Is hyperactivity in children with ADHD a functional response to demands on specific executive functions or cognitive demands in general?

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Abstract

Objective: Hyperactivity is a core and impairing deficit in the clinical model of attention-deficit/hyperactivity disorder (ADHD). However, the extent to which hyperactivity in ADHD is evoked by cognitively challenging tasks in general or by demands on specific executive functions remains unclear.

Method: A clinically evaluated and carefully phenotyped community-referred sample of 184 children ages 8–13 (M=10.40, SD=1.50; 61 girls) with ADHD (n=119) and without ADHD (neurotypical children and children with psychiatric disorders other than ADHD) were administered multiple, counterbalanced executive (working memory, inhibitory control, set shifting) and non-executive tests. Objective measures of gross-motor movement (hyperactivity) were obtained using actigraphy.

Results: Using bifactor s-1 modeling, results indicate that children with ADHD demonstrate moderately elevated levels of motor movement relative to Non-ADHD children. Additionally, findings indicated that hyperactivity in ADHD reflects the outcome of at least two similarly important factors: (a) a baseline level of elevated motor movement that is independent of environmental demands on their executive and non-executive cognitive abilities (d=0.72), and (b) additional elevations attributable to demands placed on specific executive functions, with working memory and inhibition demands evoking similarly large, differential increases in movement for children with ADHD above and beyond their elevated baselines (Δd=0.80).

Conclusion: These findings suggests that executive function demands exacerbate but do not fully explain hyperactivity in ADHD and/or there are at least two pathways to hyperactivity in ADHD – hyperactivity caused by environmental demands that challenge their underdeveloped

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Conflict of Interest:
The authors have no conflicts of interest to report.

Ethical Approval:
All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent:
Informed consent was obtained from all individual participants included in the study.

executive functions, and hyperactivity caused by one or more other factors that need future research to identify.

Keywords

Executive function; ADHD; hyperactivity; actigraphy

Hyperactivity, a core and impairing deficit in the clinical model of attention-deficit/hyperactivity disorder (ADHD), is characterized by excess physical movement/motor activity and/or excessive talking (APA, 2013). Meta-analytic evidence indicates that children with ADHD demonstrate elevated motor activity (i.e., hyperactivity) compared to children without ADHD, although the extent to which this excess motor movement is ubiquitous as opposed to a functional and/or adaptive response to environmental demands that challenge these children's neurocognitive vulnerabilities remains unclear (for review, see Kofler et al., 2016). Contemporary models suggest that this increased motor activity may serve a compensatory function that increases cortical arousal during cognitively demanding activities in general (e.g., Kofler et al., 2016), and during environmental demands that challenge their underdeveloped executive functions specifically (Rapport et al., 2009). Despite experimental evidence demonstrating that children with ADHD are typically not more hyperactive than their peers during low cognitive demand conditions but evidence disproportionate increases in hyperactivity when cognitive demands are increased (e.g., Patros et al., 2017; Irwin et al., 2019; Rapport et al., 2009), the extent to which hyperactivity in ADHD is evoked by cognitively challenging tasks in general or by demands on specific executive functions remains unclear. To that end, the current study used a battery of carefully controlled, counterbalanced executive function and non-executive function tasks to examine the effects of imposing varying cognitive demands on objectively-measured activity level in children with and without ADHD.

Objective Measurement of Hyperactivity

Quantifying excess physical activity (hyperactivity) has been of longstanding interest to the field, as evidenced by the broad range of subjective and objective methodologies employed over the last half century (Rapport et al., 2006; Tryon, 1991). Initial studies relied heavily on the use of subjective rating scales (Werry et al., 1968) that provide reliable information on real-world behavior but introduce key confounds (e.g., retrospective recall bias, halo effects, recency effect; Abikoff et al., 1993; Erhardt et al., 1999) and have been unable to successfully differentiate excess motor movement (hyperactivity) from theoretically distinct constructs such as impulsivity (DuPaul et al. 2016; Kofler et al., 2020). To address these issues, studies began to incorporate more objective approaches, such as direct observation (Whalen et al., 1978; Abikoff et al., 1984) or analog measures such as floor grid-crossing counts (Milich, Loney, & Landau, 1982). While more objective than rating scales, these observational methodologies require considerable training and retraining to achieve acceptable levels of interrater reliability and reduce observational drift (Reid, 1982). Due in part to the high human resource costs and time commitments required for observational methods, the use of technological methods to capture gross motor activity has increased in both frequency and in sophistication over the years, beginning in the late

1970s and early 1980s with ultrasonic sensors (Saxon, Magee, & Siegel, 1977), pedometers (Plomin & Foch, 1981), actometers (Porrino et al., 1983), and stabilometric cushions (Conners & Kronsberg 1984). More recent approaches to measuring hyperactivity include infrared motion analysis (Teicher et al., 1996), video compression algorithms (Wehrmann & Muller, 2015), and actigraphy (Halperin et al., 1992; Rapport et al., 2009; Kofler et al., 2016; Kofler, Sarver, & Wells, 2020).

Actigraphs resemble small wristwatches and are a non-invasive method to objectively, reliably, precisely, and cost-effectively record motor activity in experimental or natural settings over prolonged durations (Rapport et al., 2006). Actigraphs have been demonstrated to be reliable measures of motor movement (r=.90–.99 for two actigraphs placed at the same site at the same time; Tryon, 1991), particularly when data from multiple actigraphs are aggregated (Wood et al., 2008; Eaton, 1983; Rapport et al., 2009). Evidence for the validity of actigraphs includes significant correlations with other direct and indirect objective measures of hyperactivity, such as pedometers (r = .89–.96; De Craemer et al., 2015), precision pendulums (r = .99; Tryon, 2005), and room respiration calometry (r =.88–.89; Puyau et al., 2002). In addition, actigraphs correlate moderately with subjective ratings of children's hyperactivity while wearing the actigraphs (r=.32–.58), which provides additional validity evidence while highlighting that informants are considering much more than just children's motor movement when rating their (hyper)activity level (Rapport et al., 2006). Lastly, experimental and meta-analytic evidence indicates that actigraphs are better at differentiating children with ADHD from other clinical disorders than rating scales (Matier-Sharma et al., 1995; Kofler et al., 2016). Critically for our purposes, actigraphs offer unobtrusive yet precise measurement of motor movement that is time-locked and can therefore be synced with the environmental conditions in which the movement occurred, thereby allowing us to experimentally manipulate demands on candidate cognitive processes and concurrently measure the effect of this manipulation on children's hyperactivity (Kofler et al., 2018b; Rapport et al., 2009; Hudec et al., 2015).

Hyperactivity in ADHD

Activity level is the first enduring personality trait to develop, with discernible individual differences emerging as early as 28 weeks gestation (Walters et al., 1965) and predictive of positive post-natal developmental outcomes and activity level at 4 months of age (which in turn predicts activity level into childhood and beyond; for review see Rapport, 2006). Developmental and longitudinal studies indicate that activity level changes across the lifespan, following a curvilinear pattern (Eaton et al., 2001; Galéra et al., 2011; Rapport et al., 2006; Shaw et al., 2005), with higher motor activity associated with positive behavioral characteristics in early childhood (e.g., inquisitiveness, positive social interactions) but negative characteristics after age five (e.g., restlessness, distractibility) as expectations change and children are expected to sit and engage in academic and other cognitively challenging activities for longer durations (for review, see Rapport et al., 2006).

