Are Episodic Buffer Processes Intact in ADHD? Experimental Evidence and Linkage with Hyperactive Behavior

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Abstract

Working memory deficits are present in a substantial proportion of children with ADHD, and converging evidence links these deficits with ADHD-related behavioral and functional impairments. At the same time, working memory is not a unitary construct, and evidence is lacking regarding the role of several components of this system in ADHD. Preclinical behavioral studies are needed to fractionate the multicomponent working memory system, determine which specific subcomponent(s) are impaired in ADHD, and more importantly link these subcomponent(s) with specific ADHD-related behavioral symptoms/functional impairments. The current study reflects one piece of that puzzle, and focuses on the episodic buffer component of working memory. Across multiple testing days, a well-characterized sample of 86 children ages 8–13 (*M*=10.52, *SD*=1.54; 34 girls; 64% Caucasian/Non-Hispanic) with ADHD (*n*=49) and without ADHD (*n*=37) completed three counterbalanced working memory tests that were identical in all aspects except the key subcomponent process (phonological, visuospatial, episodic buffer). Gross motor movement during these and control tasks were measured using 4 high-precision actigraphs. There was no evidence of group differences in gender, age, SES, or IQ. Bayesian mixed-model ANOVAs indicated that the ADHD group performed significantly worse on all three working memory tests (*d*=1.17–1.44) and was significantly more hyperactive than controls (*d*=0.66–1.05) during the visuospatial and episodic buffer tests. In contrast, the ADHD and Non-ADHD groups were equivalent with regard to effects of episodic buffer demands on performance and hyperactive behavior. The most parsimonious conclusion is that the episodic buffer is likely intact in ADHD, and unrelated to ADHD hyperactivity symptoms.

Keywords

ADHD; working memory; episodic buffer; phonological; visuospatial; actigraph

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Working memory deficits are present in a substantial proportion of children with attention-deficit/hyperactivity disorder (ADHD; Kasper et al., 2012), and show strong cross-sectional (Bunford et al., 2014; Kofler et al., 2016) and longitudinal (Halperin et al., 2008; van Lieshout et al., 2016) associations with the disorder's primary behavioral and functional impairments. For example, working memory has been linked with core behavioral symptoms of inattention (Kofler et al., 2010; Gathercole et al., 2008), hyperactivity (Rapport et al., 2009), impulsivity (Raiker et al., 2012), and overall ADHD symptom severity (van Lieshout et al., 2016). In addition, working memory shows strong covariation with ADHD-related impairments in social (Bunford et al., 2015; Tseng & Gau, 2013) and family functioning (Kofler et al., 2016). Academically, working memory deficits appear to explain the link between ADHD and reading problems (Friedman et al., 2017), and account for over 70% of individual differences in children's math computation performance (Swanson & Kim, 2007).

At the same time, working memory is not a unitary construct (Nee et al., 2013; Wager & Smith, 2003), and evidence is lacking regarding the role of several components of this system in ADHD. Preclinical behavioral studies are needed to fractionate the multicomponent working memory system, determine which specific subcomponent(s) are impaired in ADHD, and more importantly link these subcomponent(s) with specific ADHD-related behavioral symptoms and functional impairments (Chacko et al., 2014). For example, working memory deficits have been linked with ADHD-related hyperactive behavior based on subjective ratings, objective observations, and mechanically-measured gross motor movement (for review, see Kofler et al., 2016), but the extent to which this replicated link is attributable to specific subcomponents of working memory is less clear (Hudec et al., 2015). The current study reflects one piece of this puzzle by focusing on the episodic buffer component of working memory (Baddeley et al., 2010). A counterbalanced, experimental design was used to evaluate the extent to which episodic buffer deficits were detectable in ADHD, and probe the extent to which established links between working memory deficits and hyperactive behavior in ADHD (Rapport et al., 2009; Patros et al., 2017) are driven by the episodic buffer component of working memory.

Working memory is a limited capacity, multicomponent system that serves a critical role in planning and guiding everyday behavior (Figure 1; Baddeley, 2012). The *working* component of working memory involves mental processing of internally held information, and is reified across neurocognitive models as the central executive, internal focus of attention, or secondary memory, among other terms (Baddeley, 2012; Cowan, 2011; Unsworth & Engle, 2007). This central executive is distinct from the more general executive functioning construct, and is a supervisory attentional controller responsible for monitoring, processing, reordering, and updating information held in short-term memory (Wager & Smith, 2003). No memory/storage functions are ascribed to the *working* component of working memory; instead, the central executive acts upon information currently held within three short-term storage/rehearsal (short-term memory) components: the *phonological*, *visuospatial*, and *episodic buffer* subsystems.

The episodic buffer is a passive, fragile store in which newly-encoded information gains automatic access and overwrites currently stored information (Rudner & Rönnberg, 2008).

Whereas the phonological and visuospatial subsystems provide this function for single-modality information (verbal and non-verbal, respectively; Nee et al., 2013), the episodic buffer is evoked when information from multiple modalities must be bound and stored as a unitary episode or chunk (e.g., when learning the spatial positions of the phonological letters on a computer keyboard). Additional examples of day-to-day activities likely to rely, at least in part, on the episodic buffer include learning the name of a new child at school (i.e., linking her name [verbal] with her appearance [visual]); giving her directions to the school library (linking the building's name [verbal] with its physical location [spatial]); and integrating what she is saying (verbal) with her body language (non-verbal) to facilitate comprehension.

The Baddeley model (Baddeley, 2000; Baddeley, 2012) posits that the episodic buffer may not be an anatomically distinct store, but rather reflect central executive-mediated, distributed, and synchronous firing of the phonological and visuospatial subsystems. Recent evidence, however, suggests that the episodic buffer may be supported by right frontal/prefrontal and temporoparietal regions (Prabhakaran et al., 2000; Zhang et al., 2004) that have been shown to be anatomically distinct from frontal regions associated with the central executive (Bor & Owen, 2007; Osaka et al. 2004; Rudner & Ronnberg, 2008). Importantly, the episodic buffer's cross-modality binding function does not appear to require central executive/controlled attention processes (Allen et al., 2006). The central executive is also uninvolved in rehearsing information – which is handled in the storage systems (Baddeley et al., 2011) – and its supervisory role in initial encoding and interference control is equivalent regardless of whether target information is phonological, visuospatial, or episodic (Baddeley et al., 2010; 2011). This latter point is critical to the current study's experimental manipulation, because it indicates that performance differences across our three task variants cannot be attributable to differential central executive processes.

Emerging evidence indicates that the episodic buffer is functional in children as young as age four (Alloway et al., 2004) and predicts important social and academic outcomes in preschool (Alloway et al., 2005) and school-aged children (Wang et al., 2015). Despite extensive evidence that working memory in general is associated with objectively-assessed hyperactivity in ADHD (Hudec et al., 2015; Kofler et al., 2016; Patros et al., 2017; Rapport et al., 2009; Sarver et al., 2015), to our knowledge there have been no studies that have fractionated the working memory system to determine whether episodic buffer processes drive this association. More generally, only one study has investigated episodic buffer functioning in children with ADHD. Alderson and colleagues (2015) administered three variants of a phonological working memory task that differed based on how the numbers and letters were presented. In their episodic buffer condition, children simultaneously saw and heard each alphanumeric character (e.g., the symbol "A" appeared on screen as the sound/a/was played via speakers). Relative to auditory-only and visual-only control tasks, the episodic buffer (auditory + visual) task evoked improved performance in the Non-ADHD group but not the ADHD group. Alderson et al. (2015) concluded that ADHD is associated with impaired ability to benefit from bound multimodal information processed by the episodic buffer.

