ADHD and anxiety exhibit fewer impulsivity symptoms (Newcorn et al., 2001). However, executive function deficits commonly associated with ADHD do not appear to be moderated by the presence of other behavioral disorders, such as ODD (Thorell & Wåhlstedt, 2006).

Impairments Associated with ADHD

The effects of ADHD span beyond symptoms of inattention and hyperactivity/impulsivity to influence impairments in cognitive (Hervey, Epstein, & Currey, 2004), behavioral (Barkley, 2006; DuPaul, McGoey, Eckert, & VanBrakle, 2001), academic (Daley & Birchwood, 2009), and social areas (Frederick & Olmi, 1994; Kofler et al., 2011). For example, children with ADHD exhibit behavioral impairments

(Barkley, 2006; DuPaul et al., 2001) as evidenced by higher behavioral ratings on parent and teacher behavior rating scales, and a higher frequency of disruptive behaviors in the classroom setting (DuPaul et al., 2001). Children with ADHD, relative to non-affected children, are also more likely to have an increased risk for physical injury, particularly with respect to frequency and severity (Barkley, 2006).

Children with ADHD frequently show impairment with interpersonal relationships (Frederick & Olmi, 1994; Kofler et al., 2011), such as peer rejection due to rule violations, poor anger regulation, and a lack of group cooperation (Guevremont & Dumas, 1994). Although children with ADHD typically have knowledge of how to behave appropriately in social situations, they are more likely to engage in negative social behaviors such as bragging and interrupting others (Cervantes et al., 2013). Approximately 52% of children with ADHD are rejected by peers and are not as socially preferred compared to same-aged children without ADHD (Cervantes et al., 2013; Hoza et al., 2005).

Academic deficits associated with ADHD are detected from the early stages of preschool (Mariani & Barkley, 1997) and persist through university education (Daley & Birchwood, 2009). Preschoolers diagnosed with ADHD exhibit greater impairments with their scholastic readiness, including deficits in fundamental mathematics or reading skills (Mariani & Barkley, 1997). In addition, children with ADHD have a higher likelihood of being diagnosed with a learning disorder (Barkley, 2002). College students with ADHD diagnoses and documented disabilities often report needing more time to complete assignments and exams, and having to work harder to achieve good grades relative to their typically developing peers (Lewandowski et al., 2008).

Finally, children with ADHD experience impairments across a broad range of executive functions (Barkley, 1997; Pennington, Groisser, & Welsch, 1993), including behavioral inhibition (Barkley 1997; Willcutt et al., 2001) and working memory (Rapport et al., 2008), and the impairments remain after accounting for intelligence (Halperin, Trampush, Miller, Marks, & Newcorn, 2008; Willcutt et al., 2005) and socioeconomic status (SES; Halperin et al., 2008). ADHD-related executive function deficits increase the risk for academic underachievement, learning disorders, and grade retentions, even after controlling for comorbid disorders (Daley and Birchwood, 2009), medication treatment, age of onset, or number of ADHD symptoms (Biederman et al., 2004). The large-magnitude executive function (e.g., response inhibition, vigilance, spatial working memory, and planning) deficits associated with ADHD (Willcutt et al., 2005) appear to reduce affected children's abilities to remember to turn in homework, avoid losing items, and organize academic schedules (Langberg, Dvorsky, & Evans, 2013). Further, early symptoms of ADHD and associated executive function deficits are predictors of behavioral problems in later years (Wåhlstedt, Thorell, & Bohlin, 2008), while working memory deficits appear to be particularly related to ADHD-related social problems (Kofler et al., 2011). Overall, children with ADHD exhibit executive function deficits that contribute to impairments across several domains.