Despite its characterization as a core and impairing symptom of ADHD, the evidence linking elevated activity level (i.e., hyperactivity) with functional outcomes is surprisingly mixed (for review, see Kofler et al., 2020). For example, studies measuring hyperactivity

in children via informant rating scales suggest that hyperactivity predicts a host of adverse near- and long-term outcomes including lower educational attainment and academic achievement, higher ratings of distractibility, strained relationships with peers, and parent-child relational difficulties (Adams et al., 2009; Bagwell et al., 2001; DuPaul et al., 2016; Fergusson, Lnyskey, & Horwood, 1997; Kofler et al., 2018a; Mannuzza et al., 2002; Rapport et al., 1999; Spira & Fischel, 2005). However, recent studies have criticized the construct validity of these subjective measures of hyperactivity (Gawrilow et al., 2014; Helmerhorst et al., 2012) and suggest that these negative relations may be an artifact of conflating hyperactivity with impulsivity via the use of DSM-based hyperactivity/impulsivity questionnaires (e.g., Kofler et al., 2020). To that end, studies using objective or mechanically-assessed measurement of hyperactivity indicate that increases in activity level have shown *positive* associations with inquisitiveness, academic performance, task planning, motor skills, peer interactions, and classroom deportment (Kofler et al., 2018c; Pontifex et al., 2013; Rapport et al., 2006; Smith et al., 2020; Verret et al., 2012). Experimental and meta-analytic evidence also suggests positive associations between physical movement and cognition in children with ADHD (Hudec et al., 2015; Patros et al., 2017; Kofler et al., 2016), such that children with ADHD demonstrate better performance on neurocognitive tasks when they are more physically active relative to when they are less physically active (Hartanto et al., 2016; Sarver et al., 2015).

Hyperactivity in ADHD: A Byproduct of Cognitive/Environmental Demands?

Although the DSM-5 clinical model describes hyperactivity as ubiquitous, non-goal directed behavior, several conceptual models posit that hyperactivity in ADHD may be evoked and/or exacerbated by environmental demands in general and executive function demands in particular (Barkley, 1997; Rapport et al., 2001, 2009; Zentall & Zentall, 1983). Understanding how environmental factors can produce and/or exacerbate hyperactivity in children with ADHD is critical for clarifying to the etiology, course, and pathophysiology of this chronic and impairing neurodevelopmental disorder (Kofler et al., 2016). Interestingly, meta-analytic evidence indicates that children with ADHD are significantly more hyperactive than their peers during high cognitive demand conditions (d = 1.14) but show only minimally elevated hyperactivity during low cognitive conditions (d = 0.36; Kofler et al., 2016), suggesting a functional link between these children's excess movement and the demands placed on their underdeveloped neurocognitive abilities. Studies that experimentally manipulate cognitive demands and observe effects on objectively-assessed hyperactivity are generally consistent with the meta-analytic findings and indicate a "now you see it, now you don't" (Whalen et al., 1978) pattern in which children with ADHD are typically not more hyperactive than their peers during activities such as recess, lunch, drawing/painting, watching television, and physical education classes (Porrino et al., 1983, Rapport et al., 2009, Tsujii et al., 2007), but show disproportionate increases in their motor movement during cognitively challenging tasks such as reading assignments, math assignments, and neurocognitive/executive functioning testing compared to children without ADHD (Dekkers et al., 2020, Sarver et al., 2015, Porrino et al., 1983; Rapport et al., 2009; Tsujii et al., 2007, Tsujii et al., 2009).

At the same time, the extent to which hyperactivity in ADHD is evoked by demands on specific executive functions as opposed to cognitive demands in general remains unclear (Irwin et al., 2019). Briefly defined, executive functions refer to higher-order neurocognitive processes linked with regulating thoughts and behaviors by maintaining problem sets to attain future goals (Miyake et al. 2000; Wiebe et al., 2011). Across the diverse models of executive functions, theoretical and factor analytic work provides the most empirical support for 2- or 3-factor models of executive functions in children that include working memory and inhibitory control, with set-shifting sometimes but not always emerging as a unique executive function in middle childhood (for review see Karr et al., 2008). *Working memory* refers to the active, top-down manipulation of information held in temporal memory (Baddeley, 2007) while *inhibitory control* refers to the ability to withhold or suppress a pre-potent or on-going behavioral response (Lewis & Carpendale, 2009). *Set shifting* is defined as the ability to flexibly switch back-and-forth between mental sets (Pa et al., 2010). Executive function deficits are hypothesized to be a driving factor of ADHD phenotypic behavior, including hyperactivity, in most children with ADHD (Barkley, 1997, Rapport et al., 2009, Sonuga-Barke et al., 2010; Kasper et al., 2012; Chacko et al., 2014). Evidence supporting a link between hyperactivity and executive functions includes experimental studies demonstrating systematic increases in hyperactivity as executive function demands increase, such that executive function tasks elicit more hyperactive behaviors than tasks with minimal cognitive demands such as painting (Rapport et al., 2009, Irwin et al., 2019, Hudec et al., 2015).

In terms of specific executive functions, correlational evidence suggests links between hyperactivity and both working memory (r = 0.45–0.57; Smith et al., 2020; Rapport et al., 2009) and inhibitory control (r = 0.44; Smith et al., 2020). However, a different pattern emerges based on carefully controlled experimental manipulations. Specifically, experimental studies consistently demonstrate links between increased demands on working memory and differential increases in hyperactivity for children with ADHD (Rapport et al., 2009; Dekker et al., 2020). In contrast, experimental studies show that demands on children's inhibitory control do not evoke hyperactive behavior in children with ADHD beyond levels evoked by cognitive tasks with more basic (non-executive) choice decision tasks (Alderson et al., 2012; Irwin et al., 2019). With regard to set shifting, the limited available evidence indicates that experimentally inducing set shifting demands produces systematic increases in hyperactivity at similar rates for both children with and without ADHD (Irwin et al., 2019). To our knowledge, however, no study has examined hyperactivity across all three executive functions in the same sample. Thus, it remains unclear whether specific executive functions evoke greater activity levels than others, or whether demands on any executive function evokes greater activity levels relative to demands on other (non-executive) cognitive processes.

Current Study

Taken together, the evidence base at this time indicates that hyperactivity in ADHD may reflect a functional response to environmental demands that challenge these children's neurocognitive vulnerabilities. However, the extent to which this occurs in response to any cognitively demanding activity, or differentially in response to demands on specific

cognitive processes, remains unclear. The current study is the first to use a counterbalanced battery of multiple tasks per cognitive construct and bifactor s-1 modeling (Eid et al., 2016) to comprehensively test whether elevated gross motor movement (hyperactivity) in children with ADHD occurs in response to cognitive demands in general, or to demands on specific executive functions implicated in contemporary models of ADHD pathogenesis. We hypothesized that children with ADHD would show differential increases in activity level as cognitive demands increased (Alderson et al., 2012; Rapport et al., 2009), as evidenced by significantly larger between-group (ADHD, Non-ADHD) effect sizes during high executive function demand conditions relative to low cognitive demand and non-executive function cognitive demand conditions (Hudec et al., 2015, Irwin et al. 2019). No hypotheses were offered regarding whether specific executive functions would elicit higher levels of hyperactivity than others because, to our knowledge, no studies to date have examined activity level across all three executive functions in the same sample.

Methods

Participants

The sample comprised 184 children (61 girls, 123 boys), aged 8 to 13 years (M = 10.40, SD = 1.50) from the Southeastern United States who were recruited/referred to a university-based children's learning clinic through community resources from 2015–2020 for participation in a larger study of the neurocognitive mechanisms underlying pediatric attention/behavioral problems and completed testing prior to the COVID-19 shutdown. Florida State University Institutional Review Board approval was obtained/maintained, and all parents and children gave informed consent/assent. Psychoeducational evaluations were provided to parents. Sample race/ethnicity was mixed with 129 Caucasian Non-Hispanic (70.1%), 24 Black (13.0%), 13 Hispanic (7.1%), 17 multiracial children (9.3%), and 1 Asian (.5%) children (Table 1).

Group Assignment

All children and caregivers completed 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 for DSM-5) allows differential diagnosis according to symptom onset, course, duration, quantity, severity, and impairment in children and adolescents based on DSM-5 criteria. Its psychometric properties are well established, including inter-rater agreement of .93 to 1.00, test-retest reliability of .63 to 1.00, and concurrent (criterion) validity between the K-SADS and psychometrically established parent rating scales (Kaufman et al., 1997). K-SADS interviews were supplemented with parent and teacher ratings scales from the Behavior Assessment System for Children (BASC-2/3; Reynolds & Kamphaus, 2004) and ADHD Rating Scale (ADHD-RS-4/5; DuPaul et al., 2016). A psychoeducational report was provided to parents; children selected a small toy (<$5) from a prize box after each session.