Despite the study's elegant design, significant questions remain regarding episodic buffer functioning in children with ADHD. In particular, Baddeley's model was recently updated to specify that automatic binding of language-based information (e.g., written text + auditory) occurs solely in the phonological short-term store, not the episodic buffer (Baddeley et al., 2011). This distinction between single-modality and cross-modality binding is consistent with fMRI evidence indicating that verbal and visual content may activate similar, midlateral frontal structures (Nee et al., 2013), whereas visual content is disassociated experimentally (Klauer & Zhao, 2004) and structurally (Nee et al., 2013) from spatial content (caudal superior frontal sulcus). In this view, the experimental manipulation used by Alderson and colleagues (2015) may indicate that children with ADHD exhibit intact binding of verbal information within the phonological store, rather than informing cross-modality binding associated with the episodic buffer. Taken together, these findings indicate that more work is needed to isolate cross-modality binding in ADHD, and suggest that the episodic buffer may not be reliably evoked by tasks that combine verbal with visual information (Nobre et al., 2013).

As an alternative to visual-verbal binding, recent studies have evoked the episodic buffer via tasks that require cross-modality binding of verbal and spatial information (Langerock et al., 2014; Morey, 2009). These verbal-spatial cross-modality binding tasks are more aligned with Baddeley's updated model; however, to our knowledge no study to date has examined verbal-spatial binding in children with ADHD. The current study is the first to employ experimental methodology to examine episodic buffer functioning and its impact on hyperactive behavior in children with ADHD. We administered a series of counterbalanced working memory tasks that were identical except for our key independent variable (episodic buffer demands). A novel episodic buffer task was created by combining the well-validated Rapport et al. (2008) phonological and visuospatial working memory tasks. This episodic buffer task requires cross-modality binding of spatial locations with verbal information (numbers and letters). The single-modality (phonological and visuospatial) and cross-modality (episodic buffer) tasks were matched for visual encoding and central executive demands as described above (Baddeley et al., 2011), and were administered in counterbalanced order across multiple testing days to a sample of boys and girls with and without ADHD, matched for the number of non-ADHD disorders.

In addition, all children wore multiple actigraphs and engaged in multiple control tasks to probe for links between episodic buffer deficits and ADHD hyperactive behavior. We expected the ADHD group to show decreased performance (Kasper et al., 2012) and increased hyperactive behavior (Kofler et al., 2016) during all three working memory tests. ADHD-related impairments in the episodic buffer, and the extent to which episodic buffer processes evoke hyperactive behavior in children with ADHD, would be evidenced by significant group x task (single- vs. cross-modality) interactions for the performance and actigraph data, respectively. Given previous findings that the single-modality, phonological and visuospatial stores show small or nonsignificant impairments in ADHD (Kasper et al., 2012), we used Bayesian statistics because they can provide evidence of equivalence rather than just lack of significant differences (Wagenmakers et al., 2016).

Method

Participants

The sample included 86 children aged 8 to 13 years (M = 10.52, SD = 1.54; 52 boys, 34 girls) from the Southeastern United States, recruited by or referred to a university-based children's learning clinic (CLC) through community resources (e.g., pediatricians, community mental health clinics, school system personnel, self-referral) between 2015 and 2017. Psychoeducational evaluations were provided to the parents of all participants. All parents and children gave informed consent/assent, and Florida State University's Institutional Review Board approved the study prior to data collection. Sample ethnicity was mixed with 56 Caucasian Non-Hispanic (64%), 11 Hispanic (13%), 11 African American (13%), 3 Asian (4%), and 5 multiracial children (6%).

Group Assignment

All children and their parents participated in a detailed, semi-structured clinical interview using the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Aged Children (K-SADS; Kaufman et al., 1997). The K-SADS (2013 Update) assesses onset, course, duration, severity, and impairment of current and past episodes of psychopathology in children and adolescents based on DSM-5 criteria (APA, 2013), and was supplemented with parent and teacher ratings from the Behavior Assessment System for Children (BASC-2; Reynolds & Kamphaus, 2004) and Child Symptom Inventory (CSI-IV; Gadow & Sprafkin, 2002).

Children were included in the ADHD group (n=49) based on meeting all three of the following criteria: (1) DSM-5 ADHD diagnosis by the directing clinical psychologist based on K-SADS; (2) parent ratings in the borderline/clinical range on the BASC-2 Attention Problems and/or Hyperactivity scales, or exceeding criterion scores on the parent CSI-IV ADHD-Inattentive and/or ADHD-Hyperactive/Impulsive subscales; and (3) teacher ratings in the borderline/clinical range on the BASC-2 Attention Problems and/or Hyperactivity scales, or exceeding criterion scores on the teacher CSI-IV ADHD-Inattentive and/or ADHD-Hyperactive/Impulsive subscales. All children had current impairment based on K-SADS. Seventeen children with ADHD (35%) were prescribed psychostimulants; medication was withheld for >24 hours before both research sessions.

All ADHD subtypes/presentations were eligible given the instability of ADHD subtypes (Valo & Tannock, 2010). Of the 49 children with ADHD (18 girls), 35 met criteria for Combined, 12 for Inattentive, and 2 for Hyperactive/Impulsive Presentation. To improve generalizability, children with comorbidities were included. Comorbidities reflect clinical consensus best estimates, and included anxiety (31%), oppositional defiant (10%), depressive (8%), and autism spectrum disorders (4%).[1]

[1]The pattern and interpretation of results was unchanged when excluding children with autism spectrum disorder. As recommended in the K-SADS, oppositional defiant disorder was diagnosed clinically only with evidence of multi-informant/multi-setting symptoms. ODD comorbidity is 44% in the ADHD group and 10% in the Non-ADHD group based on parent-reported symptom counts.

The Non-ADHD group comprised 37 consecutive case-control referrals (16 girls) who did not meet ADHD criteria, and included both neurotypical children and children with psychiatric disorders other than ADHD. Diagnoses in this group include anxiety (22%), autism spectrum (8%), depressive (5%), and oppositional defiant disorders (3%).[1] Neurotypical children (62%) had normal developmental histories and nonclinical parent/teacher ratings, were recruited through community resources, and completed the same evaluation as clinically-referred cases. Non-ADHD disorders were included to control for comorbidities in the ADHD group. Importantly, the ADHD and Non-ADHD groups were equivalent in the proportion of children diagnosed with a clinical disorder other than ADHD (omnibus: $BF_{01} = 14.57$, anxiety: $BF_{01} = 3.85$, depression: $BF_{01} = 6.56$, ODD: $BF_{01} = 3.28$, ASD: $BF_{01} = 5.73$).

