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Working Memory and Intraindividual Variability as Neurocognitive Indicators in ADHD: Examining Competing Model Predictions

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Objective: The current study examined competing predictions of the default mode, cognitive neuroenergetic, and functional working memory models of attention-deficit/hyperactivity disorder (ADHD) regarding the relation between neurocognitive impairments in working memory and intraindividual variability. ***Method:*** Twenty-two children with ADHD and 15 typically developing children were assessed on multiple tasks measuring intraindividual reaction time (RT) variability (ex-Gaussian: tau, sigma) and central executive (CE) working memory. Latent factor scores based on multiple, counterbalanced tasks were created for each construct of interest (CE, tau, sigma) to reflect reliable variance associated with each construct and remove task-specific, test–retest, and random error. ***Results:*** Bias-corrected, bootstrapped mediation analyses revealed that CE working memory accounted for 88% to 100% of ADHD-related RT variability across models, and between-group differences in RT variability were no longer detectable after accounting for the mediating role of CE working memory. In contrast, RT variability accounted for 10% to 29% of between-group differences in CE working memory, and large magnitude CE working memory deficits remained after accounting for this partial mediation. Statistical comparison of effect size estimates across models suggests directionality of effects, such that the mediation effects of CE working memory on RT variability were significantly greater than the mediation effects of RT variability on CE working memory. ***Conclusions:*** The current findings question the role of RT variability as a primary neurocognitive indicator in ADHD and suggest that ADHD-related RT variability may be secondary to underlying deficits in CE working memory.

Keywords: attention-deficit/hyperactivity disorder, working memory, variability, reaction time, ex-Gaussian

Children with attention-deficit/hyperactivity disorder (ADHD) are characterized frequently as consistently inconsistent, and this intraindividual variability is considered by many to reflect a stable, ubiquitous, and etiologically important characteristic of the disorder (Klein, Wendling, Huettner, Ruder, & Peper, 2006; Tamm et al., 2012). Intraindividual variability refers to moment-to-moment (within-subject) fluctuations in behavior and task performance, occurrs over a period of seconds or milliseconds rather than hours or days (Castellanos et al., 2005), and is indexed typically by reaction time (RT) dispersion during laboratory tasks. RT variability refers to inconsistency in an individual's speed of responding and has been argued to reflect primarily a subset of abnormally slow responses during laboratory tasks (Klein et al., 2006; Leth-Steensen, Elbaz, & Douglas, 2000). A recent meta-analysis of 319 ADHD RT variability studies concluded that children with ADHD displayed large magnitude increased intraindividual variability relative to typically developing groups across a wide range of tasks (Kofler et al., 2013). These large magnitude increases in RT variability were observed to a similar degree across all three ADHD subtypes, were similar in magnitude regardless of task demands, were typically as large as or larger than those reported for most other neurocognitive indices, and were decreased or normalized by stimulant medication (Klein et al., 2006; Kofler et al., 2013; Tamm et al., 2012; Willcutt, Sonuga-Barke, Nigg, & Sergeant, 2008). It is not surprising, then, that intraindividual

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variability has been proposed as an underlying trait responsible for ADHD (Russell et al., 2006) or a potential intermediate phenotype of the disorder (Buzy, Medoff, & Schweitzer, 2009; Castellanos et al., 2005).

The centrality of ADHD-related intraindividual variability is highlighted by its prominence in modern accounts of ADHD, and empirical research is needed to examine the extent to which RT variability reflects a primary neurocognitive indicator and potential novel intervention target as opposed to a secondary/tertiary outcome or correlate of other impaired/underdeveloped neurocognitive processes (Kofler et al., 2013; Tamm et al., 2012). Two models—the *default mode network* and *cognitive neuroenergetic* models—describe ADHD-related performance variability as a primary neurocognitive indicator attributable to specific, underlying neurobiological dysfunction. The default mode network model hypothesizes that disruptions in cortico-striato-thalamo-cortical neuroanatomical circuitry contribute to behavioral and cognitive differences in ADHD (Castellanos et al., 2005; Sonuga-Barke & Castellanos, 2007). In this model, children with ADHD experience predictable oscillations in their default mode (resting state) neural networks that interfere with task-oriented neural processing, producing periodic lapses of attention that interfere with working memory processing and impair academic and social functioning (Castellanos et al., 2005). Increased ADHD-related intraindividual variability, as indexed by RT variability on laboratory tasks, is viewed as the primary neurocognitive indicator of these periodic fluctuations. As such, the lapses of attention indexed by RT variability are predicted to "mediate causal pathways between putative originating causes and behavioral traits or symptoms" (Castellanos et al., 2005, p. 1417). That is, the model's neurocognitive-level predictions specify that RT variability should partially or fully explain (mediate) the relation between ADHD diagnostic status and behavioral traits and symptoms such as impaired performance on executive functioning tasks (see Figure 1).

Similarly, the cognitive neuroenergetic model posits that ADHD is caused at the neurobiological level by insufficient astrocyte functioning and resultant impairments in lactate production needed for consistent, rapid neuronal firing (Russell et al., 2006). In this model, increased performance (RT) variability is viewed as the primary neurocognitive outcome of this hypothesized impairment in consistent fuel source availability. Furthermore, RT variability is hypothesized to be largely or fully responsible for ADHD-related impaired performance on higher order neurocognitive tasks measuring working memory, response inhibition, and other executive functions (Russell et al., 2006).

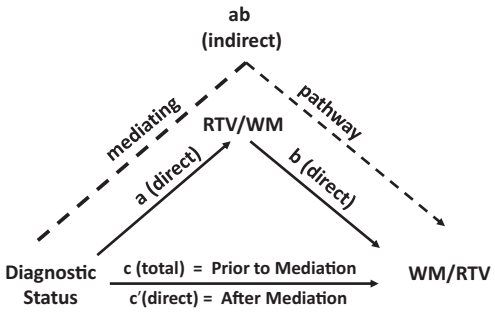

Figure 1. Visual schematic reflecting the total, direct, and indirect pathways of a bootstrapped mediation analysis before and after accounting for the mediating variable. The RTV/WM and WM/RTV variables reflect initially the prediction of CE WM by RTV (i.e., tau, sigma) in Tier II, and subsequently the prediction of RTV by CE WM in Tier III. CE = central executive; RTV = reaction time variability; WM = working memory.

Collectively, both models describe ADHD-related RT variability as a primary neurocognitive indicator of core, neurobiological deficits in ADHD. In addition, at the neurocognitive level of explanation, both models hypothesize that ADHD-related performance variability underlies impaired performance on neurocognitive tasks—that is, RT variability should mediate the relation between ADHD diagnostic status and impaired performance on tasks measuring working memory and other aspects of neurocognitive functioning. This hypothesized mediation, shown in Figure 1, was tested directly in the current study to examine neurocognitive-level predictions of the default mode and cognitive neuroenergetic models of ADHD.

In contrast, the *functional working memory* model hypothesizes that increased ADHD-related performance variability reflects a secondary outcome of underlying impairments in working memory (Rapport et al., 2008; Rapport, Chung, Shore, & Isaacs, 2001). At the neurobiological level, this model proposes that ADHD results from delayed cortical maturation (Shaw et al., 2007) and associated chronic cortical underarousal (for reviews, see Dickstein, Bannon, Castellanos, & Milham, 2006; Barry, Clarke, McCarthy, Selikowitz, & Rushby, 2005; Rapport et al., 2008), particularly in prefrontal, temporal, parietal, and interconnected regions implicated in working memory processing, storage, and rehearsal. ADHD behavioral symptoms and functional impairments are viewed as byproducts of the interaction between environmental demands and the impaired working memory functioning that results from these neurobiological vulnerabilities (Rapport et al., 2001, 2008). Working memory refers to a limited capacity system responsible for the temporary storage, rehearsal, and manipulation of internally held information for use in guiding behavior. Extensive evidence reveals two distinct storage/rehearsal subsystems, phonological and visuospatial, that are overseen by a domain-general attentional controller termed the *central executive* (CE; Baddeley, 2007). The central executive is distinct from the more general executive functioning construct and is a supervisory attentional controller responsible for processing, reordering, and updating information held in short-term memory (STM; Wager & Smith, 2003).

At the neurocognitive level of explanation, the working memory model postulates that increased ADHD-related RT variability is attributable to deficits in the CE working memory system. These large magnitude CE deficits are hypothesized to result in increased motor activity (Rapport et al., 2009), mind wandering (Kane et al., 2007), visual inattention (Burgess et al., 2010; Kofler, Rapport, Bolden, Sarver, & Raiker, 2010), and impulsive responding (Raiker, Rapport, Kofler, & Sarver, 2012) that temporarily disrupt task performance and result in an intermittent subset of abnormally slow responses. This subset of abnormally slow responses, reflected in the ex-Gaussian tau statistic described below, positively skews their RT distributions and results in increased RT variability. That is, the working memory model predicts that ADHD-related working memory deficits underlie the inconsistent response patterns that are characteristic of the ADHD

phenotype, and thus mediate the relation between ADHD diagnostic status and increased intraindividual variability on laboratory tasks (see Figure 1).