The ADHD group included 119 children who met all of the following criteria (n=36 girls): (1) DSM-5 diagnosis of ADHD Combined (n=82), Inattentive (n=35), or Hyperactive/Impulsive Presentation (n=2) by the directing clinical psychologist and multidisciplinary

treatment team based on KSADS and differential diagnosis considering all available clinical information indicating onset, course, duration, and severity of ADHD symptoms consistent with the ADHD neurodevelopmental syndrome; (2) borderline/clinical elevations on at least one parent and one teacher ADHD subscale (i.e., > 90th percentile); and (3) current impairment across multiple settings based on parent report. Children with all ADHD current presentation specifiers were eligible given the instability of ADHD subtypes (Lahey et al., 2005; Valo & Tannock, 2010; Willcutt et al., 2012). To improve generalizability (Wilens et al., 2002), children with comorbidities were included. Our standard assessment battery also included norm-referenced child internalizing disorder screeners, and additional standardized measures were administered clinically as needed to inform differential diagnosis and accurate assessment of comorbidities (e.g., child clinical interviews, additional testing). Comorbidities reflect clinical consensus best estimates and included oppositional defiant disorder (11.8%), depressive disorders (3.4%), and anxiety disorders (28.6%). As recommended in the K-SADS, oppositional defiant disorder was diagnosed clinically only with evidence of multi-informant/ multi-setting symptoms. ODD comorbidity is 34% in the ADHD group and 17% in the Non-ADHD group based on parent-reported symptom counts. Children with ADHD were screened for low academic achievement in reading (12.6%) and math (6.7%) defined by score(s) >1.5 SD below age-norms on one or more subtest(s) of the Academic Skills Battery of the Kaufman Test of Educational Achievement, Third Edition (Kaufman, 2014).

The Non-ADHD group comprised 65 consecutive case-control referrals (25 girls) who did not meet ADHD criteria and included both neurotypical children and children with psychiatric disorders other than ADHD. Neurotypical children (61.5%) had normal developmental histories and nonclinical parent/teacher ratings, were recruited through community resources, and completed the same evaluation as clinically referred cases. Clinically referred and evaluated children who did not meet ADHD criteria were also included in the Non-ADHD group. These Non-ADHD disorders were included to control for comorbidities in the ADHD group, and included best estimate diagnoses of anxiety (29.2%), depressive (9.2%), and oppositional defiant disorders (0%). Importantly, the ADHD and Non-ADHD groups did not differ in the proportion of children diagnosed with anxiety or depression (anxiety: $p = .92$, depression: $p = .09$). The ADHD and Non-ADHD groups differed in the proportion of children diagnosed with ODD as expected ($p = .004$).

Children were excluded for gross neurological, sensory, or motor impairment; nonstimulant medications that could not be withheld for testing; or reported history of seizure disorder, psychosis, or intellectual disability disorder. Psychostimulants ($N_{prescribed}= 52$) were withheld >24-hours for neurocognitive testing.

Procedures

Executive function testing occurred as part of a larger battery that involved 1–2 sessions of approximately 3 hours each. All tasks were counterbalanced across testing 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 (Rapport et al., 2009). Performance was monitored at all times by the examiner,

who was stationed just out of the child's view to provide a structured environment while minimizing improvements related to examiner demand characteristics (Gomez & Sanson, 1994).

Working Memory

The Rapport et al. (2009) computerized working memory tests and administration instructions are identical to those described in Kofler et al. (2018b). Reliability and validity evidence includes high internal consistency (α = .81–.97) and 1–3-week test-retest reliability (.76–.90; Kofler et al., 2019; Sarver et al., 2015), and expected relations with criterion working memory complex span (r = .69) and updating tasks (r = .61) (Wells et al., 2018). Six trials per set size were administered in randomized/unpredictable order (3–6 stimuli/trial; 1 stimuli/second; 24 total trials per task) as recommended (Kofler et al., 2016). Five practice trials were administered before each task (80% correct required).

Phonological working memory.—Children were presented a series of jumbled numbers and a letter (1 stimuli/second). The letter was never presented first or last to minimize primacy/recency effects and was counterbalanced to appear equally in the other serial positions. Children reordered and recalled the numbers from least to greatest, and said the letter last (e.g., 4H62 is correctly recalled as 246H).

Visuospatial working memory.—Children were shown nine squares arranged in three offset vertical columns. A series of 2.5 cm dots were presented sequentially (1 stimuli/second); no two dots appeared in the same square on a given trial. All dots were black except one red dot that never appeared first or last to minimize primacy/recency effects. Children reordered the dot locations (black dots in serial order, red dot last) and responded on a modified keyboard.

Inhibitory Control

Stop-signal inhibitory control.—The stop-signal test and administration instructions are identical to those described in Alderson et al. (2008). Psychometric evidence includes high internal consistency (α = .80; Kofler et al., 2019) and three-week test–retest reliability (.72), as well as convergent validity with other inhibitory control measures (Soreni et al., 2009). Go-stimuli are displayed for 1000 ms as uppercase letters X and O positioned in the center of a computer screen (500 ms interstimulus interval; total trial duration = 1500 ms). Xs and Os appeared with equal frequency throughout the experimental blocks. A 1000 Hz auditory tone (i.e., stop-stimulus) was presented randomly on 25% of trials. Stop-signal delay (SSD)—the latency between presentation of go- and stop-stimuli—is initially set at 250 ms and dynamically adjusted ± 50 ms contingent on participant performance. Successfully inhibited stop-trials are followed by a 50 ms increase in SSD, and unsuccessfully inhibited stop-trials are followed by a 50 ms decrease in SSD. All participants completed two practice blocks and four consecutive test blocks of 32 trials per block (24 go-trials, 8 stop-trials per block).

Go/no-go inhibitory control.—The go/no-go test and administration instructions are identical to those described in Kofler et al. (2019). Psychometric evidence includes high internal consistency (α=.95) as well as convergent validity with other inhibitory control

measures (Kofler et al., 2019). Children were presented a randomized series of vertical (go stimuli) and horizontal (no-go stimuli) rectangles in the center of a computer monitor (2000 ms presentation, jittered 800–2000 ms ISI to minimize anticipatory responding). They were instructed to quickly click a mouse button each time a vertical rectangle appeared, but to avoid clicking the button when a horizontal rectangle appeared. A ratio of 80:20 go:no-go stimuli was selected to maximize prepotency (Kane & Engle 2003; Unsworth & Engle 2007). Children completed a 10 trial practice (80% correct required) followed by 4 continuous blocks of 25 test trials each.

Set Shifting

Global-Local.—The global-local test and administration instructions are identical to those described in Irwin et al. (2019). Psychometric evidence includes high internal consistency (α = .86–.90) as well as convergent validity with other set shifting measures (Kofler et al., 2019). This computerized task uses Navon (1977) figures, which feature a “global” shape (e.g., a circle) constructed using smaller, “local” figures (e.g., triangles). Figures were presented one at a time in one of four quadrants in a clockwise rotation on a computer monitor (jittered ISI 800–2000 ms). Children were required to shift their response between global and local features depending on which quadrant the figures appeared (top quadrants: global; bottom quadrants: local). Trials with stimuli in the top left or bottom right quadrants involved set shifting (shift trials) because responses required a different rule than the previous trial; trials with stimuli in the top right or bottom left quadrants did not require shifting because they featured the same rule as the previous trial (non-shift trials). To minimize memory demands, on-screen cues (“big shape”, “small shapes”) remained on-screen next to each quadrant. One hundred and twenty test trials were administered following three blocks of 6 to 8 practice trials (100% correct required).