Learning disabilities were suspected in 21% of ADHD and 3% of Non-ADHD cases based on score(s) >1.5 *SD* below age-norms on the KTEA-3 Reading and/or Math Composites ($BF_{10} = 6.50$; Kaufman & Kaufman, 2014). Children were excluded if they presented with gross neurological, sensory, or motor impairment; history of seizure disorder, psychosis, or intellectual disability; or non-stimulant medications that could not be withheld for testing.

Procedure

Neurocognitive testing occurred as part of a larger battery that involved two sessions of approximately 3 hours each. All tasks were counterbalanced across sessions to minimize order effects. Children received brief breaks after each task, and preset longer breaks every 2–3 tasks to minimize fatigue. Children were seated in a caster-wheel swivel chair. Performance was monitored at all times by the examiner, who was stationed just outside of the testing room (out of the child's view) to provide a structured setting while minimizing performance improvements associated with examiner demand characteristics (Gomez & Sanson, 1994).

Working Memory Performance

The working memory tasks developed by Rapport et al. (2008) were used for the current study, along with a novel episodic buffer working memory task created by combining the Rapport single-modality tasks to require cross-modality binding of phonological and visuospatial information (Baddeley et al., 2010). Performance on the single-modality control tasks was reported for a subset of the current sample in Kofler et al. (2017). These tasks correctly classify children with vs. without ADHD at similar rates as parent and teacher ADHD rating scales (Tarle et al., 2017), and predict ADHD-related impairments in objectively-measured activity level (Rapport et al., 2009), attentive behavior (Kofler et al., 2010), impulsivity (Raiker et al., 2012), delay aversion (Patros et al., 2015), inhibitory control (Alderson et al., 2010), reading comprehension (Friedman et al., 2017), and academic, peer, and family dysfunction (Kofler et al., 2011, 2016). Reliability and validity evidence includes high internal consistency ($\alpha = 0.82$ to 0.97), 1–3 week test-retest reliability of .76 to .90 (Sarver et al., 2015), and strong associations with working memory complex span tasks ($r = .69$; Wells et al. (2015).

All three tasks involved serial reordering of characters presented (numbers, black dot locations), and reordering of a target stimulus (letter, red dot location) into the final serial position recalled. The target stimulus was never presented in the first or last position of the sequence to minimize 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). Stimuli are presented at a rate of 1 per second (800 ms presentation, 200 ms ISI).

Trials were presented in two, 12-trial blocks of mixed set sizes, with 6 unique trials of each memory load (set sizes 3–6 per block; 24 total trials). Short breaks were provided between each block (approximately 1 minute). Five practice trials were administered before each task (80% correct required). Mixed presentation was selected because it evokes higher working memory demands than sequential presentation (Kofler et al., 2016). Task duration was approximately 2.8 (visuospatial), 3.0 (episodic buffer), and 3.4 (phonological) minutes per block. Partial-credit unit scoring was used at each set size for each task (Conway et al., 2005).

Phonological working memory task (PHWM)—This task is similar to WISC-V Letter-Number Sequencing (Wechsler, 2014), and assesses phonological working memory based on Baddeley's (2012) model. Children were presented a series of numbers and a letter that never appeared first or last. A subset of letters were selected to minimize phonological and visual similarity effects (Baddeley et al., 2011). Children were instructed to recall the numbers in order from least to greatest, and to say the letter last (e.g., 4-H-6-2 is correctly recalled as 2-4-6-H). Visual presentation was selected to equate encoding demands across all three experimental tasks. Evidence indicates that visual-to-phonological conversion processes do not mediate ADHD-related impairments on this task (Alderson et al., 2015; Raiker et al., in press), and visual and auditory presentations produce highly similar latent estimates of ADHD-related working memory deficits (d=0.91 vs. 1.12; Alderson et al., 2015). Two trained research assistants, shielded from the participant's view, recorded oral responses independently (interrater reliability was 97.33%).

Visuospatial working memory task (VSWM)—Children were shown nine squares arranged in three offset vertical columns on a computer monitor. The columns were offset from a standard 3×3 grid to minimize the likelihood of phonological coding of the stimuli. A series of 2.5 cm diameter dots (3, 4, 5, or 6) were presented sequentially. No two dots appeared in the same square on a given trial. All dots were black except for one red dot that never appeared first or last. Children were instructed to re-order the dot sequence by keying the spatial locations on a modified keyboard, with the black dot locations in the serial order presented, followed by the red dot's location last.

Episodic Buffer Working Memory (EBWM)—The novel episodic buffer working memory task combined the phonological and visuospatial tasks (Figure 2). Task parameters were identical to the single-modality tasks. Children were presented a series of numbers and a letter that appeared in the visuospatial squares described above; no two stimuli appeared in the same square on a given trial, and the letter never appeared last. Children were instructed to remember the spatial location of each number/letter, reorder the numbers in ascending order and put the letter last (e.g., 4-H-6-2 is correctly recalled as 2-4-6-H), and respond by

keying the corresponding squares on a modified keyboard. Thus, successful performance on the task evoked the episodic buffer by requiring children to bind the phonological (numbers and letter) with the visuospatial (location each number/letter appeared) information.

Intellectual Functioning (IQ)

All children were administered the WISC-V Verbal Comprehension Index (Wechsler, 2014) to obtain an estimate of intellectual functioning.

Socioeconomic Status

Socioeconomic status (SES) was estimated using the Hollingshead (1975) scoring based on caregiver(s)' education and occupation.

Control Conditions

Children used Microsoft® Paint for five consecutive minutes at the beginning (C1) and end (C2) of both research sessions. The Paint program served as pre- and post-conditions to assess and control for potential within-day fluctuations in activity level (e.g., fatigue effects). Children sat in the same chair and interacted with the same computer used for the working memory tasks while interacting with a program that placed relatively modest demands on working memory (i.e., the Paint program allows children to draw/paint anything they like on the monitor using a variety of interactive tools[2]). Following Rapport et al. (2009), the two C1 and two C2 control conditions were separately averaged to create pre- and post-composite scores.

Activity Level

Actigraphs—Basic Motionlogger® (Ambulatory Monitoring, Inc., 2014) actigraphs are acceleration-sensitive devices that sample movement intensity 16 times per second (16 Hz), collapsed into 1-second epochs. The estimated reliability for actigraphs placed at the same site on the same person ranges from .90 to .99 (Tryon et al., 1991). Children were told that the actigraphs were "special watches" that let them play the computer learning games. Observer XT (Noldus, 2014) software was used to code start and stop times for each task, which were matched to the time stamps from the actigraphs. Actigraphs were placed on the child's non-dominant wrist and both ankles using Velcro watch bands. Different from previous studies, a fourth actigraph was attached to the back of the 4-caster swivel chair to capture gross motor movement not collected by children's extremities.