Previous studies examining working memory and intraindividual RT variability suggest a robust relation between the two constructs. For example, Schmiedek, Oberauer, Wilhelm, Süß, and Wittmann (2007) used ex-Gaussian estimation of RT variability and found an overall strong latent correlation ($r = -.72$) with established measures of working memory. Additional evidence indicates that working memory is correlated highly with RT performance and variability on a wide variety of neurocognitive tasks, including simple and choice RT tasks (Schmiedek et al., 2007), as well as more complex tasks including fluid reasoning, *n*-back updating (Schmiedek, Hildebrandt, Lövdén, Wilhelm, & Lindenberger, 2009), continuous performance, visual match-to-sample (Raiker et al., 2012), behavioral inhibition (Alderson, Rapport, Hudec, Sarver, & Kofler, 2010; Garon, Bryson, & Smith, 2008), and short term memory (STM) paradigms (Engle, Tuholski, Laughlin, & Conway, 1999; Swanson & Kim, 2007). With regard to intraindividual variability and working memory in ADHD, Buzy and colleagues (2009) reported increased ADHD-related RT variability as a function of increasing cognitive load, a finding consistent with previous studies assessing intraindividual variability during recognition-based memory tasks (Klein et al., 2006; Piek et al., 2004). The central findings of these studies imply significant shared variance between measures of working memory and intraindividual RT variability; however, the interrelation among these neurocognitive constructs could not be determined based on the correlational and experimental methods employed.

The current study is the first to test opposing, neurocognitive-level predictions stemming from the default mode network (Sonuga-Barke & Castellanos, 2007), cognitive neuroenergetic (Russell et al., 2006), and functional working memory (Rapport et al., 2008) models of ADHD by examining the model-implied interrelation between working memory and intraindividual (RT) variability. For the variability models (Castellanos et al., 2005; Russell et al., 2006), increased ADHD-related RT variability was hypothesized to partially or fully mediate the relation between diagnostic status (ADHD, typically developing) and working memory performance to the extent that performance on working memory tasks is impaired by performance variability/lapses of attention, as indexed by RT variability (see Figure 1). Support for variability model predictions would include finding that RT variability serves as a stronger mediator of the ADHD–working memory relation than working memory serves as a mediator of the ADHD–RT variability relation. For the working memory model, CE working memory processes were expected to fully mediate ADHD-related RT variability to the extent that task performance relies on working memory processes for consistent, successful execution (see Figure 1). Support for working memory model predictions would include finding that CE working memory serves as a stronger mediator of the ADHD–RT variability relation than RT variability serves as a mediator of the ADHD–working memory relation. In contrast, finding equivocal results across models—that working memory and RT variability provide similar magnitude mediation effects—would suggest that working memory and RT variability are interrelated yet distinct neurocognitive impairments rather than one impairment being an outcome of the other. To our knowledge, this is the first study to examine the model-implied interrelation between working memory components and RT variability in children with ADHD and typically developing (TD) children.

Method

Participants

The sample comprised 37 boys ages 8 to 12 years ($M = 9.45$ years, $SD = 1.10$) from the southeastern United States, recruited by or referred to the Children's Learning Clinic through community resources (e.g., pediatricians, community mental health clinics, school system personnel, self-referral). Sample ethnicity was mixed and included 25 Caucasian non-Hispanic (68%), eight Hispanic English-speaking (22%), two African American (5%) children, and two children of mixed racial/ethnic background (5%). Typically developing children (those without a suspected psychological disorder) were actively recruited through contact with neighborhood and community schools, family friends of referred children, and other community resources, and consisted primarily of self-referred families who were interested in learning more about their children's cognitive and academic strengths and weaknesses. Psychoeducational evaluations were provided to the parents of all participants.

Two groups of children participated in the study: children with ADHD and TD children without a psychological disorder. All parents and children gave their informed consent/assent to participate in the study, and the university's Institutional Review Board approved the study prior to the onset of data collection.

Group Assignment

All children and their parents participated in a detailed, semistructured clinical interview using the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Aged Children (K-SADS; Kaufman et al., 1997). In addition, all children's K-SADS interviews were supplemented with parent and teacher ratings scales, including the Child Behavior Checklist (Achenbach & Rescorla, 2001), Teacher Report Form (Achenbach & Rescorla, 2001), and the Child Symptom Inventory—Parent and Teacher (Gadow, Sprafkin, Salisbury, Schneider, & Loney, 2004).

Twenty-two children met the following criteria and were included in the ADHD-combined type group: (a) an independent diagnosis by the Children's Learning Clinic's directing clinical psychologist using *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.) criteria for ADHD-combined type based on the K-SADS semistructured interview with parent and child; (b) parent ratings of at least 2 standard deviations above the mean on the Attention Problems Clinical Syndrome scale of the Child Behavior Checklist, or exceeding the criterion score for the Parent Version of the ADHD-Combined Subtype subscale of the Child Symptom Inventory; and (c) teacher ratings of at least 2 standard deviations above the mean on the Attention Problems Clinical Syndrome scale of the Teacher Report Form, or exceeding the criterion score for the Teacher Version of the ADHD-Combined Subtype subscale of the Child Symptom Inventory. All children in the ADHD group met criteria for ADHD-combined type, and eight were comorbid for oppositional defiant disorder.

Fifteen children met the following criteria and were included in the TD group: (a) no evidence of any clinical disorder based on parent and child K-SADS interview, (b) normal developmental history by maternal report, (c) ratings below 1.5 standard deviations on the Clinical Syndrome scales of the Child Behavior Checklist and Teacher Report Form, and (d) parent and teacher ratings within the nonclinical range on all Child Symptom Inventory—Parent and Teacher subscales.

Children who presented with (a) gross neurological, sensory, or motor impairment, (b) history of a seizure disorder, (c) psychosis, or (d) Full Scale IQ score less than 85 were excluded from the study. Nine additional children were assessed but were excluded from the study because of seizure disorder ($n = 1$) or failing to meet inclusion criteria for either the ADHD or TD group ($n = 8$).[1] All children were administered the Wechsler Intelligence Scale for Children (WISC–III or WISC–IV) to estimate Full Scale IQ (Wechsler, 2003). Eleven had previously received psychostimulant trials or were currently prescribed psychostimulants (which were withheld for a minimum of 24 hr prior to each testing session). Demographic data for the two groups are provided in Table 1.

Intraindividual (RT) Variability

Intraindividual (RT) variability was assessed using two choice RT task variants, counterbalanced across 2 weeks of testing, for a total of eight experimental blocks of 32 trials per block. These tasks were administered as part of an ongoing study investigating the role of inhibitory processes in ADHD (Alderson, Rapport, Sarver, & Kofler, 2008) and its relation to ADHD symptoms (Alderson, Rapport, Kasper, Sarver, & Kofler, 2012).[2]

Choice RT task. The choice RT task and administration instructions are identical to the no-tone condition described by Alderson et al. (2008). Stimuli are displayed for 1,000 ms as uppercase letters *X* and *O* positioned in the center of a computer screen. *X*s and *O*s appeared with equal frequency throughout the experimental blocks. Each stimulus (1,000 ms duration) was preceded by a dot (i.e., fixation point) displayed in the center of the screen for 500 ms. Total trial duration was 3,500 ms. The fixation point served as an indicator that a go-stimulus was about to appear. A two-button response box 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*. All participants completed two practice blocks and four consecutive experimental blocks of 32 trials (total of 128 experimental trials). The experimental blocks required approximately 7.5 min to complete.

Ignore-tone choice RT task. The ignore-tone task and administration instructions are identical to the ignore-tone condition described by Alderson et al. (2008), and similar to the choice task with the exception of singleton distractors (1000-Hz auditory tone) that children were instructed to ignore, presented randomly on 25% of the experimental trials. This task was included as part of larger study examining the construct validity of the traditional stop-signal task, and was included as a second RT task in the current study based on evidence that singleton distractors with minimal to no relevance to the task or target stimulus can impact performance (Dalton & Lavie, 2004; Mason, Humphreys, & Kent, 2005), combined with mixed evidence regarding the impact of distractors on performance for children with ADHD (cf. van Mourik, Oosterlaan, Heslenfeld, Konig, & Sergeant, 2007). Previous research with these tasks suggests minimal impact of singleton distractors on overall mean RT (Alderson et al., 2008) or motor activity (Alderson et al., 2012) for children with ADHD and TD children.