Number-color.—The number-color test and administration instructions are identical to those described in Kofler et al. (2019). Psychometric evidence includes high internal consistency (α = .87–.95) as well as convergent validity with other set shifting measures (Kofler et al., 2019). A pair of single-digit numbers appeared on the screen, and children were instructed to click either the larger or smaller value depending on the font color (blue = bigger, yellow = smaller; colors selected for maximal discrimination across individuals with all types of color vision). Both digits were the same color on any given trial. To minimize memory demands, on-screen instructions (blue bigger, yellow smaller) remained visible throughout the task. Trials were presented in a semi-random sequence to require shifting every other trial, with an equal number of bigger-smaller and smaller-bigger shifts. Following an 8-trial practice block (100% correct required), children completed 4 consecutive blocks of 30 trials each (120 total test trials; jittered ISI 80–200 ms).

Non-Executive Cognitive Conditions

Choice Reaction Time.—The choice reaction time test and administration instructions are similar to those described by Hudec et al. (2015). In the choice reaction time task, stimuli were displayed for a maximum of 2500 ms as uppercase letters X and Y positioned in the center of a computer screen. Xs and Ys appeared with equal frequency throughout the experimental blocks. A two-button response pad was used wherein the left and right

buttons were labeled X and Y, respectively. All participants completed a practice and four consecutive experimental blocks of 15 trials (total of 60 experimental trials).

Alphanumeric Naming.—In the alphanumeric naming task, 80 stimuli were presented in 8 evenly distributed rows in the center of a computer screen. Stimuli remained on the screen and consisted of 40 letters and 40 numbers that were randomly sequenced throughout each row. Children stated the name of each letter and number out loud, reading from the top left to the bottom right. All participants completed a practice trial prior to the experimental trial.

Baseline Control Conditions

Children used Microsoft® Paint for five consecutive minutes at the beginning and end of each research session. 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 cognitive task battery while interacting with a program that placed relatively modest demands on cognitive processes (i.e., the Paint program allows children to draw/paint anything they like on the monitor using a variety of interactive tools). Following Rapport et al. (2009), the Paint beginning- and end-of-session control conditions were evaluated separately.

Objectively-measured Hyperactivity

Actigraph.—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 to play the computerized 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. Children wore actigraphs on their non-dominant wrist and both ankles. Higher scores indicate greater intensity of movement (proportional integrating measure/PIM mode).

Dependent variables.—Following Rapport et al. (2009), we computed Total Hyperactivity Scores (THS) by summing activity level across the three actigraph sites (2 ankles and 1 non-dominant hand) to index total movement, separately for each task. This approach has the advantage of increasing power while providing a broad sampling of children’s activity level (Rapport et al. 2009). Separate THS scores were computed for each of the working memory, inhibitory control, set shifting, non-executive cognitive, and Paint beginning and end tasks.

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

All children were administered the Verbal Comprehension Index of the Wechsler Intelligence Scale for Children (WISC-V; 2014). Hollingshead (1975) SES was estimated based on caregiver(s)’ education and occupation.

Data Analysis Overview

Analyses were conducted using structural equation modeling (SEM) in MPlus7 (Muthén & Muthén, 2012) and the software program Omega (Watkins, 2017). Our primary analyses were organized into two analytic Tiers. In Tier 1, we used confirmatory factor analysis (CFA) to determine the best representation of children's hyperactivity across the experimental conditions (measurement models). In Tier 2, we then added ADHD diagnostic status to the best fitting measurement model to test the extent to which children with ADHD are more hyperactive than their peers overall, or primarily/exclusively when environmental conditions challenge their neurocognitive vulnerabilities (structural models). This involved adding ADHD status (no/yes) to the best fitting measurement models and then correlating it with each latent factor(s). Because the ADHD grouping variable was dichotomous, we then converted the standardized correlation coefficients (r) to Cohen's d effect sizes (Hayes, 2009) to estimate the magnitude of elevated hyperactivity in ADHD evoked by each cognitive/non-cognitive condition. Tests of dependent rs (Steiger, 1980) were used to test whether specific cognitive conditions evoke significantly higher ADHD-related hyperactivity relative to other conditions (e.g., are children with ADHD more hyperactive during working memory conditions as compared to during low cognitive demand conditions?).

For all confirmatory models, absolute and relative fit were tested. Adequate model fit is indicated by CFI and TFI ≥ .90, and RMSEA ≤ .10. AIC and BIC were used to compare non-nested models; smaller values indicate the preferred model (Kline, 2016). For bifactor models, the software Omega (Watkins, 2017) was used to assess multidimensionality, construct reliability and replicability, and explained common variance. Omega total (ω) and omega subscale (ω_S) index the reliability of the general factor (baseline hyperactivity) and specific factors (low cognitive demand, working memory, inhibitory control, set shifting) by providing estimates of the proportion of variance attributable to sources of common and specific variance, respectively; values >.70 are preferred (Rodriguez et al., 2016b). Omega hierarchical (ω_H) and omega subscale hierarchical (ω_{HS}) estimate the proportion of reliable variance in observed scores attributable to the general factor after accounting for the specific factor, and to the specific factor after accounting for the general factor, respectively. Explained common variance (ECV) indicates the proportion of reliable variance explained by each factor. The percentage of uncontaminated correlations (PUC) is used to assess potential bias from forcing unidimensional data into a multidimensional model. When general factor $ECV > .70$ and $PUC > .70$, bias is considered low and the instrument can be interpreted as primarily unidimensional (i.e., the increased complexity of the bifactor structure is likely not warranted; Rodriguez et al., 2016a). Construct replicability (H) values > .80 suggest a well-defined latent variable that is more likely to be stable across studies (Watkins, 2017).

All items showed the expected range of scores and were screened for normality. All variables of interest had acceptable skewness < |2| and kurtosis values for SEM (kurtosis < |10|; Brown, 2006). Standardized residuals were inspected for magnitude (all positive and <1, indicating no evidence of localized ill fit). Missing data rates were low (2.95% total) and were accounted for using full information maximum likelihood. Maximum likelihood

estimation with robust standard errors (MLR) and delta scaling was specified *a priori* to handle any non-normality and non-independence of the data (Kline, 2016; Muthén & Muthén, 2017). Directionality of parameter estimates were inspected.

Transparency and Openness

The current manuscript reports recruitment procedures, sample size, all data exclusions, all measures and variables, and all experimental manipulations. The current study follows the Journal Article Reporting Standards (JARS; Appelbaum et al., 2018; Kazak, 2018). All data, research materials, and analysis code are available upon request. Data were analyzed using MPlus7 (Muthén & Muthén, 2012) and the software program Omega (Watkins, 2017). The current study's design and its analysis were not pre-registered.

Results

Preliminary Analyses

All parent and teacher ADHD rating scale scores were higher for the ADHD relative to Non-ADHD group as expected (Table 1). The ADHD group also demonstrated higher THS activity level during all task conditions (d=0.35–0.68; all p<.03). There was no significant evidence to indicate between-group differences in socioeconomic status (p=.29), IQ (p=.26), or maternal education level (p=.21), whereas the ADHD group was approximately 5 months younger than the Non-ADHD group on average (p=.04). Age, sex, and SES were controlled in all analyses; the pattern and interpretation of results is unchanged if these covariates are removed.

Tier 1: Measurement Models

Three potential models were initially proposed: (1) a single-factor model (THS scores for all 10 task conditions loading onto one general factor); (2) a correlated 5-factor model (one factor per construct: baseline, low cognitive demand, inhibitory control, set shifting, and working memory); and (3) a bifactor s-1 model (a general baseline hyperactivity factor that qualifies four construct specific factors: low cognitive demand, inhibitory control, set shifting, and working memory).

a. Initial Measurement Models.—As shown in Table 2, the single-factor model with all 10 THS indicators showed adequate model fit. In contrast, both the correlated 5-factor model and the bifactor s-1 model resulted in models that were non-positive definite. Upon closer inspection, their failure to provide acceptable solutions appeared to be because several of the factors were multicollinear, particularly for associations between the set shifting factor and the other executive functions (both r>.90), which in turn were highly correlated but not multicollinear in the bifactor model (r=.86).

b. Revised Measurement Models.—Given the convergence issues with our theoretically- and empirically-specified originally planned models, and evidence that these issues may be due to multicollinearity between set shifting and the other two executive function factors, two additional sets of correlated factor/bifactor models were investigated: First, we tested revised correlated and bifactor models that were identical to the models

described above except that we combined THS scores during all 6 executive function tasks into a single executive function factor. The decision to test a combined executive function factor was based on their high intercorrelations in the preliminary model, as well as the Miyake et al. (2000) model that describes both unity and diversity among the executive functions. As shown in Table 2, the revised correlated 3-factor correlated model showed adequate model fit and improved fit relative to the single factor model. In contrast, the revised 3-factor bifactor s-1 model solution remained inadmissible (non-positive definite).