Dependent variables: Activity level—Following Rapport et al. (2009), we computed Total Movement Level (TML) by summing activity level across the four actigraph sites (2 ankle, 1 non-dominant hand, 1 chair) to index total movement for each task. Separate TML scores were computed for the two consecutive blocks of each WM task, and the C1 and C2 control conditions, to probe for time-on-task effects. This approach has the additional advantage of increasing power while providing a broader sampling of children's activity level (Rapport et al., 2009).

[2]As noted by Rapport et al. (2009), successful interaction with the Paint program requires working memory processes such as focused attention and interaction with long-term memory, as well as limited storage/rehearsal processes.

Data Analysis Overview

A two-tiered data analytic approach was employed to examine the study's primary hypotheses. Tier I probed for episodic buffer deficits in ADHD using two, complementary analyses. Between-group performance differences by task were probed initially (Bayesian mixed-model ANOVA), followed by intra-individual analyses that compared each child's lowest single-modality performance (visuospatial or phonological) with their cross-modality performance (episodic buffer) as a function of group (ADHD, Non-ADHD). These two approaches are based on disparate assumptions regarding how the episodic buffer functions neurologically. The first analysis is consistent with evidence that the episodic buffer is a separate short-term store (Rudner & Rönnberg, 2008); thus, the phonological and visuospatial tasks serve as robust controls for stimulus encoding, central executive processing, and response output. The second analysis is based on the synchronous firing hypothesis (Baddeley, 2012); in this case, deficits in the phonological or visuospatial subsystems would cap performance on the cross-modality task.

Tier II examined the impact of increasing episodic buffer demands on actigraph-measured gross motor activity as a function of group (ADHD, Non-ADHD), task (Control, PHWM, VSWM, EBWM), and task block (WM: 1, 2, C: Pre, Post); task block was included to probe and control for time-on-task effects.

Bayesian Analyses

The benefits of Bayesian methods over null hypothesis significance testing (NHST) are well documented (Rouder & Morey, 2012; Wagenmakers et al., 2016); for our purposes, Bayesian analyses were selected because they allow stronger conclusions by estimating the magnitude of support for both the alternative and null hypotheses (Rouder & Morey, 2012). Bayes factor mixed-model ANOVAs with JZS default prior scales (Rouder & Morey, 2012; Wagenmakers et al., 2016) were conducted using JASP 0.8.1 (JASP Team, 2017). Instead of a *p*-value, these analyses provide BF_{10}, which is the Bayes Factor of the alternative hypothesis (H_1) against the null hypothesis (H_0). BF_{10} is an odds ratio, where values above 3.0 are considered moderate evidence supporting the alternative hypothesis (conceptual equivalent of $p < .05$). BF_{10} values above 10.0 are considered strong (>30 = very strong, >100 = decisive/extreme support; Wagenmakers et al., 2016).

Conversely, BF_{01} is the Bayes Factor of the null hypothesis (H_0) against the alternative hypothesis (H_1). BF_{01} is the inverse of BF_{10} (i.e., $BF_{01} = 1/BF_{10}$), and is reported when the evidence indicates a lack of an effect (i.e., favors the null hypothesis; Rouder & Morey, 2012). BF_{01} values are interpreted identically to BF_{10} (>3.0 = moderate, >10.0 = strong, >100 = decisive/extreme support for the null hypothesis that a predictor does *not* have an effect on an outcome; Rouder & Morey, 2012).

Thus, finding $BF_{10} = 10.0$ would indicate that the observed data are 10 times more likely under the alternative hypothesis model (e.g., strong evidence for deficits in the ADHD vs. Non-ADHD group), whereas $BF_{01} = 10$ would indicate that the observed data are 10 times more likely under the null hypothesis model (e.g., strong evidence that the ADHD and Non-ADHD groups are equivalent). Between-group comparisons are supplemented with Cohen's

d effect sizes. Between-group *p*-values are shown in Table 1 for comparison; interpretation of results is unchanged if NHST is used instead of Bayesian analyses (except that non-significant *p*-values cannot be interpreted as evidence of equivalence).

Results

Bayesian Power Analysis

A simulation study was conducted to estimate power for between-group tests using the R BayesFactor package and BayesianPowerTtest script (Lakens, 2016) optimized by Zimmerman (2016), with parameters as follows (*N*=86; r-scale=1; *k*=100,000 simulated experiments; BF threshold=3.0). Results indicated power=.81 for supporting the alternative hypothesis based on a true effect of *d*=0.74 (meta-analytic estimate for ADHD/Non-ADHD working memory differences from Kasper et al., 2012; 81% of simulations correctly supported H_1 at $BF_{10} > 3.0$, 18% provided equivocal support at BF_{10} values between 1/3 and 3, and only 1% incorrectly supported H_0). Notably, we have power=1.0 for detecting the expected effect size of 1.60, which is the mean effect size reported across 9 prior studies using the current study's Rapport PHWM and VSWM tasks to compare ADHD and Non-ADHD groups (cited above). Power=.78 for supporting the null if true (i.e., for *d*=0.0; 78% of simulations supported H_0, 21% provided equivocal support, and only 1% incorrectly supported H_1). Power analysis based on traditional NHST for *t*-tests (G-Power 3.1; Faul et al., 2007), with two-tailed alpha=.05 and power=.80, indicates that our *N*=86 can reliably reject the null hypothesis for effects of *d*=0.61 or higher.

To our knowledge, Bayesian power analysis for repeated-measures ANOVA is not yet available. Power analysis based on traditional NHST, with alpha=.05, power=.80, and 3 measurements (PHWM, VSWM, EBWM) indicates that our *N*=86 can reliably detect interaction effects of *d*=0.28 or larger. Interactions of *d*=0.31 are detectable for post-hoc tests that include 2 measurements. Thus, the study is sufficiently powered to address its primary aims.

Preliminary Analyses

Outliers beyond 3 *SD* were windsorized relative to the within-group mean (ADHD, Non-ADHD). This process affected 1.34% of data points. All parent and teacher ADHD rating scale scores were higher for the ADHD relative to Non-ADHD group as expected (Table 1). In contrast, there was no evidence to indicate between-group differences in age (BF_{10} = 0.49), IQ (BF_{10} = 0.79), or SES (BF_{10} = 0.60); we therefore report simple model results with no covariates.

Tier I: Phonological, Visuospatial, and Episodic Buffer Working Memory Performance

Functionally-Distinct Episodic Buffer Model—The 2 (group: ADHD, Non-ADHD) x 3 (task: PHWM, VSWM, EBWM) x 4 (set size: 3–6) Bayesian mixed-model ANOVA indicated decisive evidence supporting main effects of group, task, and set size (all $BF_{10} > 100$). With reference to the main effects model, the evidence also decisively supported the addition of the group x task interaction (BF_{10} = 92.50). There was decisive evidence for

very large ADHD-related deficits on the phonological (d = 1.17), visuospatial (d = 1.44), and episodic buffer (d = 1.22) working memory tasks (all BF_{10} > 100).