Stimuli were displayed for 1,000 ms as uppercase letters *X* and *O* positioned in the center of a computer screen. *X*s and *O*s appeared with equal frequency throughout the experimental blocks. Each 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 stimulus was about to appear. A 1000-Hz auditory tone, delivered through sound-deadening headphones, was generated by the computer and presented randomly on 25% of the experimental trials. Children were instructed to ignore these singleton distractors. A two-button response box 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*. Total trial duration was 3,500 ms. All participants completed two practice blocks and four consecutive experimental blocks of 32 trials, for a total of 128 experimental trials. The experimental blocks required approximately 7.5 min to complete.

Dependent variables: RT variability. Two tasks counterbalanced across testing days were used to index RT variability to control for task-specific, test–retest, and random error as recommended (Shipstead, Redick, & Engle, 2010).[3,4] Anticipatory responses (RTs < 150 ms) were excluded as recommended (e.g., Schmiedek et al., 2007). To assess intraindividual variability, we implemented ex-Gaussian estimation of correct trials using QMPE 2.18, which reliably implements quantile maximum likelihood estimation with as few as 40 trials per child (Cousineau, Brown, & Heathcote, 2004). This method is currently considered the gold standard for isolating performance attributable to RT variability (Kofler et al., 2013; Schmiedek et al., 2007) and separates each child's RT distribution into exponential ("ex-") and normal (Gaussian) components. Within the normal component, this approach provides estimates of mu and sigma. Mu reflects the mean RT of the normal distribution, and sigma reflects the variability of this normal component. If a child's RTs are normally distributed, then mu and sigma will equal their mean RT and standard deviation of RTs, respectively, and tau will equal zero. Tau indexes the exponential component of the RT distribution and reflects the subset of extremely slow responses that otherwise have a strong influence on mean RT and standard deviation of RT calculation. Tau is similar conceptually to a distribution's skewness, but is considered a more reliable metric (Schmiedek et al., 2007). A latent factor was created using principal components factor analysis separately for sigma (59.87% variance accounted for; both factor loadings $r = .77$; eigenvalue $= 1.20$) and tau (79.61% variance accounted for; both factor loadings $r = .89$; eigenvalue $= 1.59$) to remove task-specific, test–retest, and random error and

[1] Of these eight children, four were diagnosed with an anxiety disorder, two with a depressive disorder, and two with an autism spectrum disorder.

[2] Performance data derived from these tasks were used to create dissimilar metrics and evaluate conceptually unrelated hypotheses for a subset of the current sample in a previous study (Alderson et al., 2008).

[3] Because no task is process pure, generalizability of results requires experimenters to use multiple measures of each construct and create factor scores to extract common variance and remove all random and task-specific, nonconstruct error (for review and specific examples, see Shipstead et al., 2010).

Table 1
Sample, Demographic, and Performance Variables

Variable	Group: ADHD (n = 22) *M*	ADHD *SD*	Typically developing (n = 15) *M*	Typically developing *SD*	$F(1, 36)$
Age (years)	9.17	0.93	9.86	1.23	3.79^{+}
FSIQ	102.41	12.11	108.80	11.93	2.52
SES	45.61	10.39	51.93	8.75	3.74^{+}
CBCL Attention Problems	74.38	9.51	55.21	6.73	42.51^{***}
TRF Attention Problems	66.38	9.09	53.36	5.09	23.65^{***}
CSI–Parent	75.76	8.63	47.79	9.96	78.09^{***}
CSI–Teacher	67.71	10.38	48.93	8.69	31.18^{***}
Mu factor (*z*-score)	−0.22	1.21	0.32	0.45	2.78^{+}
Sigma factor (*z*-score)	0.23	1.17	−0.33	0.56	2.95^{+}
Tau factor (*z*-score)	0.38	1.12	−0.55	0.37	9.59^{**}
Central executive factor (*z*-score)	−0.55	0.88	0.81	0.48	29.90^{***}

Note. All analyses were conducted using bias-corrected bootstrapping (N = 5,000 samples). ADHD = attention-deficit/hyperactivity disorder; FSIQ = Full Scale Intelligence Quotient; SES = socioeconomic status; CBCL = Child Behavior Checklist T scores; TRF = Teacher Report Form; CSI = Child Symptom Inventory severity T scores.
$^{+}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

reflect reliable variance associated with intraindividual performance (RT) variability across the two choice RT task variants.[4]

Working Memory

The phonological (PH) and visuospatial (VS) working memory tasks were administered as part of an ongoing series of experiments attempting to falsify key predictions from the functional working memory model (Rapport et al., 2001) regarding the role of working memory functioning in ADHD-related behavioral and functional impairments.[5] Each child was administered four PH and four VS set size conditions (i.e., PH and VS set sizes = 3, 4, 5, and 6) across the four testing sessions. Each of the eight working memory set size conditions contained 24 unique trials of the same stimulus set size, and were counterbalanced across the four testing sessions to control for order effects and potential proactive interference effects across set size conditions (Conway et al., 2005). Five practice trials were administered before each task; children were required to achieve 80% correct before advancing to the full task. Evidence for reliability and validity of the eight working memory tasks includes high internal consistency (α = .82 to .97) and demonstration of the expected magnitude of relations (Swanson & Kim, 2007) with an established measure of STM (WISC-IV Digit Span raw scores: r = .50 to .66; Raiker et al., 2012). Performance data (average stimuli correct per trial) were calculated as recommended (Conway et al., 2005).

PH working memory task. The PH working memory task is similar to the Letter-Number Sequencing subtest of the WISC-IV (Wechsler, 2003) and assesses phonological working memory based on Baddeley's (2007) model. Children were presented a series of jumbled numbers and a capital letter on a computer monitor. Each number and letter (4 cm height) appeared on the screen for 800 ms, followed by a 200-ms interstimulus interval. The letter never appeared in the first or last position of the sequence to minimize potential primacy and recency effects, and was counterbalanced across trials to appear an equal number of times in the other serial positions (i.e., Position 2, 3, 4, or 5). Children were instructed to recall the numbers in order from smallest to largest and to say the letter last (e.g., 4 H 6 2 is correctly recalled as 2 4 6 H). Two trained research assistants, shielded from the participant's view, listened to the children's vocalizations through headphones in a separate room and recorded oral responses independently (interrater reliability was 96.4%). The PH working memory tasks required approximately 4 min (set size = 3) to 7 min (set size = 6) to complete.

VS working memory task. Children were shown nine squares arranged in three offset vertical columns on a computer monitor. A series of 2.5-cm diameter dots (3, 4, 5, or 6) were presented sequentially in one of the nine squares during each trial such that no two dots appeared in the same square on a given trial. All but one dot that was presented within the squares were black; the exception was a red dot that never appeared as the first or last stimulus in the sequence. Children were instructed to indicate the black dots in the serial order presented by pressing the corresponding squares on a computer keyboard and to indicate the serial position of the red dot last. The VS working memory tasks required approximately 4 min (set size = 3) to 6 min (set size = 6) to complete.

Dependent variable: Working memory. Estimates of CE working memory were computed at each set size using the regression approach described by Rapport et al. (2008) as recommended (Swanson & Kim, 2007). Extensive neuropsychological (Baddeley, 2007), neuroanatomical (Smith, Jonides, & Koeppe, 1996),

[4] A one-factor solution best described each construct based on eigenvalues for a second factor all falling below 1.0 for sigma (0.80), tau (0.41), and CE (0.33).

[5] Working memory performance data were used previously to evaluate conceptually unrelated research hypotheses for a subset of participants in the current study (Alderson et al., 2010; Raiker et al., 2012; Rapport et al., 2008) and were not used previously to evaluate RT variability on the choice RT tasks described herein.

neuroimaging (Fassbender & Schweitzer, 2006; Wager & Smith, 2003), and factor analytic (Alloway, Gathercole, & Pickering, 2006) investigations support the distinct functioning of the CE, PH storage/rehearsal, and VS storage/rehearsal components (Baddeley, 2007). This point is central to our approach for estimating CE working memory functioning.