Second, we tested revised single-factor, 4-correlated factors, and 4-factor bifactor s-1 models, this time with the set shifting tasks excluded (i.e., baseline-paint/general factor, low cognitive demand, working memory, and inhibition). The decision to exclude set shifting was based on its multicollinearity with the other executive functions in the preliminary bifactor model, combined with mixed empirical evidence regarding whether or not set shifting is a unique executive function in the current sample's age range (Karr et al., 2008). The revised single-factor model showed adequate and improved model fit as compared to the initial 10-indicator single-factor model. The revised 4-correlated factors model showed adequate and improved model fit compared to both the revised single-factor model and the earlier 3-correlated factors model (Table 2). Similarly, the revised 4-factor bifactor model showed adequate fit and improved model fit compared to the revised single-factor model and earlier 3-correlated factors model but was not easily differentiated from the revised 4-correlated factors model as described below.

In summary, the revised 4-correlated factors and 4-factor bifactor s-1 models demonstrated the best model fit of all tested models (Table 2). The revised 4-correlated factors model was slightly better than (ΔBIC=12, ΔAIC=3), indistinguishable from (CFI, TLI >.99), or slightly worse (ΔSRMR=−.01) than the revised 4-factor bifactor s-1 model. Importantly, however, the revised 4-correlated factors model continued to show factors that were multicollinear (Figure 2). Therefore, consistent with prior work where a well-fitting model is ultimately rejected due to multicollinearity among factors (for review, see Allan & Lonigan, 2019), the revised 4-correlated factors model was rejected. Examination of additional indices for the bifactor model indicated that the percent of uncontaminated correlations was high (PUC =.86) but explained common variance and factor-specific variance explained were moderate for the general baseline hyperactivity factor (*ECV*=.68, ω_H=.80), supporting the multidimensionality of the data and the use of a bifactor s-1 model. In addition, the revised bifactor model produced factors that were either uncorrelated (general factor with all specific factors) or were correlated across a range of levels that did not approach multicollinearity (Figure 1). As such, the revised 4-factor bifactor s-1 model was selected. This model showed excellent fit, best differentiated children's hyperactivity levels across experimental conditions, most directly addressed our primary questions of interest, and was consistent with meta-analytic evidence demonstrating that baseline (general) level of physical/motor movement in children is, in part, modulated by task-specific demands in the environment (Kofler et al., 2016).

Explained common variance and factor-specific variance explained were moderate for the general baseline hyperactivity factor (*ECV*=.68, ω_H=.80), and the specific factors explained meaningful but more modest portions of the variance in children's hyperactivity level

above and beyond the general baseline hyperactivity factor (*ECV*=.07–.14, ω_H=.20–.39). Inspection of the factor reliability and replicability indices for the bifactor model indicated there was support for the reliability of the general baseline hyperactivity factor (ω=.90) and all specific condition factors (ω=.70–.80). Taken together, this evidence provides further support for our decision to select the revised 4-factor bifactor model given its excellent model fit, adequate to excellent reliability, high construct replicability, and ability to differentiate children's motor movement across the experimental conditions of interest. At the same time, we acknowledge that it may have been reasonable to adopt the revised 4-correlated factors model despite its multicollinearity issues, and have therefore examined its associations with ADHD status as a sensitivity analyses. As reported below, results were highly consistent when based on the correlated factors vs. bifactor model.

Tier 2: Structural Models

The Tier 2 structural model involved adding ADHD status (no/yes) to the bifactor s-1 model and then correlating it with the general factor (baseline hyperactivity) and specific factors (hyperactivity during the low cognitive demand, inhibition, and working memory conditions; Table 3). As shown in Figure 1, children with ADHD demonstrated overall higher levels of motor movement than Non-ADHD children that was independent of the impact of cognitive demands (baseline hyperactivity: *d*=0.72, *r*=.34, *p*=.003). Interestingly, when controlling for this baseline level of elevated motor movement, ADHD was also strongly associated with additional increases in hyperactivity during the working memory (Δ*d*=0.80, *r*=.37, *p*=.004) and inhibition conditions (Δ*d*=0.80, *r*=.37, *p*=.04). In contrast, the low cognitive demand conditions failed to evoke additional increases in hyperactivity for children with ADHD relative to baseline (Δ*d*=0.24, *r*=.12, *p*=.37). Tests of dependent *r*s indicated that the working memory and inhibition conditions (*p*>.99; WM=IC) evoked significantly greater increases in hyperactivity relative to the low cognitive demand conditions for children with ADHD (both *p*<.001; i.e., WM=IC>Low).

Sensitivity Analyses

Sensitivity analyses were conducted by repeating the Tier 2 analysis above, this time using the revised 4-correlated factors model (Table 3). Results were largely consistent with the revised 4-factor bifactor s-1 model. As shown in Figure 2, ADHD status was moderately associated with increased hyperactivity during the baseline paint (*d*=0.72, *r*=.34, *p*=.003) and strongly associated with increased hyperactivity during the working memory (*d*=1.12, *r*=.49, *p*<.001) and inhibition conditions (*d*=1.12, *r*=.49, *p*<.001,). Different from the primary model, without controlling for baseline hyperactivity levels, children with ADHD also demonstrated moderate elevations in hyperactivity during the low cognitive demand conditions (*d*=0.52, *r*=.25, *p*=.02). Results of the tests of dependent *r*s indicated that ADHD diagnosis was associated lower hyperactivity during the baseline and low cognitive demand conditions (*p*=.11; Paint=LowCog), and higher hyperactivity during the working memory and inhibition conditions (all *p*≤.004; Paint=Low<WM=IC).

Discussion

The current study was the first to use objective measurement of children's hyperactivity while experimentally manipulating neurocognitive demands via a counterbalanced battery to comprehensively test the extent to which elevated gross motor movement (hyperactivity) in children with ADHD occurs in response to cognitive demands in general, or to demands on specific executive functions implicated in contemporary models of ADHD pathogenesis (e.g., Kofler et al., 2016). Additional strengths include the relatively large and clinically evaluated sample of boys and girls, assessment of motor movement during multiple tests per construct, and use of multiple actigraphs attached to different body positions to provide a broad sampling of children's motor movement. Overall, the current findings indicated that children with ADHD demonstrate moderately elevated levels of motor movement relative to Non-ADHD children that are observable even under baseline conditions with relatively minimal cognitive demands (d=0.72). This estimate is highly consistent with prior studies based on adult (Hudec et al., 2015) and male child samples (Patros et al., 2016), but is somewhat higher than expected based on previous meta-analytic findings and experimental studies indicating children with ADHD are often not more hyperactive than their peers during baseline activities such as recess, lunch, drawing/painting, and watching television (d=0.36; Kofler et al., 2016; Porrino et al., 1983, Rapport et al., 2009, Tsujii et al., 2007). Together, these findings highlight the equifinality in behavioral outcomes for children with ADHD. When combined with the increases in hyperactivity seen under the high cognitive demands (noted below), this pattern of results suggests that (a) executive function demands exacerbate but do not fully explain hyperactivity in ADHD, which is partially inconsistent with theoretical models (e.g., Rapport et al., 2009; Porrino et al., 1983); and/or (b) there are at least 2 pathways to hyperactivity in ADHD – hyperactivity caused by environmental demands that challenge their underdeveloped executive functions, and hyperactivity caused by one or more other factors that we need future research to identify.