Importantly, the group x task interaction was attributable to differential group effects across the single-modality tasks, but not the addition of episodic buffer demands (Figure 3, top left). That is, controlling for main effects, there was decisive evidence for disproportionate, ADHD-related performance decrements on the visuospatial relative to phonological task (BF_{10} > 100). In contrast, there was significant support for the null hypothesis of equivalent task-related changes for both groups across the visuospatial and episodic buffer tasks (BF_{01} = 6.67), despite main effects indicating decisive differences for both task and group (both BF_{10} > 100). Taken together, these findings indicate that adding episodic buffer demands resulted in decreased accuracy for both groups, but this manipulation did not differentially affect the ADHD group. Instead, the ADHD group demonstrated similarly large deficits on all three tasks (d = 1.17–1.44). Thus, their performance deficits on the episodic buffer task appear more parsimoniously explained by their overall working memory deficits, rather than a unique impairment in the episodic buffer.

Synchronous Firing Model—We selected each child's lowest single-modality performance (collapsed across set sizes), which occurred on the visuospatial task for 89.8% of the ADHD group (44 of 49) and 86.5% of the Non-ADHD group (32 of 37) (BF_{01}=5.13). The 2 group (ADHD, Non-ADHD) x 2 task (Episodic Buffer vs. Lowest Single Modality) Bayesian mixed-model ANOVA revealed decisive support for effects of group and task (BF_{10} > 100). In contrast, there was significant evidence against a group x task interaction (BF_{01} = 3.35). In other words, the ADHD group had lower scores on the single-modality (d = 1.45) and episodic buffer (d = 1.22) conditions, and both groups recalled somewhat fewer stimuli during the episodic buffer (M = 2.43, SD = 0.86) relative to the single-modality condition (M = 2.65, SD = 0.87). However, the addition of episodic buffer demands effected children with ADHD and Non-ADHD children equivalently.

Tier II: Effects of Increasing Episodic Buffer Demands on Actigraph-Measured Hyperactivity

The 2 group x 4 task (Control, PHWM, VSWM, EBWM) x 2 block Bayesian mixed-model ANOVA revealed strong evidence favoring main effects of group (BF_{10} = 59.29) and task (BF_{10} > 100), but no significant evidence of differences across blocks (BF_{01} = 2.50). Accounting for main effects, the evidence also strongly supported the addition of the group x task interaction (BF_{10} = 71.67). Probing the interaction revealed decisive evidence for higher ADHD-related activity level during the visuospatial task (d = 1.05, BF_{10} > 100), and moderate evidence during the episodic buffer task (d = 0.66, BF_{10} = 11.64), but no significant evidence of group differences during the phonological (d = 0.41, BF_{10} = 1.07) or control conditions (d = 0.46, BF_{10} = 1.55).

Importantly, the group x task interaction was attributable to differential group effects between the control and the visuospatial working memory task (BF_{10} > 100). There was no significant evidence for a group x task interaction between the control and phonological (BF_{01} = 1.96) or control and episodic buffer tasks (BF_{10} = 2.62), indicating that both groups

showed similar increases during the working memory tasks with a verbal component (PHWM, EBWM). There was significant evidence against an interaction between the phonological and episodic buffer tasks (BF_{01} = 6.21).

Evidence for a group x task interaction was strong between the visuospatial and episodic buffer tasks (BF_{10} = 11.17), but not in the direction that would link episodic buffer demands with ADHD-related hyperactivity (Figure 3, bottom). That is, the interaction was due to significant *decreases* in hyperactivity for the ADHD group under the EBWM condition (BF_{10} = 5.50), whereas the non-ADHD group did not change significantly (BF_{01} = 1.59)

Collectively, both groups exhibited strong evidence for higher activity level during the three working memory tasks relative to the control conditions (all BF_{10} > 100); however, between-group differences during the episodic buffer task were similar to or smaller than those observed during the single modality PH and VS tasks, respectively. In addition, the ADHD group showed significant *decreases* in hyperactivity during the episodic buffer relative to visuospatial task, and both groups showed decreased hyperactivity during the episodic buffer relative to phonological task. This pattern suggests that group differences in hyperactivity are more parsimoniously attributable to shared working memory demands in general, and visuospatial working memory demands in particular, rather than increased episodic buffer demands. Taken together, experimentally increasing episodic buffer demands failed to evoke greater deficits in working memory performance or objectively-measured hyperactivity for children with ADHD.

Discussion

The current study was the first to experimentally examine episodic buffer functioning in children with ADHD, using a verbal and spatial binding task. Of primary interest in the current study was the extent to which the episodic buffer is impaired in ADHD and/or exerts a causal role in ADHD-related hyperactive behavior. Overall, the current findings are consistent with large magnitude working memory deficits in children with ADHD (Kasper et al., 2012), but indicate that the episodic buffer is likely intact in these children (Alderson et al., 2015). Additionally, strong evidence of performance decrements on the episodic buffer relative to visuospatial/phonological tasks were found, indicating that episodic buffer performance was not merely an artifact of each child's lowest single-modality task performance. Overall, these findings provide support for a distinct episodic buffer in middle childhood (Alloway et al., 2004).

The current study was the first to experimentally investigate episodic buffer functioning and examine the extent to which this working memory component evokes hyperactive behavior in children with ADHD. Overall, children with ADHD exhibited large magnitude deficits across all three working memory tasks (*d* = 1.17–1.44). These findings were consistent with meta-analytic best-case estimates (Kasper et al., 2012), and extend previous findings by demonstrating that effect sizes remain very large even when controlling for comorbidities. Taken together, this pattern of results provides additional data confirming that pediatric ADHD is associated with large magnitude deficits in working memory when measured using tests with prominent central executive demands (Kasper et al., 2012).

Of primary interest in the current study was the extent to which the episodic buffer, a critical yet understudied component of the working memory system (Baddeley et al., 2011), is impaired in ADHD and/or exerts a causal role in ADHD-related hyperactive behavior. Studies fractionating working memory subcomponents are critical given the disappointing but replicated finding that extant medications do not actuate cortical networks associated with working memory (Rubia et al., 2014) and that 'working memory' training programs fail to sufficiently target working memory for children with ADHD (Rapport et al., 2013). Our experimental manipulation was successful in evoking episodic buffer demands, as evidenced by similar performance decrements for both ADHD and Non-ADHD children. These main effects of episodic buffer demands were detected consistently, despite disparate theoretical assumptions regarding episodic buffer functioning when comparing separate modalities (EB vs. PH, VS) and cross-modality (episodic buffer) vs. single-modality performance. Administering tasks that were identical in all respects except for the key process (episodic buffer demands) allowed stronger conclusions regarding episodic buffer functioning in ADHD, particularly when combined with Bayesian analyses that allowed conclusions regarding between-group equivalence.