Because no one task or measure is likely to provide an uncontaminated estimate of CE functioning, cognitive scientists have recently embraced an alternative approach to estimate latent constructs that are hypothesized to be domain-general and upstream from subsidiary processes such as the PH and VS storage/rehearsal subsystems (Engle et al., 1999). Briefly, this approach calculates a predicted score by regressing the lower level subsystem processes onto each other based on the assumption that shared variance between the measures (e.g., between the PH and VS tasks) reflects the domain-general, higher order supervisory mechanism of the two processes. The approach is valid to the extent that the higher order CE is domain-general rather than domain-specific—that is, that there is a single higher order system or mechanism responsible for the subsidiary systems rather than a separate controller unique to each subsystem. Studies examining Baddeley's (2007) working memory model uniformly support a domain-general CE (e.g., Alloway et al., 2006) that provides oversight for the distinct PH and VS working memory subsystems (Smith et al., 1996). Contemporary studies have adopted this approach to partition and examine storage/rehearsal and processing (CE) components of working memory using PH storage/rehearsal and PH storage/rehearsal + processing tasks (e.g., Colom, Abad, Rebollo, & Shih, 2005; Engle et al., 1999; Swanson & Kim, 2007), as well as PH and VS working memory tasks (e.g., Rapport et al., 2008). The extraction of "common and perfectly reliable variance" (Swanson & Kim, 2007, p. 158) between working memory tasks using regression or structural equation model-based techniques has the additional benefit of reducing or eliminating variance related to nonworking memory processes and measurement error (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000).[6]

CE working memory functioning was estimated using the unstandardized predicted scores computed by regressing VS scores onto PH scores at each set size and PH scores onto VS scores at each set size. These eight scores were combined via factor analysis (84.06% variance accounted for; factor loadings $r = .89$ to .95; eigenvalue = 3.36) to provide an estimate of CE functioning (i.e., shared variance between VS and PH scores) based on the preceding statistical/methodological rationale. Thus, the CE working memory factor was based on eight set size conditions of 24 trials each, and the RT variability metrics were each based on eight experimental blocks of 32 trials each.

Procedure

All children participated in four consecutive Saturday assessment sessions. The PH, VS, and choice RT tasks were administered as part of a larger battery of laboratory-based tasks that required the child's presence for approximately 2.5 hr per session. All tasks were counterbalanced across testing sessions to minimize order effects.[7] Children completed all tasks while seated alone in an assessment room. Performance was monitored at all times by the examiner, who was stationed just out of the child's view to provide a structured setting while minimizing performance improvements associated with examiner demand characteristics (Gomez & Sanson, 1994; Power, 1992). All children received brief (2–3 min) breaks following each task and preset longer (10–15 min) breaks after every two to three tasks to minimize fatigue. Children were seated approximately 0.66 m from the computer monitor for all tasks.

Mediation Analyses

All analyses were completed using a bias-corrected bootstrapping procedure to minimize Type II error following the steps recommended by Shrout and Bolger (2002). Bootstrapping is appropriate for total sample sizes as low as 20 (Efron & Tibshirani, 1993), and was used to estimate and determine the statistical significance of all total, direct, and indirect effects. All continuous variables were standardized as *z*-scores based on the full sample to facilitate between-model and within-model comparisons and allow unstandardized regression coefficients (*B* weights) to be interpreted as Cohen's *d* effect sizes when predicting from a dichotomous grouping variable (Hayes, 2009). AMOS Version 18.0.2 was used for all analyses, and 5,000 samples were derived from the original sample ($N = 37$) by a process of resampling with replacement (Shrout & Bolger, 2002).

Separate mediation models were tested to examine predictions from the variability and working memory models of ADHD. Adopting mediation analysis terminology, the total effect represents the relation between diagnostic status (ADHD, TD) and RT variability prior to examining whether working memory serves as a significant mediator of this relation (and vice versa; Figure 1 path c). In contrast, the direct effects represent the regression coefficients across models for diagnostic status (ADHD, TD) predicting CE working memory or RT variability (tau, sigma; Figure 1 path a), as well as CE working memory predicting children's RT variability scores (and vice versa; see Figure 1 path b). The magnitude of the pathway in which diagnostic status predicts RT variability scores after accounting for the potential mediating influence of CE working memory (and vice versa) also is considered a direct effect and is reported separately (Figure 1 path c′). The residual difference in effect magnitude before (c pathway) and after (c′ pathway) accounting for the mediating variable reflects the indirect effect for each of the mediating pathways (Figure 1 path ab).

Effect ratios (indirect effect divided by total effect) were calculated to estimate the proportion of each significant total effect that

[6] We acknowledge that some reliable, shared variance may be related to noncentral executive shared method factors, as experimental conditions between our two tasks were as identical as possible by design. Similarly, some shared variance in the RT variability sigma and tau factors may be related to nonvariability factors given that the two RT tasks were designed to be identical with the exception of the presence/absence of singleton distractors. Based on the converging evidence above, however, we believe that a latent approach to isolating working memory and RT variability components provides a more valid estimate of component processes than the use of any single task.

[7] The working memory tasks were counterbalanced across the four weekly testing sessions; the RT variability tasks were counterbalanced across the first 2 weeks of testing to ensure that performance on the ignore-tone task was not contaminated by exposure to a meaningful auditory tone during administration of the traditional stop signal task in Week 3 (Alderson et al., 2008).

Table 2
Intercorrelations Among Variables

Variable	1	2	3	4	5	6	7	8	9	10	11
1. **Group (ADHD/TD)**	—										
2. **CE factor**	**.68*****	—									
3. **Tau factor**	**−.46****	**−.64*****	—								
4. **Sigma factor**	**−.28**†	**−.42*****	**.17, *ns***	—							
5. CE3	.52***	.89***	−.68***	−.50**	—						
6. CE4	.62***	.91***	−.55***	−.38*	.79***	—					
7. CE5	.66***	.95***	−.56***	−.35*	.80***	.82***	—				
8. CE6	.69***	.91***	−.56***	−.31†	.69***	.75***	.88***	—			
9. Sigma IT	−.09, *ns*	−.14, *ns*	−.17, *ns*	.77***	−.22, *ns*	−.12, *ns*	−.12, *ns*	−.07, *ns*	—		
10. Sigma CRT	−.34*	−.50**	.43**	.77***	−.55***	−.47**	−.42**	−.41*	.20, *ns*	—	
11. Tau IT	−.48**	−.52***	.89***	.19, *ns*	−.48**	−.48**	−.45**	−.51***	−.26, *ns*	.43**	—
12. Tau CRT	−.35*	−.62***	.89***	.11, *ns*	−.74***	−.50**	−.54**	−.49**	−.05, *ns*	.35*	.59***

Note. Correlations with group are biserial correlations; bold type indicates factor scores used in the study's primary analyses. ADHD = attention-deficit/hyperactivity disorder; TD = typically developing; CE = central executive working memory; IT = ignore-tone (singleton distractor) choice reaction time task; CRT = choice reaction time task.
† $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$. *ns* = $p \geq .12$.

was attributable to the mediating pathway (indirect effect). Cohen's *d* effect sizes, standard errors, 90% confidence intervals for indirect effects, and effect ratios are shown in Tables 2 and 3. Ninety percent confidence intervals were selected over 95% confidence intervals because the former are more conservative for evaluating mediating effects (Shrout & Bolger, 2002).[8]

Intercorrelations between diagnostic status, CE working memory, and intraindividual variability (tau, sigma) were computed in Tier I of the analyses to determine whether mediation analyses were justified. Tiers II and III examined the extent to which CE working memory and RT variability serve as statistically significant mediators of one another, and Tier IV directly contrasted the variability and working memory models using *z*-score tests for equality of regression coefficients (Paternoster, Mazerolle, & Piquero, 1998) to examine the interrelation among effects.

Results

Preliminary Analyses

All variables were screened for univariate/multivariate outliers and tested against $p < .001$. No significant outliers were found. All parent and teacher behavior rating scale scores were significantly higher for the ADHD group relative to the TD group as expected (see Table 1). Children with ADHD and TD children did not differ with respect to age ($p = .06$), socioeconomic status ($p = .06$), or Full Scale IQ ($p = .12$). In addition, Full Scale IQ was not analyzed as a covariate because it shares significant variance with working memory ($r = .75$ to $.79$; Wechsler, 2003) and would result in removing substantial variance associated with working memory from working memory (Ackerman, Beier, & Boyle, 2005). We therefore report simple model results with no covariates so that *B* weights of key pathways can be interpreted as Cohen's *d* effect sizes when predicting from the dichotomous ADHD/TD grouping variable (Hayes, 2009).

Tier I: Intercorrelations and Mediator-Independent Effect Sizes

Intercorrelations between all variables were computed via bootstrapping (90% confidence intervals) as a first step to determine whether mediation analyses were justified (see Table 2). Diagnostic status was coded as 0 = ADHD and 1 = TD such that positive values indicate higher scores for the TD relative to ADHD group. Higher scores reflect better performance on CE working memory and more variable performance on the RT variability indices.

Examination of effect sizes independent of potential mediators indicated large magnitude between-group differences in CE ($d = 1.36$, $SE = 0.22$) and RT variability as indexed by tau ($d = -0.93$, $SE = 0.30$), indicating that children with ADHD demonstrated less developed CE functioning and increased performance variability relative to TD children. The large CE and tau effect sizes did not differ significantly in magnitude ($z = 1.16$, $p = .12$; Paternoster et al., 1998); the CE effect size was larger than the moderate magnitude effect size for sigma ($d = -0.56$, $SE = 0.28$; $z = 2.25$, $p = .01$), whereas the difference between tau and sigma did not reach statistical significance ($z = 0.99$, $p = .16$).