Of primary interest in the current study was the extent to which hyperactivity in ADHD is evoked by any cognitively demanding activity or specifically by demands on specific cognitive process(es). Modern theoretical models and experimental evidence suggests that hyperactivity in ADHD may reflect, at least in part, a functional response to environmental demands that challenge these children's underdeveloped executive functions (e.g., Rapport et al., 2009, Hudec et al., 2015, Kofler et al., 2016). However, the extent to which these differential increases in activity level are linked with demands on specific executive functions as opposed to cognitive demands in general has not been clear. Interestingly, when controlling for baseline levels of elevated motor movement, evoking demands on working memory and inhibitory control each produced large magnitude increases in hyperactivity for children with ADHD (both Δd=0.80). In other words, children with ADHD exhibit medium magnitude elevations relative to their peers even during minimal cognitive demands conditions, and these differences become even more pronounced when their underdeveloped executive functions are challenged. These findings are consistent with meta-analytic evidence showing that children with ADHD are significantly more hyperactive than their peers during high cognitive demand conditions but show only minimally elevated hyperactivity during low cognitive conditions (Kofler et al., 2016). More specific to working

memory, these findings are consistent with experimental studies that demonstrate links between increased working memory demands and differential increases in hyperactivity for children with ADHD (Rapport et al., 2009; Dekker et al., 2020) and extend prior work by using latent estimates and controlling for baseline hyperactivity in a larger sample of boys and girls with and without ADHD. Similarly, the current findings are consistent with intervention studies showing that (a) improving working memory produces significant reductions in actigraph-measured hyperactivity; and (b) treatment-related improvements in working memory predict treatment-related reductions in actigraph-measured hyperactivity in children with ADHD (Kofler et al., 2018, Kofler et al., 2020b).

With regards to hyperactivity evoked by inhibitory control demands, the current study found similar, large magnitude increases that were equivalent to those elicited by working memory demands. These findings appear to be in contrast with (a) experimental studies showing that increasing inhibitory control demands may not elicit actigraph-measured hyperactive behavior in children with ADHD beyond those evoked by basic (non-executive) choice decision tasks (Alderson et al., 2012; Irwin et al., 2019); and (b) intervention studies demonstrating that improving inhibitory control does not produce significant reductions in actigraphy-measured hyperactivity (Kofler et al., 2020b). A potential explanation for the differences between our findings and previous studies may be that prior work examined changes in hyperactivity during single inhibitory control tasks with relatively smaller samples. The current study builds on these studies by examining latent estimates of hyperactivity across multiple tasks per construct using a bifactor s-1 modeling approach that controlled for children's baseline activity level, thus allowing us to obtain more precise estimates of construct-specific hyperactivity and more direct comparisons regarding whether specific executive functions evoke greater activity levels than others. To that end, the current study found that increasing inhibitory control and working memory demands both produced similar, large magnitude increases in hyperactivity for children with ADHD relative to their baseline levels. These findings indicate that hyperactivity in ADHD is, at least in part, attributable to demands placed on each executive functions, with working memory and inhibition demands evoking similar, differential increases in motor movement for children with ADHD relative to their peers.

In contrast, the low cognitive demand conditions did not evoke additional increases in hyperactivity for children with ADHD beyond their baseline elevations. At first glance, this finding appears to be discrepant from previous studies demonstrating differential increases in hyperactivity for children with ADHD between tasks with minimal vs. low cognitive demands (Alderson et al., 2012, Hudec et al., 2015, Irwin et al. 2019). However, consistent with previous studies, inspection of Table 1 indicates significant ADHD/Non-ADHD between-group differences during both low cognitive demand tasks (d=0.35–0.53). In contrast, when using bifactor s-1 modeling to control for children's baseline motor activity levels, the group differences in hyperactivity during the low cognitive demand conditions disappeared. Thus, the current findings may not directly contradict prior work, but instead add to the evidence by showing that increasing cognitive demands in general does not differentially affect children with ADHD (Irwin et al., 2019) and by showing that the variance between children with and without ADHD during the low cognitive demand conditions is primarily, if not entirely, accounted for by their baseline activity levels.

Notably, while the bifactor s-1 model of hyperactivity was selected for as it was identified as a well-fitting model, it best differentiated children's hyperactivity levels across experimental conditions, and it most directly addressed our primary questions of interest, a revised 4-correlated factors model was also tested and found to have a somewhat better fit (ΔBIC=12, ΔAIC=3) but was indistinguishable from (CFI, TLI >.99) the revised bifactor s-1 model. While the 4-correlated model not selected for the purposes of this study, the 4-correlated factor model was a variable well-fitting model that could have been selected and would have likely given different implications given its structure not discussed further here.

Limitations

The unique contribution of the current study was its latent assessment of hyperactivity across control, low cognitive, and high executive function demands in a large and carefully phenotyped sample of children with and without ADHD. Additional strengths of the study include the use of objectively measured hyperactivity (i.e., actigraphy) across multiple measures of each cognitive construct of interest. At the same time, several caveats merit consideration when interpreting results. First, despite the study's relatively large sample size and that the ADHD and non-ADHD groups were equivalent with regard to race/ethnicity, SES, and maternal education level, the majority of the participants in the study were identified as White, male and relatively high maternal education levels were reported (i.e., majority of mother's reported having a Bachelor's degree or higher). As such, the findings from the study may not generalize to historically-excluded racial minority groups, male children, as well as children who have mothers with relatively lower levels of education. While the current study is the first to examine the extent to which hyperactivity in children with ADHD is elicited by multiple executive and nonexecutive neurocognitive processes, we were unable to include hyperactivity during set shifting tasks in the final model given its multicollinearity with hyperactivity during the working memory and inhibitory control tasks. Inspection of Table 1 indicate similar magnitude ADHD/Non-ADHD between-group differences in hyperactivity during the set shifting tasks (d=0.56–0.62) as compared to hyperactivity during the other executive function tasks (i.e., working memory and inhibitory control; d=0.55–0.68). Future work may benefit from including additional measures of these constructs to further parse apart the associations between pediatric ADHD and hyperactivity during set shifting conditions. Along those lines, future work is necessary to examine how other neurocognitive and cognitive-behavioral processes, such as reward sensitivity (Kallen et al., 2020), emotion regulation (Groves et al., 2020), and processing speed (Cook et al., 2018) impact or are impacted by hyperactivity for children with ADHD. Similarly, it remains to be seen how other non-neurocognitive processes, such as fine motor skills (Mokobane et al., 2019), may affect motor movement in children with ADHD.

Additionally, our ADHD group included children with any 'current presentation' specifier based on meta-analytic evidence that children with the Inattentive vs. Combined (Inattentive + Hyperactive/Impulsive) presentations do not differ in terms of actigraph-measured movement, and that both presentations demonstrate similarly elevated activity level at the group and individual levels relative to neurotypical and clinical controls (Kofler et al., 2016). However, other meta-analytic evidence suggests that the ADHD presentations may differ in terms of neuropsychological functioning (Willcutt et al., 2012), and still other evidence

questions the validity of categorical ADHD subtype/presentation sub-groupings altogether (e.g., Valo & Tannock, 2010). Future work with larger samples of each ADHD presentation is needed to investigate whether different neurocognitive/executive functions may evoke differential increases in objectively-measured hyperactivity across ADHD subgroups. Lastly, while the inclusion of children with other forms of child psychopathology was considered a strength because it improves the generalizability of our findings (Wilens et al., 2002), the extent to which co-occurring disorders may suppress or exacerbate motor movement in children with ADHD remains unclear (Lea et al., 2018) despite our finding that the ADHD and Non-ADHD groups did not differ in the rates of most of these common comorbidities. Future work using a larger neurotypical samples is necessary to investigate these relations.