Intact Episodic Buffer Functioning in ADHD

Taken together, the preponderance of evidence indicates that the episodic buffer is likely intact in ADHD, based on no interaction effects favoring ADHD-related decrements and moderate-to-strong evidence supporting equivalence (Figure 3, top). The significant evidence *against* episodic buffer deficits in ADHD was unexpected given large between-group differences in performance on the episodic buffer task (d=1.2), as well as evidence of ADHD-related underdevelopment/hypoactivation (Shaw et al., 2007; Smith et al., 2006) in right frontal/prefrontal and temporoparietal regions thought to support cross-modality binding in adults (Prabhakaran et al., 2000; Zhang et al., 2004). In contrast, the findings were consistent with overall large-magnitude working memory deficits in ADHD when using tasks with a prominent executive component (Kasper et al., 2012), as well as emerging evidence of strong convergence between episodic buffer and visuospatial functioning in children (Gray et al., 2017). Thus, a parsimonious explanation for our findings is that ADHD-related deficits on the episodic buffer task are driven primarily by their overall working memory deficits, as well as by their underdeveloped visuospatial storage/rehearsal system (Kasper et al., 2012). That is, between-group differences increased disproportionately across the phonological to visuospatial tasks, but did not increase further when children had to bind phonological and visuospatial stimuli.

Further, examination of the actigraph data indicates significant support for between-group equivalence with regard to episodic buffer effects on gross motor movement, based on interaction effects that favor the null (PH vs. EB) or show *decreases* for the ADHD group (VS vs. EB). That is, the ADHD group was significantly less hyperactive during the episodic buffer task relative to the visuospatial working memory task. These findings are consistent with meta-analytic evidence that ADHD-related hyperactive behavior is most pronounced during laboratory-based executive functioning tests (Kofler et al., 2016), and extends these findings by demonstrating this association in an experimental manipulation that provides stronger evidence of causality. Notably, however, the evidence indicated strongly that this

increased hyperactive behavior could not be attributed specifically to episodic buffer processes, but was more parsimoniously explained by general working memory demands common across the three tasks (Rapport et al., 2009).

Assessing Functional Linkages Between Neurocognition and ADHD Behavioral Symptoms

More generally, our results are consistent with replicated evidence that both ADHD and Non-ADHD children show higher gross motor movement when environmental demands challenge working memory (Kofler et al., 2016), and extend these findings by demonstrating this effect across multiple tasks counterbalanced across testing days. Interestingly, both ADHD and Non-ADHD groups demonstrated decisive evidence of increased gross motor movement during working memory tasks relative to less cognitively demanding tasks that were otherwise equivalent in terms of setting, technology, and environmental stimulation (Kofler et al., 2016). A potential explanation may be that, regardless of diagnostic status, most children upregulate their gross motor activity to augment psychophysiological arousal in response to task demands that tax their working memory system (Sarver et al., 2015). The differential increases for children with ADHD may be a byproduct of their underdeveloped cortical structures that support working memory (Shaw et al., 2007). That is, this functional response may not be triggered for children with neurotypical working memory when classroom and other environmental demands are congruent with their age-normative neurocognitive abilities (Kofler et al., 2016).

In contrast, children with ADHD may need to increase their motor movement more frequently because day-to-day expectations challenge their underdeveloped working memory abilities (i.e., executive processing demands that are chronologically but not developmentally appropriate). This hypothesis is consistent with the strong associations between working memory and myriad academic and functional skills (e.g., Jaroslawska et al., 2016; Swanson & Kim, 2007), evidence linking underdeveloped working memory with ADHD-related academic, social, and family impairments (Bunford et al., 2015; Friedman et al., 2016; Kofler et al., 2016), and positive associations between gross motor activity and cognitive task performance for children with ADHD (Hartanto et al., 2016; Sarver et al., 2015). Taken together, these findings provide strong support for etiological models that propose a functional role for hyperactive gross motor movement in ADHD (Rapport et al., 2009).

The Episodic Buffer: Synchronous Firing or Separate Short-Term Store?

Combined with results of a recent study (Gray et al., 2017), the current findings appear well-suited to address the extent to which the episodic buffer is a functionally distinct store (Rudner & Rönnberg, 2008) as opposed to the product of CE-mediated, synchronous firing of the visuospatial and phonological storage systems (Baddeley, 2000; Baddeley et al., 2011; 2012). Recently published evidence suggests that the episodic buffer and visuospatial sketchpad are empirically indistinguishable in 7–9-year-old children (Gray et al., 2017). In light of these findings, it remains possible that previous evidence of a distinct episodic buffer in 4–6-year-old children may be related to the exclusion of visuospatial tasks in that study (Alloway et al., 2004). Alternatively, the tasks used by Gray et al., (2017) to assess the episodic buffer merit scrutiny regarding the extent to which they evoke cross-modality

binding as defined in Baddeley's updated model (Baddeley et al., 2011). The current findings add to this conflicting literature, and provide support for a distinct episodic buffer in middle childhood. That is, we found strong evidence of performance decrements on the episodic buffer relative to visuospatial and phonological tasks, with a performance pattern that could not be explained by the synchronous firing hypothesis (i.e., episodic buffer performance was not merely an artifact of each child's lowest single-modality task performance). Nonetheless, additional work is clearly needed to clarify the episodic buffer's development across childhood.

Limitations

The unique contribution of the current study was its experimental investigation of episodic buffer functioning in ADHD, and its relation with objectively-measured hyperactive behavior, in a well-characterized sample of children with and without ADHD. Several caveats merit consideration when interpreting the present findings despite these and other methodological refinements (e.g., Bayesian modeling, control for ADHD-related comorbidities). Independent experimental replications with larger samples and ecologically-valid outcomes are needed to assess the extent to which these findings account for ADHD-related functioning in naturalistic settings. In addition, Bayesian methods provided increased confidence for interpreting null findings as evidence of equivalence; however, it remains possible that the ADHD group's large magnitude visuospatial working memory deficits may have masked episodic buffer deficits that may be evident when binding and processing other cross-modality information (Gray et al., 2017). This explanation is unlikely given significant evidence of VS-to-EB performance decrements for both groups, but merits consideration in future studies of cross-modality binding.

Further, our experimental manipulation assessed episodic buffer functioning within the context of tasks with prominent executive components. Thus, although executive processing demands were equivalent across tasks (Baddeley et al., 2011), it remains possible that ADHD-related decrements would be detected on tasks that require cross-modality binding without active processing of the bound information. Similarly, despite theoretical models suggesting a single episodic buffer (Baddeley et al., 2011), it remains possible that cross-modality binding deficits may be detected for other combinations of stimuli (e.g., spatial, color, tactile, auditory). Finally, the lack of a group x task interaction between the visuospatial and episodic buffer tasks may be related to the former's requirement that children bind visual (color) and spatial (location) information. Although the binding of visual and spatial information occurs in the visuospatial short-term store rather than episodic buffer (Baddeley et al., 2011), this between-task difference could have influenced results to the extent that children with ADHD demonstrate differential impairments on visual relative to spatial memory.

Clinical and Research Implications

Collectively, current and past findings indicate that working memory deficits are a robust predictor of ADHD-related behavioral symptoms and functional impairments both cross-sectionally and longitudinally (Friedman et al., 2016; Kofler et al., 2016; van Lieshout et al., 2016). Studies that fractionate the multicomponent working memory system are needed to

clarify the nature of these associations, toward identification of viable intervention targets within an experimental therapeutics framework. The current study reflects one piece of a body of preclinical research needed to understand the neurocognitive origins of ADHD cognitive impairments and their role in the disorder's symptom expression (Chacko et al., 2014).