Tier II: Test of Variability Models of ADHD

Standardized *B* weights (interpreted as Cohen's *d* effect sizes when predicting from the dichotomous grouping variable; Hayes, 2009), standard errors, and 90% confidence intervals for all bootstrap analyses are displayed in Table 3.

Total effects of diagnostic status on working memory components. Examination of the total effect (Figure 1 path c; Table 3) revealed that diagnostic status was related significantly to CE working memory (Cohen's $d = 1.36$) prior to accounting for the potential mediating role of RT variability (as indexed by tau, sigma).

[8] Briefly, the wider 95% confidence interval increases the likelihood that the confidence interval for c′ will include 0.0, indicating that diagnostic status and the dependent variable are no longer related significantly after accounting for the mediator (i.e., full mediation in Baron & Kenny, 1986, terminology). In contrast, the narrower 90% confidence interval is less likely to include 0.0, and therefore is likely to result in a more conservative conclusion regarding the magnitude of the relation between diagnostic status and the dependent variable after accounting for the mediator (i.e., partial mediation). For discussion and specific examples of this phenomenon, see Shrout and Bolger (2002).

Table 3
Reaction Time Variability as a Mediator of the Relation Between ADHD Status and CE Working Memory Functioning

		Reaction time variability component			
		Tau		Sigma	
Path	Effect	*B*	*SE*	*B*	*SE*
CE	Direct	*B*	*SE*	*B*	*SE*
a	Dx → RTV	−0.93***	0.30	−0.56*	0.28
b	RTV → CE	−0.41**	0.12	−0.25**	0.12
c	Dx → CE	1.36***	.22	1.36***	0.22
c′	Dx → CE	0.98***	0.24	1.22***	0.23
	Indirect (through RTV mediator)				
ab	Dx → CE	0.39***	0.13	0.14*	0.11
	90% CI of bootstrap	[0.20, 0.64]		[0.08, 0.37]	
	Effect ratio	0.29		0.10	

Note. Path labels reflect standard nomenclature (cf. Fritz & MacKinnon, 2007) and are depicted in Figure 1; c and c′ reflect the direct effect of diagnostic status on CE working memory before and after accounting for reaction time variability, respectively. ADHD = attention-deficit/hyperactivity disorder; CE = central executive; Dx = diagnostic status; RT = reaction time; RTV = reaction time variability.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Tau mediating CE working memory. Diagnostic status significantly predicted tau (Figure 1 path a; Cohen's $d = -0.93$), wherein an ADHD diagnosis was associated with a greater subset of abnormally slow responses (Table 3). Tau was related also to CE ($B = -0.41$). Examination of the mediation pathway (Figure 1 path ab) revealed that diagnostic status exerted a significant small-to-medium magnitude indirect effect on CE ($d = 0.39$) through its impact on tau. In doing so, it was associated with a modest reduction in the magnitude of ADHD-related CE deficits (*d* changed from 1.36 to 0.98; ER = 0.29). The relation between diagnostic status and CE remained significant ($p < .0005$) after accounting for tau. Examination of the effect ratio indicated that tau accounted for 29% of the relation between diagnostic status and CE, such that large magnitude CE deficits remained.

Sigma mediating working memory components. Diagnostic status significantly predicted sigma (Figure 1 path a; Cohen's $d = -0.56$), wherein an ADHD diagnosis was associated with moderately more variable performance across all trials (Table 2). Sigma was also related to CE ($B = -0.25$). Examination of the mediation pathway (Figure 1 path ab) revealed that diagnostic status exerted a significant, small magnitude indirect effect on CE ($d = 0.14$) through its impact on sigma. In doing so, it was associated with a modest reduction in the magnitude of ADHD-related CE deficits (*d* changed from 1.36 to 1.22; ER = 0.10). The relation between diagnostic status and CE remained significant ($p \leq .001$). Examination of the effect ratio indicated that 10% of the relation between diagnostic status and CE working memory was attributable to the indirect effect of RT variability; large magnitude between-group differences in CE working memory ($p < .0005$) remained after accounting for this relation.

Tier III: Test of the Working Memory Model of ADHD

Standardized *B* weights (interpreted as Cohen's *d* effect sizes when predicting from the dichotomous grouping variable), standard errors, and 90% confidence intervals for all bootstrap analyses are displayed in Table 4.

Total effects of diagnostic status on RT variability. Examination of the total effects (Figure 1 path c; Table 4) across RT variability metrics revealed that diagnostic status was related

Table 4
CE Working Memory as a Mediator of Relation Between ADHD Status and Reaction Time (RT) Variability

		Central executive working memory	
Path	Effect	*B*	*SE*
Tau	Direct		
a	Dx → CE	1.36***	0.22
b	CE → tau	−0.60***	0.19
c	Dx → tau	−0.93***	0.25
c′	Dx → tau	−0.11	0.19
	Indirect (through CE mediator)		
ab	Dx → tau	−0.82***	0.28
	90% CI of bootstrap	[−1.35, −0.42]	
	Effect ratio	0.88	
Sigma	Direct		
a	Dx → CE	1.36***	0.22
b	CE → sigma	−0.43**	0.24
c	Dx → sigma	−0.56*	0.28
c′	Dx → sigma	0.02	0.37
	Indirect (through CE mediator)		
ab	Dx → sigma	−0.58**	0.36
	90% CI of bootstrap	[−1.31, −0.09]	
	Effect ratio	1.00	

Note. Path labels reflect standard nomenclature (cf. Fritz & MacKinnon, 2007) and are depicted in Figure 1; c and c′ reflect the direct effect of diagnostic status on intraindividual variability before and after accounting for working memory, respectively. ADHD = attention-deficit/hyperactivity disorder; CE = central executive; Dx = diagnostic status.
* $p < .05$. ** $p < .01$. *** $p < .001$.

significantly to both tau (Cohen's $d = -0.93$) and sigma (Cohen's $d = -0.56$) prior to accounting for the potential mediating role of CE working memory. These values indicate that children with ADHD demonstrated greater RT variability relative to TD children across both metrics.

CE working memory mediating RT variability. Diagnostic status significantly predicted CE (Figure 1 path a; Cohen's $d = 1.36$), wherein an ADHD diagnosis was associated with lower CE performance. CE was related also to RT variability (Figure 1 path b) as indexed by both tau ($B = -0.60$) and sigma ($B = -0.43$). Examination of the mediation pathway (Figure 1 path ab) revealed that diagnostic status exerted a significant large magnitude indirect effect on RT variability through its impact on children's CE performance (Cohen's $d = -0.82$ and -0.58 across the tau and sigma models, respectively). In doing so, it reduced significantly the magnitude of ADHD-related RT variability (Figure 1 path c′) as indexed by tau (d changed from -0.93 to -0.11; ER = 0.88) and sigma (d changed from -0.56 to 0.02; ER = 1.0). Examination of the effect ratios indicated that 88% to 100% of the relation between diagnostic status and both RT variability metrics was conveyed via the indirect effect through CE performance such that direct relations between diagnostic status and RT variability were no longer detectable ($ps \geq .75$).

Tier IV: Model Comparisons

A final set of analyses was undertaken to examine evidence of model-implied directionality between CE working memory and RT variability by comparing the magnitude of mediation effects between models. Specifically, the absolute values of the means and standard errors of the c′ pathways (Tables 2 and 3) were used to calculate a z-score to test the equality of regression coefficients (Paternoster et al., 1998).

Results indicated that significantly larger magnitude between-group differences (Figure 1 path c′) remained for CE after accounting for the mediating influence of tau (CE $d = 0.98$, $SE = 0.24$) and sigma (CE $d = 1.22$, $SE = 0.23$) when compared with the magnitude of between-group differences in tau (tau $d = -0.11$, $SE = 0.19$) and sigma (sigma $d = 0.02$, $SE = 0.37$) after accounting for CE ($zs \geq -2.75$, $p \leq .003$). These results suggest that CE functioning may exert a larger magnitude effect on RT variability than RT variability exerts on CE functioning. In addition, this pattern was apparent for both tau and sigma, suggesting CE involvement in both preventing abnormally slow responses (tau) and maintaining consistent performance across all trials of a task (sigma).