Clinical and Research Implications

The current study examined the extent to which hyperactivity in pediatric ADHD occurs in response to any cognitively demanding activity as opposed to differentially in response to demands on specific cognitive processes. Taken together, our findings indicated that hyperactivity in ADHD reflects the outcome of at least two similarly important factors: (a) a baseline level of elevated motor movement that is independent of environmental demands on their executive and non-executive cognitive abilities (d=0.72), and (b) additional elevations attributable to demands placed on each executive functions, with working memory and inhibitory control demands evoking similarly large, differential increases in movement for children with ADHD above and beyond their elevated baselines (Δd=0.80). These findings suggest clinicians, educators, and caregivers should carefully consider the extent to which hyperactive behavior, such as fidgeting or shifting in one's seat, are considered impairing and need to be restricted versus whether they are compensatory behaviors necessary for task-completion. As such, mental health providers providing biopsychosocial treatments and/or behavioral management training for children ADHD are encouraged to emphasize rewarding successful completion of prosocial behaviors rather than attempting to stifle movement. Consistent with meta-analytic and prior experimental evidence suggesting functional links between children's excess movement and the demands placed on their underdeveloped neurocognitive abilities (Kofler et al., 2016; Rapport et al., 2009; Dekker et al., 2020; Hudec et al., 2015), results of the current study provide further empirical support for the potential benefits of developing interventions to strengthen working memory and/or inhibitory control abilities to reduce hyperactive behaviors in children with ADHD (Kofler et al., 2018d; Kofler et al., 2020b).

While the current study examined changes in hyperactivity during varying levels of cognitively demanding tasks, a potentially important area of research is still needed to differentiate hyperactivity (i.e., excess gross motor movement) from general activity levels. In particular, future work is needed to determine if relations between activity level and activities of daily living/functioning are linearly or curvilinearly associated. Disentangling the nuances of activity verse hyperactivity levels across development could serve as a refence point for identifying those with and without ADHD. Lastly, understanding the full set of neurocognitive and other environmental factors that produce/exacerbate hyperactivity in these children will be important for clarifying the etiology, course, and pathophysiology of ADHD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

Abikoff H, Courtney M, Pelham WE, & Koplewicz HS (1993). Teachers' ratings of disruptive behaviors: The influence of halo effects. Journal of abnormal child psychology, 21(5), 519–533.

Abikoff H, & Gittelman R (1984). Does behavior therapy normalize the classroom behavior of hyperactive children?. Archives of general psychiatry, 41(5), 449–454.

Adams R, Finn P, Moes E, Flannery K, & Rizzo AS (2009). Distractibility in attention/deficit/hyperactivity disorder (ADHD): The virtual reality classroom. Child Neuropsychology, 15(2), 120–135.

Alderson RM, Rapport MD, Kasper LJ, Sarver DE, & Kofler MJ (2012). Hyperactivity in boys with attention deficit/hyperactivity disorder (ADHD): the association between deficient behavioral inhibition, attentional processes, and objectively measured activity. Child Neuropsychology, 18(5), 487–505.

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

Ambulatory Monitoring. (2004). The Micromini Motionlogger Actigraph and family of recorders user's manual (3rd ed.). New York, NY: Author.

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (DSM-5®). American Psychiatric Pub.

Appelbaum M, Cooper H, Kline RB, Mayo-Wilson E, Nezu AM, & Rao SM (2018). Journal article reporting standards for quantitative research in psychology: The APA Publications and Communications Board task force report. American Psychologist, 73(1), 3.

Baddeley A (2007). Working memory, thought, and action (Vol. 45). OuP Oxford.

Bagwell CL, Molina BS, Pelham WE Jr, & Hoza B (2001). Attention-deficit hyperactivity disorder and problems in peer relations: Predictions from childhood to adolescence. Journal of the American Academy of Child & Adolescent Psychiatry, 40(11), 1285–1292.

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

Chacko A, Kofler M, & Jarrett M (2014). Improving outcomes for youth with ADHD: A conceptual framework for combined neurocognitive and skill-based treatment approaches. Clinical child and family psychology review, 17(4), 368–384.

Conners CK, & Kronsberg S (1985). Measuring activity level in children. Psychopharmacology bulletin, 21(4), 893.

Cook NE, Braaten EB, & Surman CB (2018). Clinical and functional correlates of processing speed in pediatric attention-deficit/hyperactivity disorder: a systematic review and meta-analysis. Child Neuropsychology, 24(5), 598–616.

De Craemer M, De Decker E, Santos-Lozano A, Verloigne M, De Bourdeaudhuij I, Deforche B, & Cardon G (2015). Validity of the Omron pedometer and the actigraph step count function in preschoolers. Journal of Science and Medicine in Sport, 18(3), 289–293.

Dekkers TJ, Rapport MD, Calub CA, Eckrich SJ, & Irurita C (2020). ADHD and hyperactivity: The influence of cognitive processing demands on gross motor activity level in children. Child Neuropsychology, 1–20.

DuPaul GJ, Power TJ, Anastopoulos AD, & Reid R (2016). ADHD rating scale-5 for children and adolescents: checklists, norms, and clinical interpretation. Guilford Publications.

DuPaul GJ, Reid R, Anastopoulos AD, Lambert MC, Watkins MW, & Power TJ (2016). Parent and teacher ratings of attention-deficit/hyperactivity disorder symptoms: Factor structure and normative data. Psychological Assessment, 28(2), 214.

Eaton WO (1983). Measuring activity level with actometers: Reliability, validity, and arm length. Child Development, 720–726.

Eaton WO, McKeen NA, & Campbell DW (2001). The waxing and waning of movement: Implications for psychological development. Developmental Review, 21(2), 205–223.

Eid M, Krumm S, Koch T, & Schulze J (2018). Bifactor models for predicting criteria by general and specific factors: Problems of nonidentifiability and alternative solutions. Journal of Intelligence, 6(3), 42.

Erhardt D, Epstein JN, Conners CK, Parker JDA, & Sitarenios G (1999). Self-ratings of ADHD symptoms in auts II: Reliability, validity, and diagnostic sensitivity. Journal of Attention Disorders, 3(3), 153–158.

Fergusson DM, Lynskey MT, & Horwood LJ (1997). Attentional difficulties in middle childhood and psychosocial outcomes in young adulthood. Journal of Child Psychology and Psychiatry, 38(6), 633–644.

Galéra C, Côté SM, Bouvard MP, Pingault JB, Melchior M, Michel G, … & Tremblay RE (2011). Early risk factors for hyperactivity-impulsivity and inattention trajectories from age 17 months to 8 years. Archives of general psychiatry, 68(12), 1267–1275.

Gawrilow C, Kühnhausen J, Schmid J, & Stadler G (2014). Hyperactivity and motoric activity in ADHD: characterization, assessment, and intervention. Frontiers in psychiatry, 5, 171.

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

Groves NB, Kofler MJ, Wells EL, Day TN, & Chan ES (2020). An examination of relations among working memory, ADHD symptoms, and emotion regulation. Journal of abnormal child psychology, 48(4), 525–537.

Halperin JM, Matier K, Bedi G, Sharma V, & Newcorn JH (1992). Specificity of inattention, impulsivity, and hyperactivity to the diagnosis of attention-deficit hyperactivity disorder. Journal of the American Academy of Child & Adolescent Psychiatry, 31, 190–196.

Hartanto TA, Krafft CE, Iosif AM, & Schweitzer JB (2016). A trial-by-trial analysis reveals more intense physical activity is associated with better cognitive control performance in attention-deficit/hyperactivity disorder. Child Neuropsychology, 22(5), 618–626.

Helmerhorst HHJ, Brage S, Warren J, Besson H, & Ekelund U (2012). A systematic review of reliability and objective criterion-related validity of physical activity questionnaires. International Journal of Behavioral Nutrition and Physical Activity, 9, 103.

Hollingshead AB (1975). Four factor index of social status. Yale: New Haven, CT.

Hudec KL, Alderson RM, Patros CH, Lea S, Tarle S, & Kasper L (2015). Hyperactivity in boys with attention-deficit/hyperactivity disorder (ADHD): The role of executive and non-executive functions. Research in developmental disabilities, 45, 103–109.

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

Kallen AM, Perkins ER, Klawohn J, & Hajcak G (2020). Cross-sectional and prospective associations of P300, RewP, and ADHD symptoms in female adolescents. International Journal of Psychophysiology, 158, 215–224.

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

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

Kasper LJ, Alderson RM, & Hudec KL (2012). Moderators of working memory deficits in children with attention-deficit/hyperactivity disorder (ADHD): a meta-analytic review. Clinical psychology review, 32(7), 605–617.