Updated conceptualizations of human cognition and working memory point to the existence of a multimodal episodic buffer (Baddeley et al., 2011) that may be present in children as young as age 4 (Alloway et al, 2004). At the same time, the current results are consistent with those of Alderson et al. (2015) in indicating that lower-level binding processes are likely intact in children with ADHD, as well as recent findings suggesting strong overlap between visuospatial and episodic buffer functioning in children (Gray et al., 2017). Further, the current results confirm the link between working memory and hyperactivity in ADHD (Patros et al., 2017; Rapport et al., 2009), but provide strong evidence that this link is not attributable to episodic buffer processes. Taken together, these findings suggest that targeting the episodic buffer is unlikely to yield an efficient method of improving ADHD symptoms (Chacko et al., 2014), and that this major new area of human cognition is unlikely to contribute to the search for ADHD endo-/intermediate-phenotypes. In contrast, the current findings suggest that episodic buffer functioning may reflect the first known area of frontal/prefrontal functioning in which ADHD is associated with resiliency (Lerner et al., 2009). Future work is needed to determine whether this area of relative strength can be harnessed to accommodate deficits in other aspects of neurocognitive functioning for these children (Raiker et al., in press).

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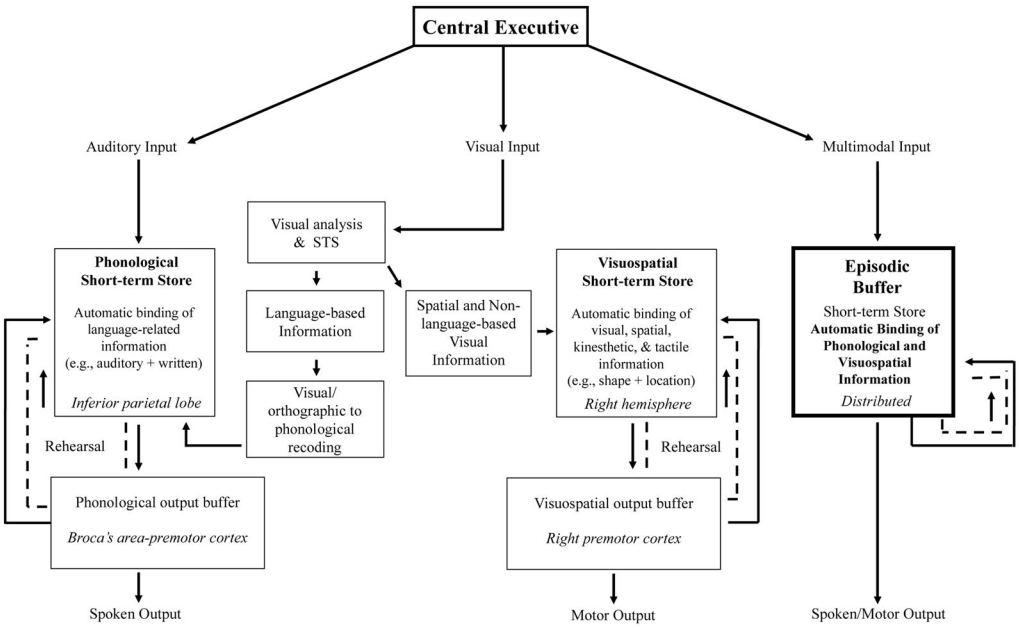

Figure 1.

Adapted and expanded version of Baddeley's (2011) working memory model and associated anatomical loci. Adapted and expanded from Rapport et al. (2008) with permission from both authors.

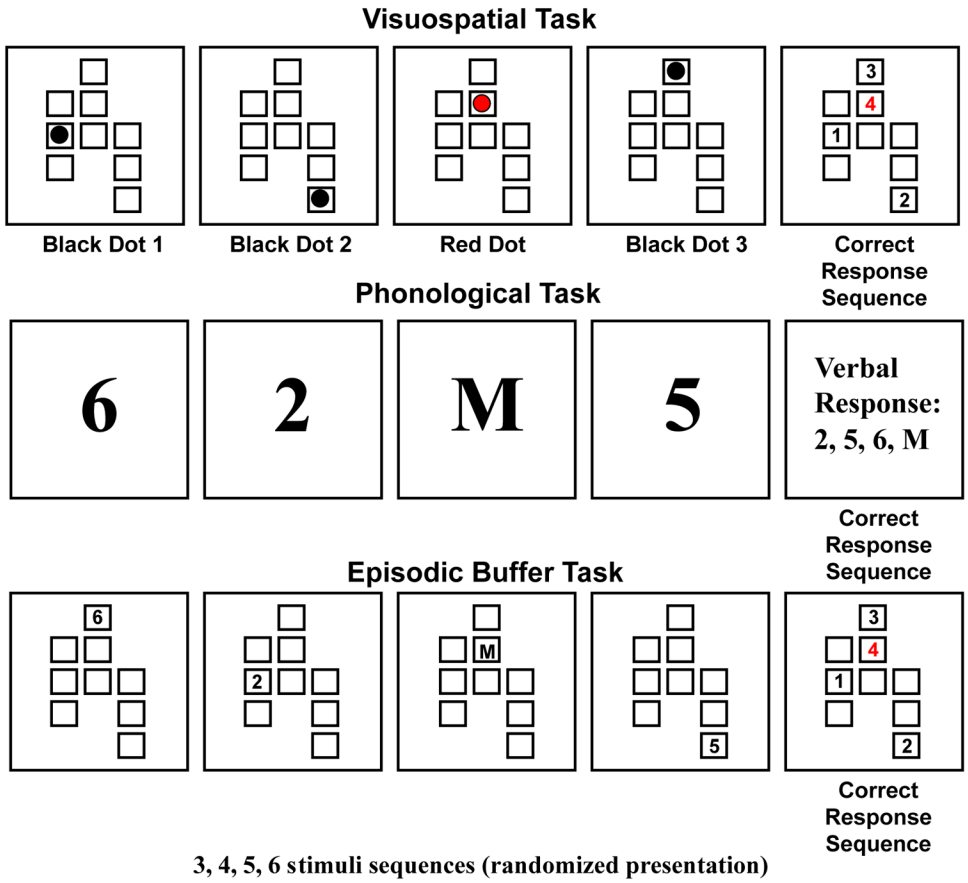

Figure 2.

Visual schematic of the visuospatial (top), phonological (middle), and episodic buffer (bottom) working memory tasks. For demonstration purposes, the episodic buffer example uses the same stimuli as the phonological example and the same response location order as the visuospatial example.