APPENDIX B

MODELS OF ADHD

Exploration of endophenotypic (Castellanos & Tannock, 2002), neurobiological (Curatolo, D'Agati, & Moavero, 2010), genetic (Kuntsi et al., 2014), and animal models (Bari & Robbins, 2013) have all aided in the conceptualization of ADHD. Researchers have also aimed to explain the symptoms and impairments exhibited by children with ADHD through the development of models that suggest deficits of executive functions serve as a central feature of the disorder. For example, extant models describe causal pathways that identify the core deficits of ADHD as behavioral inhibition (Barkley, 1997; Sergeant, 2000) or working memory (Rapport et al., 2001). These ADHD models and their conceptualization of ADHD-related executive function deficits are reviewed below.

Cognitive-Energetic Model of ADHD

Sergeant's (2000) cognitive-energetic model (CEM) of ADHD suggests that the disorder manifests from cognitive deficits that are also dependent on the energetic state of the child. CEM describes the disorder and variations in behavior in terms of efficient information processing through three interdependent levels: computational mechanisms of attention, energetic stages, and management mechanisms (i.e., executive functions; Sergeant, 2000). The computational mechanisms of attention include encoding,

searching, decisions, and motor organization, while the energetic states are associated with effort, arousal, and activation (Sergeant, 2005). *Effort* is the energy used to complete a task. The management mechanisms include the processes of planning, monitoring, detecting errors, and correcting errors (Sergeant, 2005), and are related to executive functioning (Sergeant, 2000). The CEM of ADHD suggests information processing is influenced by executive functions via effort and other energetic states; thus, the CEM model suggests that studies examining only executive functions deficits may be oversimplified (Sergeant, 2000). The parameters surrounding the CEM, however, do not provide sufficient information to develop or test hypotheses about the relationship between ADHD-related executive function deficits and the energetic states.

Neurodevelopmental Model of ADHD

Halperin and Schulz's (2006) Neurodevelopmental Model of ADHD integrates neuropsychological and neuroimaging research to propose that symptoms of ADHD are related to the underdevelopment of neural mechanisms. The proposed model accounts for the developmental trajectories of the prefrontal cortex and executive functions, and consequently suggests that the main cause of ADHD is likely due to multiple, interrelated systems and not due to lesions or damage in the prefrontal cortex. In addition, factors such as compensatory strategies and neural plasticity are hypothesized to account for individual differences in symptom reduction throughout the lifespan, and correct identification of these factors may help to target new intervention strategies such as cognitive enrichment training (Halperin & Schulz, 2006; Halperin, Bédard, & Curchak-Lichtin, 2012). However, the neurological focus of Halperin and Schulz's model is weakened by the inability to infer a cause due to potential confounds (i.e., environmental

factors) of individual behaviors and lack of consistent findings in existing research (e.g., Zametkin et al., 1990, 1993).

Inhibition Models of ADHD

Brief history and overview of the race horse model. Contemporary models of inhibition were derived from Gray's (1982) neurological model and early studies of reaction time (Welford, 1952). Logan and Cowan's (1984) seminal Horse-Race Model of Behavioral Inhibition suggests behavioral inhibition depends on the relative finishing times of stochastically independent go- and stop-processes that are initiated by prepotent stimuli (any stimulus that occurs before a reinforced behavior; Logan et al., 1997; Williams et al., 1999) and stop-signals, respectively. That is, inhibition occurs when the stop-process is able to overtake the go-process/prepotent response (Logan & Cowan, 1984). A relatively slow reaction time to stop-stimuli (i.e., stop-signal reaction time: SSRT) decreases the likelihood of inhibiting behavior.

Logan (1982) first examined the horse race model by observing skilled typists and how quickly they were able to stop typing after hearing an auditory tone, or stop-signal. Subsequent studies utilized the go/no-go (GNG) paradigm (Donders, 1969) to study the internalized reaction to a stop-signal (Logan, Cowan, & Davis, 1984), automatic responses among individuals (Logan, 1979), and stages of information processing that interact with memory demands (Logan, 1980). The GNG paradigm requires individuals to respond to go-stimuli (e.g., letters A, B, C) via a simple reaction time task and to withhold responses when presented with stop or no-go stimuli (e.g., the letter X; Donders, 1969; Logan, 1980).