Discussion

The current study is the first to test competing predictions stemming from the variability (Castellanos et al., 2005; Russell et al., 2006) and functional working memory (Rapport et al., 2001, 2008) models by examining neurocognitive-level predictions regarding the interrelation between working memory and performance variability in ADHD. Examination of the relation between ADHD status and both constructs revealed a pattern of between-groups differences highly consistent with recent meta-analytic reviews (Kasper, Alderson, & Hudec, 2012; Kofler et al., 2013; Willcutt et al., 2008). Specifically, inspection of the total effects (Cohen's d effect sizes) prior to accounting for potential mediators (Figure 1 c path) across models indicated that children with ADHD demonstrated similar, large magnitude deficits in CE working memory and RT variability as indexed by tau. In contrast, smaller magnitude between-group differences were apparent for variability within the normal distribution (sigma). The significantly larger magnitude between-group difference in tau relative to sigma ($p < .001$) was highly consistent with results from multiple, independent investigations (e.g., Epstein et al., 2011; Leth-Steensen et al., 2000; Vaurio, Simmonds, & Mostofsky, 2009) and a meta-analysis (Kofler et al., 2013), and provides additional evidence that RT variability in ADHD is attributable primarily to a subset of abnormally slow responses (indexed by tau) rather than inconsistent performance across all task trials (sigma). Of primary interest in the current study was the extent to which this subset of abnormally slow responses indexes a primary neurocognitive indicator of the disorder, as predicted by the default mode network (Castellanos et al., 2005) and cognitive neuroenergetic models (Russell et al., 2006), or whether increased RT variability in children with ADHD is attributable to alternate neurocognitive mechanisms and processes as proposed by the functional working memory model (Rapport et al., 2001, 2008).

As expected, the working memory and RT variability constructs were related highly across most comparisons. Specifically, strong interrelations emerged between CE working memory and both RT variability indices ($r = -.42$ to $-.64$), similar to previous findings in both nonclinical (Schmiedek et al., 2007) and ADHD samples (Buzy et al., 2009; Klein et al., 2006; Piek et al., 2004). These findings suggest that well-developed CE working memory abilities are associated with more consistent, less variable performance on laboratory tasks. Inspection of the mediation results across models provides additional information about the interrelation between these variables and reveals a consistent pattern in which ADHD-related CE working memory deficits fully mediated RT variability as measured by both tau and sigma. In doing so, CE working memory accounted for 88% to 100% of between-group differences in RT variability, and between-group differences were no longer detectable for either of the RT variability metrics after accounting for ADHD-related CE dysfunction (effect sizes = -0.11 to 0.02; both 90% confidence intervals include 0.0, indicating $p > .10$). In addition, this pattern was apparent for both tau and sigma, suggesting CE involvement in both preventing abnormally slow responses (tau) and maintaining consistent performance across all trials of a task (sigma). This pattern of results is consistent with previous experimental studies finding an association between ADHD-related increases in RT variability and increases in cognitive load on a visual serial addition working memory task (Buzy et al., 2009), as well as previous studies demonstrating a relation between RT variability and performance on recognition-based memory tasks (Klein et al., 2006; Piek et al., 2004). In contrast, both RT variability metrics emerged as significant, partial mediators of the relation between diagnostic status and CE working memory. RT variability accounted for 10% to 29% of ADHD-related CE deficits across models, and large magnitude between-group differences in CE working memory remained after accounting for RT variability ($d = 0.98$ to 1.22).

Finally, direct comparison of variability and working memory model predictions (Tier IV) provided additional support for the observed interrelation pattern such that CE working memory exerted a stronger mediating role on RT variability than RT variability exerted on CE working memory for both tau and sigma. Collectively, this pattern of results is inconsistent with models describing performance variability as a primary neurocognitive indicator in ADHD, and suggests that ADHD-related RT variability may reflect a secondary outcome of CE working memory deficits. That is, ADHD-related inconsistent performance on RT tasks appears to reflect a behavioral outcome of CE working memory failures rather than impaired working memory performance reflecting an outcome of ADHD-related variability. These findings add to the growing literature implicating CE working memory deficits in many of the hallmark behavioral and functional impairments associated with ADHD. Specifically, recent behavioral and neurocognitive research suggests that CE deficits may underlie ADHD-related impairments in attentive behavior (Burgess et al., 2010; Kofler et al., 2010), hyperactivity (Rapport et al., 2009), impulsive responding (Raiker et al., 2012), and behavioral disinhibition (Alderson et al., 2010; Lee, Riccio, & Hynd, 2004), and strongly predict ADHD-related social problems (Kofler et al., 2011).

Future research is needed to determine which CE working memory subprocesses (focus of attention; mental processing, reordering, and updating of information held in STM) contribute to increased RT variability in ADHD. Among these candidate processes, periodic lapses in attention are among the most frequent attributions for increased ADHD-related RT variability (e.g., Castellanos et al., 2005; Tamm et al., 2012). This ascription is supported by evidence that the abnormally slow RTs that skew RT distributions and contribute to higher tau scores are often preceded or followed by omission errors (Epstein et al., 2010), as well as previous findings of large magnitude visual attention deficits in ADHD (Kofler, Rapport, & Alderson, 2008). Given that RT variability is frequently attributed to attentional lapses (Castellanos et al., 2005; Tamm et al., 2012), however, the current results suggest that shared attentional demands or other third variable explanations are unlikely to account for the working memory–RT variability relation. In addition, attentional lapse models are inconsistent with experimental and meta-analytic conclusions that attentional processes may be intact in ADHD (Huang-Pollock & Nigg, 2003; Huang-Pollock, Nigg, & Carr, 2005; van der Meere & Sergeant, 1987) or secondary to deficits in the CE component of working memory (Burgess et al., 2010; Kofler et al., 2010). Similarly, Epstein et al. (2010) found that temporal performance-based indices of inattention (omission errors) and behavior inhibition could not account for ADHD-related impairments in RT variability (Cohen's *d* changed minimally from .78 to .70). Finally, recent evidence reveals that children with ADHD continue to demonstrate large magnitude working memory deficits, even after accounting for their visual attention during working memory tasks, whereas between-group differences in objectively observed attentive behavior were not detectable after accounting for ADHD-related CE deficits (Kofler et al., 2010). Thus, it appears likely that examination of processes beyond attentive behavior will be needed to explain the replicated finding that children with ADHD are consistently inconsistent in their performance on neurocognitive tasks. In the current study, we were unable to fractionate the CE to examine the impact of specific CE subprocesses because the working memory tasks required children to engage multiple CE functions, including mentally processing, reordering, and updating PH and VS information held in STM.

The unique contribution of the current study is its examination of opposing, neurocognitive-level predictions from the variability (Castellanos et al., 2005; Russell et al., 2006) and functional working memory models (Rapport et al., 2001, 2008) of ADHD. Several caveats merit consideration despite methodological refinements such as the use of bias-corrected bootstrapping procedures and error-free latent factor scores derived from multiple tasks measuring each neurocognitive construct. Generalization of findings from highly controlled, laboratory-based experimental investigations is always limited to some extent, and smaller *N* studies are at inherent risk for Type II errors. Our cell sizes were nevertheless sufficient based on power analysis for bias-corrected, bootstrapped mediation analyses (Efron & Tibshirani, 1993; Shrout & Bolger, 2002). Furthermore, large magnitude ADHD-related deficits in CE working memory remained after accounting for the impact of RT variability ($d = -0.98$ to -1.22), and examination of ADHD-related RT variability revealed small, nonsignificant effect sizes (effect sizes $= -0.11$ to 0.02) after accounting for CE working memory. That is, increased power with a larger sample may, at best, suggest that RT variability only contributes to a small portion of between-group performance differences relative to the larger contribution of CE working memory. In addition, conclusions from the current study are limited to boys ages 8–12 years with the combined type of ADHD and may vary when a broader age range, other subtypes, and/or girls are examined. Although previous research has demonstrated that the ADHD subtypes perform similarly on both working memory (Mayes, Calhoun, Chase, Mink, & Stagg, 2009) and RT variability tasks (Epstein et al., 2011; Kofler et al., 2013; Vaurio et al., 2009), it is unclear whether the relation among these neurocognitive constructs follows a similar pattern across the phenotypes.

Finally, the current findings are based on children's RT distributions derived from contemporary laboratory-based tasks; however, the extent to which CE working memory impairments may account for variability in classroom behavior (Kofler et al., 2008), physiological indices (Castellanos et al., 2005), and other types of tasks and settings is currently unknown and warrants empirical scrutiny. Importantly, the current study addressed neurocognitive-level predictions; the findings do not invalidate neurobiological predictions of the models tested, but rather question the role of RT variability as a primary neurocognitive indicator and suggest that working memory may be upstream from RT variability and thus a potentially more direct measure of the biological systems in question.