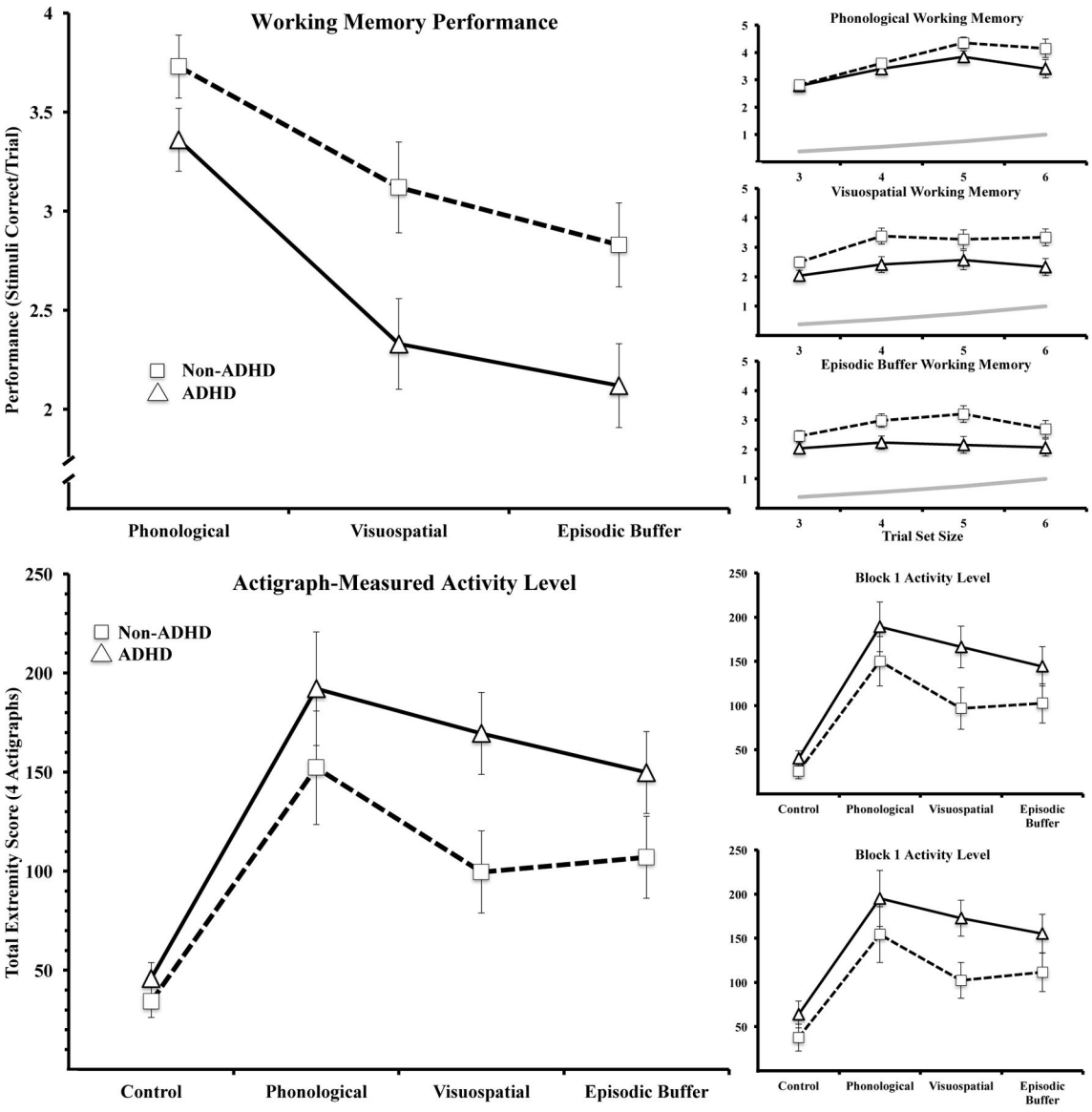

Figure 3.

Performance (top left) and actigraph-measured activity level (bottom left) during the experimental conditions. Insets depict performance x set size for each of the three working memory task variants (top right) and activity level x block (bottom right). Error bars reflect 95% confidence intervals. Grey bars in the performance insets (top right) reflect chance responding.

Table 1

Sample and Demographic Variables

Variable	ADHD (*n*=49)		Non-ADHD (*n*=37)		Cohen's *d*	BF_{10}	*p*
	M	*SD*	*M*	*SD*			
Gender (Girls/Boys)	18/31		16/21		--	0.31	.54, *ns*
Age	10.33	1.47	10.77	1.61	−0.29	0.49	.19, *ns*
SES	46.95	12.24	50.81	11.30	−0.33	0.60	.14, *ns*
WISC-V VCI	103.69	14.67	108.51	10.54	−0.37	0.79	.09, *ns*
BASC-2 Attention Problems (T-score)							
Parent	65.61	8.29	58.92	10.90	0.71	18.89	.002
Teacher	62.22	8.73	55.24	11.37	0.70	18.27	.002
BASC-2 Hyperactivity (T-score)							
Parent	67.02	12.68	56.32	13.20	0.83	93.96	< .001
Teacher	61.14	14.52	53.57	13.05	0.55	3.31	.01
Working Memory Performance Data (Stimuli Correct/Trial)							
Phonological Working Memory	3.27	0.54	3.85	0.42	−1.17	>100	< .001
PH 3	2.78	0.28	2.81	0.26	−0.09	0.25	.67, *ns*
PH 4	3.40	0.57	3.61	0.41	−0.41	1.04	.07, *ns*
PH 5	3.84	0.76	4.35	0.67	−0.70	17.77	.002
PH 6	3.41	1.21	4.15	1.14	−0.63	8.07	.005
Visuospatial Working Memory	2.22	0.76	3.27	0.68	−1.44	>100	< .001
VS 3	2.03	0.64	2.50	0.39	−0.84	>100	< .001
VS 4	2.41	0.97	3.38	0.59	−1.17	>100	< .001
VS 5	2.56	1.16	3.27	1.06	−0.63	7.83	.005
VS 6	2.33	1.02	3.33	1.40	−0.84	>100	< .001
Episodic Buffer Working Memory	2.04	0.71	2.94	0.77	−1.22	>100	< .001
EB 3	2.03	0.68	2.45	0.55	−0.67	11.95	.003
EB 4	2.23	0.80	2.98	0.85	−0.91	>100	< .001
EB 5	2.15	1.02	3.20	1.17	−0.97	>100	< .001
EB 6	2.07	1.04	2.69	1.31	−0.54	3.23	.02
Actigraph Total Movement Level (PIM)							
Baseline 1	40.02	30.15	25.50	18.00	0.57	4.38	.01
Baseline 2	63.78	54.10	37.58	31.30	0.57	4.37	.01
PH	192.09	102.23	152.21	88.61	0.41	1.07	.06, *ns*
VS	169.49	73.92	99.61	55.10	1.05	>100	< .001
EB	149.79	73.88	107.00	49.23	0.66	11.64	.003

Note. Between-group comparisons favoring the null are shown in grey font. BF_{01} can be computed as the inverse of BF_{10} ($1/BF_{10}$). BASC-2 = Behavior Assessment System for Children (T-scores); BF = Bayes Factor; EB = Episodic Buffer Working Memory (Stimuli Correct/Trial); PH = Phonological Working Memory (Stimuli Correct/Trial); PIM = Proportional Integrating Measure; VS = Visuospatial Working Memory (Stimuli Correct/Trial); VCI = WISC-V Verbal Comprehension Index (IQ; standard scores).

* $p \leq .05$,

** $p \leq .01$,

*** $p \leq .001$