The SS paradigm was subsequently developed to further test Logan & Cowan's (1984) model predictions, and similar to the GNG task, required individuals to respond to go-stimuli and withhold or discontinue responses when presented with a stop-stimulus. Unlike the GNG task's use of a simple-reaction-time paradigm to present go-stimuli, the SS task presents go-stimuli via a choice reaction time task. Consequently, whereas the GNG task is well suited to test hypotheses of automaticity, the ability to perform an action without requiring attention for completion, and inhibition of automatic-prepotent responses (Logan, 1979), the choice reaction time component of the SS task allows for examination of behavioral inhibition within the context of more complex cognitive processes (Logan & Cowan, 1984; Verbruggen & Logan, 2009). Specifically, studies that have compared the simple and choice reaction time tasks found that choice reaction time tasks were affected by memory demands, whereas simple reaction time tasks were not (Logan, 1979, 1980). Use of the choice task also allows for examination of speed/accuracy tradeoffs and whether or not participants are attending to the go-stimuli. The SS task is currently the predominant inhibition paradigm, likely due to its ability to examine covert SSRT processes described in Logan's race model of inhibition (Verbruggen & Logan, 2009).

Behavioral inhibition and ADHD. Schachar and Logan's (1990) seminal study was the first to examine behavioral inhibition processes in children with ADHD and found that affected children exhibited slower reaction times and fewer instances of inhibitory success compared to typically developing peers. Subsequent experimental (Logan et al., 1997) and meta-analytic (Oosterlaan et al., 1998) provided evidence of reliable, medium-magnitude ADHD-related response inhibitions, slower reaction times,

and slower inhibitory processes. These findings ultimately led to the inclusion of behavioral inhibition in models of ADHD.

Barkley's (1997) inhibition model of ADHD proposed that behavioral inhibition deficits are the core feature of the disorder. His model builds from Gray's (1982, 1991) model that described an underactive behavioral inhibition system, and Quay's (1997) later description of behavioral activation and behavioral inhibition systems. Barkley (1997) characterizes behavioral inhibition as a multidimensional executive function that is responsible for (1) inhibiting a prepotent response, (2) delaying an immediate response for deciding whether to respond or inhibit, and (3) preventing interfering information from affecting the response process (interference control). Behavioral inhibition processes are hypothesized to be activated during tasks associated with delay of gratification, goal-directed responses, or problem solving (Barkley, 1997, 2006). This model proposes that behavioral inhibition processes directly and indirectly influence motor activity through the executive functions of working memory, emotion regulation, internalization of speech, and reconstitution. Consequently, this model's framework suggests that inhibition is upstream of other executive functions. However, it is difficult to determine whether the inhibition differences found are due to symptoms relating to ADHD or to the high comorbidities with other behavioral disorders (e.g., ODD; Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001). In addition, several studies have raised questions about the hierarchy of executive functions included in this model, and more specifically, have suggested that ADHD-related disinhibition is downstream of working memory deficits (Alderson et al., 2007; Alderson et al., 2010, in press; Kofler et al., 2014).

The dual pathway model (Sonuga-Barke, 2003), another inhibition-based model of ADHD, suggests that deficits of ADHD arise from impairment of inhibition and the reward circuit. The expression of inattentive, overactive, and impulsive behaviors exhibited by children with ADHD is a result of having a negative emotional response to delays, known as *delay aversion* (Sonuga-Barke, 2002, 2003, 2005). This model suggests that ADHD is a deficit of vigilance and motivation, where children are hypervigilant to their surroundings and scan for escape cues during delays (Sonuga-Barke, 2003). A relatively recent update of the model (i.e., triple pathway model of ADHD) includes the three domains of inhibition, temporal processing (i.e., timing), and delay processing (Sonuga-Barke, Bitsaku, & Thompson, 2010). The temporal processing, inhibition, and delay pathways share similar neural correlates, and temporal processing was found to be dissociable from inhibition and delay deficits (Sonuga-Barke, 2005; Sonuga-Barke et al., 2010). Further, the model proposes that executive dysfunction (e.g., working memory) and delay aversion are two separate neuropsychological components of ADHD (Sonuga-Barke, Dalen, & Remington, 2003). Although this model is rather detailed, Sonuga-Barke's proposed model implies that ADHD-related working memory and inhibition deficits are relatively ubiquitous and dependent on moment-to-moment variability in motivation. A growing body of findings (e.g., Alderson, Rapport, Kasper, Sarver, & Kofler, 2012; Hudec, Alderson, Kasper, & Patros, 2013; Rapport et al., 2009) raises questions about the validity of this position.