The search to identify the neurocognitive architecture of ADHD parallels our search for the underlying causes of this potentially debilitating disorder of brain, behavior, and development, and holds promise for developing improved diagnostic tools, identifying novel intervention targets, and expanding our understanding of the underlying mechanisms and processes responsible for the hallmark behavioral symptoms and functional impairments asso-

ciated with ADHD. The current study adds to a growing literature implicating CE working memory deficits in core ADHD behavioral, neurocognitive, and functional impairments, and suggests that interventions targeting these processes may be beneficial for ameliorating ADHD symptoms. Investigations using active training components that place greater demands on CE processes are needed to determine the extent to which cognitive training of these processes can result in greater magnitude and broader treatment effects.

References

Achenbach, T. M., & Rescorla, L. A. (2001). *Manual for the ASEBA School-Age Forms & Profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families.

Ackerman, P. L., Beier, M. E., & Boyle, M. O. (2005). Working memory and intelligence: The same or different constructs? *Psychological Bulletin, 131,* 30–60. doi:10.1037/0033-2909.131.1.30

Alderson, R. M., Rapport, M. D., Hudec, K. L., Sarver, D. E., & Kofler, M. J. (2010). Competing core processes in ADHD: Do working memory deficiencies underlie behavioral inhibition deficits? *Journal of Abnormal Child Psychology, 38,* 497–507. doi:10.1007/s10802-010-9387-0

Alderson, R. M., Rapport, M. D., Kasper, L. J., Sarver, D. E., & Kofler, M. J. (2012). Hyperactivity in children with attention-deficit/hyperactivity disorder (ADHD): The association between deficient behavioral inhibition, attentional processes, and objectively measured activity. *Child Neuropsychology, 18,* 487–505. doi:10.1080/09297049.2011.631905

Alderson, R. M., Rapport, M. D., Sarver, D. E., & Kofler, M. J. (2008). ADHD and behavioral inhibition: A re-examination of the stop-signal task. *Journal of Abnormal Child Psychology, 36,* 989–998. doi:10.1007/s10802-008-9230-z

Alloway, T. P., Gathercole, S. E., & Pickering, S. J. (2006). Verbal and visual–spatial short-term and working memory in children: Are they separable? *Child Development, 77,* 1698–1716. doi:10.1111/j.1467-8624.2006.00968.x

Baddeley, A. (2007). *Working memory, thought, and action*. New York, NY: Oxford University Press. doi:10.1093/acprof:oso/9780198528012.001.0001

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, statistical considerations. *Journal of Personality and Social Psychology, 51,* 1173–1182. doi:10.1037/0022-3514.51.6.1173

Barry, R. J., Clarke, A. R., McCarthy, R., Selikowitz, M., & Rushby, J. A. (2005). Arousal and activation in a continuous performance task: An exploration of state effects in normal children. *Journal of Psychophysiology, 19,* 91–99. doi:10.1027/0269-8803.19.2.91

Burgess, G. C., Depue, B. E., Ruzic, L., Willcutt, E. G., Du, Y. P., & Banich, M. T. (2010). Attentional control activation relates to working memory in ADHD. *Biological Psychiatry, 67,* 632–640. doi:10.1016/j.biopsych.2009.10.036

Buzy, W. M., Medoff, D. R., & Schweitzer, J. B. (2009). Intra-individual variability among children with ADHD on a working memory task: An ex-Gaussian approach. *Child Neuropsychology, 15,* 441–459. doi:10.1080/09297040802646991

Castellanos, F. X., Sonuga-Barke, E. J. S., Scheres, A., Di Martino, A., Hyde, C., & Walters, J. R. (2005). Varieties of ADHD-related intra-individual variability. *Biological Psychiatry, 57,* 1416–1423. doi:10.1016/j.biopsych.2004.12.005

Colom, R., Abad, F. J., Rebollo, I., & Shih, P. (2005). Memory span and general intelligence: A latent-variable approach. *Intelligence, 33,* 623–642. doi:10.1016/j.intell.2005.05.006

Conway, A. R., Kane, M. J., Bunting, M. F., Hambrick, D. Z., Wilhelm, O., & Engle, R. W. (2005). Working memory span tasks: A methodological review and user's guide. *Psychonomic Bulletin & Review, 12,* 769–786. doi:10.3758/BF03196772

Cousineau, D., Brown, S., & Heathcote, A. (2004). Fitting distributions using maximum likelihood: Methods and packages. *Behavior Research Methods, Instruments, & Computers, 36,* 742–756. doi:10.3758/BF03206555

Dalton, P., & Lavie, N. (2004). Auditory attentional capture: Effects of singleton distractor sounds. *Journal of Experimental Psychology: Human Perception and Performance, 30,* 180–193. doi:10.1037/0096-1523.30.1.180

Dickstein, S. G., Bannon, K., Castellanos, F. X., & Milham, M. P. (2006). The neural correlates of ADHD: An ALE meta-analysis. *Journal of Child Psychology and Psychiatry, 47,* 1051–1062. doi:10.1111/j.1469-7610.2006.01671.x

Efron, B., & Tibshirani, R. J. (1993). *An introduction to the bootstrap*. New York, NY: Chapman & Hall/CRC.

Engle, R. W., Tuholski, S. W., Laughlin, J. E., & Conway, A. R. A. (1999). Working memory, short-term memory, and general fluid intelligence: A latent variable approach. *Journal of Experimental Psychology: General, 128,* 309–331. doi:10.1037/0096-3445.128.3.309

Epstein, J. N., Hwang, M. E., Antonini, T., Langberg, J. M., Altaye, M., & Arnold, L. E. (2010). Examining predictors of reaction times in children with ADHD and normal controls. *Journal of the International Neuropsychological Society, 16,* 138–147. doi:10.1017/S1355617709991111

Epstein, J. N., Langberg, J. M., Rosen, P. J., Graham, A., Narad, M. E., Anonini, T. N., . . . Altaye, M. (2011). Evidence for higher reaction time variability for children with ADHD on a range of cognitive tasks including reward and event rate manipulations. *Neuropsychology, 25,* 427–441. doi:10.1037/a0022155

Fassbender, C., & Schweitzer, J. B. (2006). Is there evidence for neural compensation in ADHD? A review of the functional neuroimaging literature. *Clinical Psychology Review, 26,* 445–465. doi:10.1016/j.cpr.2006.01.003

Fritz, M. S., & MacKinnon, D. P. (2007). Required sample size to detect the mediated effect. *Psychological Science, 18,* 233–239. doi:10.1111/j.1467-9280.2007.01882.x

Gadow, K. D., Sprafkin, J., Salisbury, H., Schneider, J., & Loney, J. (2004). Further validity evidence for the Teacher Version of the Child Symptom Inventory—4. *School Psychology Quarterly, 19,* 50–71. doi:10.1521/scpq.19.1.50.29408

Garon, N., Bryson, S. E., & Smith, I. M. (2008). Executive function in preschoolers: A review using an integrative framework. *Psychological Bulletin, 134,* 31–60. doi:10.1037/0033-2909.134.1.31

Gomez, R., & Sanson, A. V. (1994). Effects of experimenter and mother presence on the attentional performance and activity of hyperactive boys. *Journal of Abnormal Child Psychology, 22,* 517–529. doi:10.1007/BF02168935

Hayes, A. F. (2009). Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Communication Monographs, 76,* 408–420. doi:10.1080/03637750903310360

Huang-Pollock, C. L., & Nigg, J. T. (2003). Searching for the attention deficit in ADHD: The case of visuospatial orienting. *Clinical Psychology Review, 23,* 801–830. doi:10.1016/S0272-7358(03)00073-4

Huang-Pollock, C. L., Nigg, J. T., & Carr, T. H. (2005). Deficient attention is hard to find: Applying the perceptual load model of selective attention to attention deficit hyperactivity disorder subtypes. *Journal of Child Psychology and Psychiatry, 46,* 1211–1218. doi:10.1111/j.1469-7610.2005.00410.x

Kane, M. J., Brown, L. H., McVay, J. C., Silvia, P. J., Myin-Germeys, I., & Kwapil, T. R. (2007). For whom the mind wanders, and when: An experience-sampling study of working memory and executive control in daily life. *Psychological Science, 18,* 614–621. doi:10.1111/j.1467-9280.2007.01948.x

Kasper, L. J., Alderson, R. M., & Hudec, K. L. (2012). Moderators of working memory deficits in children with ADHD: A meta-analytic review. *Clinical Psychology Review, 32,* 605–617. doi:10.1016/j.cpr.2012.07.001

Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., . . . Ryan, N. (1997). Kiddie Schedule for Affective Disorders and Schizophrenia for School-Aged Children–PL: Initial reliability and validity data. *Journal of the American Academy of Child & Adolescent Psychiatry, 36,* 980–988. doi:10.1097/00004583-199707000-00021