Brief Overview of Working Memory Models

Functional working memory model of ADHD. In contrast to inhibition models (Barkley, 1997; Sonuga-Barke et al., 2010) that suggest inhibition deficits are upstream

of other ADHD-related executive function deficits, Rapport and colleagues' (2001) functional working memory model suggests that working memory is the central deficit of ADHD and is upstream of behavioral inhibition. Working memory is currently defined as a limited capacity system that allows for temporary storage and active manipulation of mental information (Baddeley, 2007; Cowan, 1997).

Cognitive models of working memory. In the 1890's, William James suggested memory storage consisted of two features, primary (short-term storage in conscious awareness) and secondary (long-term memory that must be retrieved) memory. This hypothesis diverged from existing views that memory storage was composed of a single system (Baddeley, 2007). However, James's theory was not pursued until Hebb (1949) later differentiated memory into short-term and long-term memory. This led to additional research towards creating a theory of multiple storage systems (Peterson & Peterson, 1959; Shallice & Warrington, 1970). Working memory was later derived from the construct "short-term store" due to the development of a system that integrated the ability to manipulate information stored in short-term memory (Atkinson & Shiffrin, 1968; Baddeley, 2007; Miller et al., 1960). Several contemporary researchers have since developed models for the structure of working memory.

Cowan's embedded processes model (1988) suggests stimuli enter a brief sensory storage system that activates a section of long-term memory (activated memory). A "spotlight" or portion of activated memory (i.e. focus of attention) within an individual's conscious awareness is working memory (Cowan, 1988, 1999). More recently, Unsworth and Engle's (2007) dual-component model suggests working memory consists of two components: (1) one's ability to maintain accessible information in limited-capacity

memory storage (primary memory), and (2) the retrieval of information from stored information that is relevant to the context (secondary memory; Unsworth & Engle, 2007). Similar to Cowan's model, Unsworth and Engle (2007) view primary/working memory as a unitary component, regardless of stimulus modality (e.g., phonological or visuospatial).

In contrast, Baddeley and Hitch's (1974) seminal multi-component model introduced three components of working memory: the central executive (CE), phonological (PH) loop, and visuospatial (VS) sketchpad. A fourth component, the episodic buffer, was added in an updated model (Baddeley, 2000). The CE is a domain-general component that is responsible for focusing attention, dividing and switching attention between storage/rehearsal systems, and blocking interference from external stimuli (Baddeley, 1996, 2007). The CE is associated with the prefrontal cortex and allocates information to either the VS or PH working memory components (D'Esposito et al., 1995).

The PH buffer/loop involves the temporary storage and rehearsal of auditory information (Baddeley & Hitch, 1974; Baddeley, 2007). The PH buffer refers to the storage component, whereas the PH loop refers to a separate rehearsal component. The PH buffer relies on the PH loop to refresh the stored information, which previous research suggests has a key role in the acquisition and comprehension of language, vocabulary, and syntax (Baddeley, 2007). For example, patients with short-term PH memory deficits are able to process simple sentences, but are not able to understand longer, complex sentences, suggesting that PH working memory is required to comprehend the sentence's meaning (Vallar & Baddeley, 1987). In addition, Gathercole

& Baddeley (1990) demonstrated that the PH loop assisted children in vocabulary acquisition, as non-word repetition predicted memory a year later. Together, these studies provide support for separate storage and rehearsal subcomponents of the PH working memory system (Vallar & Baddeley, 1984).