Klein, C., Wendling, K., Huettner, P., Ruder, H., & Peper, M. (2006). Intra-subject variability in attention-deficit hyperactivity disorder. *Biological Psychiatry, 60,* 1088–1097. doi:10.1016/j.biopsych.2006.04.003

Kofler, M. J., Rapport, M. D., & Alderson, R. M. (2008). Quantifying ADHD classroom inattentiveness, its moderators, and variability: A meta-analytic review. *Journal of Child Psychology and Psychiatry, 49,* 59–69. doi:10.1111/j.1469-7610.2007.01809.x

Kofler, M. J., Rapport, M. D., Bolden, J., Sarver, D. E., & Raiker, J. S. (2010). ADHD and working memory: The impact of central executive deficits and exceeding storage/rehearsal capacity on observed inattentive behavior. *Journal of Abnormal Child Psychology, 38,* 149–161. doi:10.1007/s10802-009-9357-6

Kofler, M. J., Rapport, M. D., Bolden, J., Sarver, D. E., Raiker, J. S., & Alderson, R. M. (2011). Working memory deficits and social problems in children with ADHD. *Journal of Abnormal Child Psychology, 39,* 805–817. doi:10.1007/s10802-011-9492-8

Kofler, M. J., Sarver, D. E., Rapport, M. D., Raiker, J. S., Orban, S. A., Friedman, L. M., & Kolomeyer, E. G. (2013). Reaction time variability in ADHD: A meta-analytic review of 319 studies. *Clinical Psychology Review*. Advance online publication. doi:10.1016/j.cpr.2013.06.001

Lee, D., Riccio, C. A., & Hynd, G. W. (2004). The role of executive functions in ADHD: Testing predictions from two models. *Canadian Journal of School Psychology, 19,* 167–189. doi:10.1177/082957350401900109

Leth-Steensen, C., Elbaz, Z. K., & Douglas, V. I. (2000). Mean response times, variability, and skew in the responding of ADHD children: A response time distributional approach. *Acta Psychologica, 104,* 167–190. doi:10.1016/S0001-6918(00)00019-6

Mason, D. J., Humphreys, G. W., & Kent, L. (2005). Insights into the control of attentional set in ADHD using the attentional blink paradigm. *Journal of Child Psychology and Psychiatry, 46,* 1345–1353. doi:10.1111/j.1469-7610.2005.01428.x

Mayes, S. D., Calhoun, S. L., Chase, G. A., Mink, D. M., & Stagg, R. E. (2009). ADHD subtypes and co-occurring anxiety, depression, and oppositional-defiant disorder: Differences in Gordon Diagnostic System and Wechsler Working Memory and Processing Speed Index scores. *Journal of Attention Disorders, 12,* 540–550. doi:10.1177/1087054708320402

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., & Howerter, A. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41,* 49–100. doi:10.1006/cogp.1999.0734

Paternoster, R., Mazerolle, P., & Piquero, A. (1998). Using the correct statistical test for the equality of regression coefficients. *Criminology, 36,* 859–866. doi:10.1111/j.1745-9125.1998.tb01268.x

Piek, J. P., Dyck, M. J., Nieman, A., Anderson, M., Hay, D., Smith, L. M., . . . Hallmayer, J. (2004). The relationship between motor coordination, executive functioning and attention in school aged children. *Archives of Clinical Neuropsychology, 19,* 1063–1076. doi:10.1016/j.acn.2003.12.007

Power, T. J. (1992). Contextual factors in vigilance testing of children with ADHD. *Journal of Abnormal Child Psychology, 20,* 579–593. doi:10.1007/BF00911242

Raiker, J. S., Rapport, M. D., Kofler, M. J., & Sarver, D. E. (2012). Objectively measured impulsivity and ADHD: Testing competing predictions from the working memory and behavioral inhibition models. *Journal of Abnormal Child Psychology, 40,* 699–713. doi:10.1007/s10802-011-9607-2

Rapport, M. D., Alderson, R. M., Kofler, M. J., Sarver, D. E., Bolden, J., & Sims, V. (2008). Working memory deficits in boys with ADHD: The contribution of central executive and subsystem processes. *Journal of Abnormal Child Psychology, 36,* 825–837. doi:10.1007/s10802-008-9215-y

Rapport, M. D., Bolden, J., Kofler, M. J., Sarver, D. E., Raiker, J. S., & Alderson, R. M. (2009). Hyperactivity in boys with ADHD: A ubiquitous core symptom or manifestation of working memory deficits? *Journal of Abnormal Child Psychology, 37,* 521–534. doi:10.1007/s10802-008-9287-8

Rapport, M. D., Chung, K., Shore, G., & Isaacs, P. (2001). A conceptual model of child psychopathology: Implications for understanding attention deficit hyperactivity disorder and treatment efficacy. *Journal of Clinical Child Psychology, 30,* 48–58. doi:10.1207/S15374424JCCP3001_6

Russell, V. A., Oades, R. D., Tannock, R., Killeen, P. R., Auerbach, J. G., Johansen, E. B., & Sagvolden, T. (2006). Response variability in attention-deficit/hyperactivity disorder: A neuronal and glial energetics hypothesis. *Behavioral and Brain Functions, 2,* 30. doi:10.1186/1744-9081-2-30

Schmiedek, F., Hildebrandt, A., Lövdén, M., Wilhelm, O., & Lindenberger, U. (2009). Complex span versus updating tasks of working memory: The gap is not that deep. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 35,* 1089–1096. doi:10.1037/a0015730

Schmiedek, F., Oberauer, K., Wilhelm, O., Süß, H.-M., & Wittmann, W. W. (2007). Individual differences in components of reaction time distributions and their relations to working memory and intelligence. *Journal of Experimental Psychology: General, 136,* 414–429. doi:10.1037/0096-3445.136.3.414

Shaw, P., Eckstrand, K., Sharp, W., Blumenthal, J., Lerch, J. P., Greenstein, D., . . . Rapoport, J. L. (2007). ADHD is characterized by a delay in cortical maturation. *Proceedings of the National Academy of Sciences, USA, 104,* 19649–19654. doi:10.1073/pnas.0707741104

Shipstead, Z., Redick, T. S., & Engle, R. W. (2010). Does working memory training generalize? *Psychologica Belgica, 50,* 245–276.

Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods, 7,* 422–445. doi:10.1037/1082-989X.7.4.422

Smith, E. E., Jonides, J., & Koeppe, R. A. (1996). Dissociating verbal and spatial working memory using PET. *Cerebral Cortex, 6,* 11–20. doi:10.1093/cercor/6.1.11

Sonuga-Barke, E. J., & Castellanos, F. X. (2007). Spontaneous attentional fluctuations in impaired states and pathological conditions: A neurobiological hypothesis. *Neuroscience & Biobehavioral Reviews, 31,* 977–986. doi:10.1016/j.neubiorev.2007.02.005

Swanson, L., & Kim, K. (2007). Working memory, short-term memory, and naming speed as predictors of children's mathematical performance. *Intelligence, 35,* 151–168. doi:10.1016/j.intell.2006.07.001

Tamm, L., Narad, M. E., Antonini, T. N., O'Brien, K. M., Hawk, L. W., & Epstein, J. N. (2012). Reaction time variability in ADHD: A review. *Neurotherapeutics, 9,* 500–508. doi:10.1007/s13311-012-0138-5

van der Meere, J. J., & Sergeant, J. A. (1987). A divided attention experiment with pervasively hyperactive children. *Journal of Abnormal Child Psychology, 15,* 379–392. doi:10.1007/BF00916456

van Mourik, R., Oosterlaan, J., Heslenfeld, D. J., Konig, C. E., & Sergeant, J. A. (2007). When distraction is not distracting: A behavioral and ERP study on distraction in ADHD. *Clinical Neuropsychology, 118,* 1855–1865.

Vaurio, R. G., Simmonds, D. J., & Mostofsky, S. H. (2009). Increased intra-individual reaction time variability in attention-deficit/hyperactiv-

ity disorder across response inhibition tasks with different cognitive demands. *Neuropsychologia, 47,* 2389–2396. doi:10.1016/j.neuropsychologia.2009.01.022

Wager, T. D., & Smith, E. E. (2003). Neuroimaging studies of working memory: A meta-analysis. *Cognitive, Affective, & Behavioral Neuroscience, 3,* 255–274. doi:10.3758/CABN.3.4.255

Wechsler, D. (2003). *Wechsler Intelligence Scale for Children—Fourth Edition*. San Antonio, TX: Psychological Corporation.

Willcutt, E. G., Sonuga-Barke, E. J. S., Nigg, J. T., & Sergeant, J. A. (2008). Recent developments in neuropsychological models of childhood psychiatric disorders. *Advances in Biological Psychiatry, 24,* 195–226. doi:10.1159/000118526

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