The VS sketchpad receives information from multiple domains (i.e., touch, vision, etc.) for the integration of visual and spatial information (Baddeley 2007; Baddeley & Hitch 1974). The VS sketchpad assists with tasks that require temporary storage, recall, and/or manipulation of objects, and the object's location in three-dimensional space (Steenhuis & Goodale, 1988; Vogel, Woodman, & Luck, 2001). Recent research has also suggested that the VS sketchpad is involved with visually manipulating information for mathematic skills (Holmes, Adams, & Hamilton, 2008). Overall, similar to the PH buffer/loop, research has found VS memory to have a separate storage and rehearsal components that decode visual objects and their locations (Baddeley, 2007).

The fourth component of working memory, the episodic buffer, was added to the model to account for communication between working memory and long-term memory, and between the PH loop and VS sketchpad (Baddeley, 2007). This addition to the model enables the buffer to temporarily store bound information from multiple modalities, long-term memory, and other sources (Baddeley, 2000), such as combining visual features (e.g., color, shape, position) with a physical object (Allen, Baddeley, & Hitch, 2006; Wheeler & Treisman, 2002). When the episodic buffer interacts with the CE, the CE can allocate where attention is directed and what information is stored in long-term memory (Baddeley, 2000, 2007).

The current study will focus on Baddeley's multiple-component model described below, due to the model's support from experimental and neuropsychological research (Baddeley, 2000) and its prevalence within ADHD research (Kasper et al., 2012).

Working memory and ADHD. The functional working memory model of ADHD is based on Baddeley's (2003) multi-component working memory model and suggests that impaired working memory underlies phenotypic ADHD features such as poor behavioral organization and stimulation seeking (Rapport et al., 2001, 2008). These working memory deficits directly impact performance on cognitive and behavioral tasks that lead to the deficits seen in the academic, social, and other settings seen in individuals with ADHD (Rapport et al., 2001). Unlike Barkley's (1997) behavioral inhibition model, working memory is seen as upstream of behavioral inhibition, meaning working memory deficits account for deficits seen in behavioral inhibition (Alderson et al., 2010, in press; Rapport et al., 2008). In addition, behavioral disinhibition and other models of ADHD infer that the problems lie within the individual, rather than as an impairment also influenced by environmental factors (Rapport et al., 2001). In contrast, Rapport and colleagues' (2009) model suggests an interaction between a working memory deficit and the environment accounts for the impairments associated with ADHD. The functional working memory model also suggests treatments should target underlying core deficits of the disorder (e.g., working memory deficits), rather than peripheral symptoms such as off-task behavior, impulsivity, or excessive motor activity (Arnsten, 2006; Rapport et al., 2001).

Evidence of PH storage/rehearsal, VS storage/rehearsal, and CE deficits has been demonstrated in children with ADHD, relative to typically developing peers (Rapport et

al., 2001, 2008). More recently, findings from Bolden, Rapport, Raiker, Sarver, & Kofler (2012) suggest children with ADHD experience deficits in both the PH storage and PH rehearsal subsystems. Moreover, meta-analytic studies have shown large magnitude ADHD-related deficits in PH (Kasper et al., 2012; Willcutt et al., 2005) and VS (Martinussen et al., 2005) working memory performance. Most recently, findings from Alderson et al. (2014) indicate that children with ADHD do not benefit from information presented in dual modalities (i.e., visually and aurally) to the same extent as their non-affected peers, implicating deficits of the episodic buffer component of working memory.

Recent research has also supported the working memory model's hypothesis that deficits of working memory serve as a core feature of ADHD that underlies phenotypic features described by the DSM-5 (Alderson et al., 2010, 2014, in press; Rapport et al., 2008). For example, increased demands on working memory appear to be functionally related to ADHD-related symptoms of hyperactivity (Hudec et al., 2013; Rapport et al., 2009), and inattentive behavior (Kofler, Rapport, Bolden, Sarver, & Raiker, 2010). Working memory deficits have also been shown to mediate ADHD-related social deficits (Kofler et al., 2011). Collectively, working memory deficits appear to influence several domains of problems typically seen in children with ADHD.

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