Kaufman AS (2014). Kaufman Test of Educational Achievement (KTEA-3) technical & interpretive manual. Bloomington, MN: Pearson.

Kazak AE (2018). Journal article reporting standards.

Kofler MJ, Groves NB, Singh LJ, Soto EF, Chan ES, Irwin LN, & Miller CE (2020). Rethinking hyperactivity in pediatric ADHD: Preliminary evidence for a reconceptualization of hyperactivity/impulsivity from the perspective of informant perceptual processes. Psychological assessment, 32(8), 752.

Kofler MJ, Harmon SL, Aduen PA, Day TN, Austin KE, Spiegel JA, … & Sarver DE, (2018a). Neurocognitive and behavioral predictors of social problems in ADHD: A Bayesian framework. Neuropsychology, 32(3), 344.

Kofler MJ, Irwin LN, Soto EF, Groves NB, Harmon SL, & Sarver DE (2018b). Executive functioning heterogeneity in pediatric ADHD. Journal of Abnormal Child Psychology, 1–14.

Kofler MJ, Raiker JS, Sarver DE, Wells EL, & Soto EF (2016). Is hyperactivity ubiquitous in ADHD or dependent on environmental demands? Evidence from meta-analysis. Clinical psychology review, 46, 12–24.

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

Kofler MJ, Sarver DE, Austin KE, Schaefer HS, Holland E,… & Lonigan CJ (2018d). Can working memory training work for ADHD? Development of central executive training and comparison with behavioral parent training. Journal of consulting and clinical psychology, 86, 964.

Kofler MJ, Sarver DE, & Wells EL (2020). Working memory and increased activity level (hyperactivity) in ADHD: Experimental evidence for a functional relation. Journal of attention disorders, 1087054715608439.

Kofler MJ, Spiegel JA, Soto EF, Irwin LN, Wells E, & Austin K (2019). Do working memory deficits underlie reading problems in ADHD? Journal of Abnormal Child Psychology,47,433–446.

Kofler MJ, Wells EL, Singh LJ, Soto EF, Irwin LN, … & Lonigan CJ (2020b). A randomized controlled trial of central executive training (CET) versus inhibitory control training (ICT) for ADHD. Journal of consulting and clinical psychology, 88, 738.

Lahey BB, Pelham WE, Loney J, Lee SS, & Willcutt E (2005). Instability of the DSM-IV subtypes of ADHD from preschool through elementary school. Archives of general psychiatry, 62(8), 896–902.

Lea SE, Alderson RM, Patros CH, Tarle SJ, Arrington EF, & Grant DM (2018). Working memory and motor activity: a comparison across ADHD, generalized anxiety disorder, and healthy control groups. Behavior therapy, 49, 419–434.

Lewis C, & Carpendale JI (2009). Introduction: Links between social interaction and executive function. New Directions for Child and Adolescent Development, 2009, 1–15.

Mannuzza S, Klein RG, & Moulton JL (2002). Young adult outcome of children with “situational” hyperactivity: a prospective, controlled follow-up study. Journal of abnormal child psychology, 30(2), 191–198.

Matier-Sharma K, Perachio N, Newcorn JH, Sharma V, & Halperin JM (1995). Differential diagnosis of ADHD: Are objective measures of attention, impulsivity, and activity level helpful?. Child Neuropsychology, 1(2), 118–127.

Milich R, Loney J, & Landau S (1982). Independent dimensions of hyperactivity and aggression: A validation with playroom observation data. Journal of Abnormal Psychology, 91(3), 183.

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

Mokobane M, Pillay BJ, & Meyer A (2019). Fine motor deficits and attention deficit hyperactivity disorder in primary school children. South African Journal of Psychiatry, 25.

Muthén LK and Muthén BO (2017). Mplus User’s Guide, Eighth Edition. Los Angeles, CA: Muthén & Muthén.

Navon D (1977). Forest before trees: The precedence of global features in visual perception. Cognitive Psychology, 9, 353–383.

Noldus Information Technology. (2012). The Observer XT. Wageningen, Netherlands: Author.

Pa J, Possin KL, Wilson SM, Quitania LC, Kramer JH, Boxer AL, … & Johnson JK (2010). Gray matter correlates of set-shifting among neurodegenerative disease, mild cognitive impairment, and healthy older adults. Journal of the International Neuropsychological Society, 16, 640–650.

Patros CH, Alderson RM, Hudec KL, Tarle SJ, & Lea SE (2017). Hyperactivity in boys with ADHD: The influence of underlying visuospatial working memory and self-control processes. Journal of experimental child psychology, 154, 1–12.

Plomin R, & Foch TT (1981). Hyperactivity and pediatrician diagnoses, parental ratings, specific cognitive abilities, and laboratory measures. Journal of Abnormal Child Psychology, 9(1), 55–64.

Pontifex MB, Saliba BJ, Raine LB, Picchietti DL, & Hillman CH (2013). Exercise improves behavioral, neurocognitive, and scholastic performance in children with attention-deficit/hyperactivity disorder. The Journal of pediatrics, 162(3), 543–551.

Porrino LJ, Rapoport JL, Behar D, Sceery W, Ismond DR, & Bunney WE (1983). A naturalistic assessment of the motor activity of hyperactive boys: I. Comparison with normal controls. Archives of General Psychiatry, 40(6), 681–687.

Puyau MR, Adolph AL, Vohra FA, & Butte NF (2002). Validation and calibration of physical activity monitors in children. Obesity research, 10(3), 150–157.

R Core Team (2018). R: A language and environment for statistical computing. R Foundation for Statistical Computing. https://www.r-project.org.

Rodriguez A, Reise SP, & Haviland MG (2016a). Applying bifactor statistical indices in the evaluation of psychological measures. Journal of Personality Assessment, 98, 223–237.

Rodriguez A, Reise SP, & Haviland MG (2016b). Evaluating bifactor models: Calculating and interpreting statistical indices. Psychological Methods, 21, 137–150.

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

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

Rapport MD, Kofler MJ, & Himmerich C (2006). Activity measurement. In Clinician's handbook of child behavioral assessment (pp. 125–157). Academic Press.

Rapport MD, Scanlan SW, & Denney CB (1999). Attention-deficit/hyperactivity disorder and scholastic achievement: A model of dual developmental pathways. Journal of Child Psychology and Psychiatry, 40(8), 1169–1183.

Reid JB (1982). Observer training in naturalistic research. New Directions for Methodology of Social & Behavioral Science.

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

Saxon SA, Magee JT, & Siegel DS (1977). Activity level patterns in the hyperactive ritalin responder and non-responder. Journal of Clinical Child & Adolescent Psychology, 6(3), 27–29.

Shaw DS, Lacourse E, & Nagin DS (2005). Developmental trajectories of conduct problems and hyperactivity from ages 2 to 10. Journal of Child Psychology and Psychiatry, 46(9), 931–942.

Smith JN, Raiker JS, Fosco WD, Jusko ML, Campez M, Little K, … & Musser ED (2020). Executive Functioning and Activity in Children: a Multimethod Examination of Working Memory, Inhibition, and Hyperactivity. Journal of abnormal child psychology, 48(9), 1143–1153.

Sonuga-Barke E, Bitsakou P, & Thompson M (2010). Beyond the dual pathway model: evidence for the dissociation of timing, inhibitory, and delay-related impairments in attention-deficit/hyperactivity disorder. Journal of the American Academy of Child & Adolescent Psychiatry, 49(4), 345–355.

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

Spira EG, & Fischel JE (2005). The impact of preschool inattention, hyperactivity, and impulsivity on social and academic development: A review. Journal of Child Psychology and Psychiatry, 46(7), 755–773.

Steiger JH (1980). Tests for comparing elements of a correlation matrix. Psychological bulletin, 87(2), 245.

Teicher MH, Ito Y, Glod CA, & Barber NI (1996). Objective measurement of hyperactivity and attentional problems in ADHD. Journal of the American Academy of Child & Adolescent Psychiatry, 35(3), 334–342.

Tryon WW (2005). The reliability and validity of two ambulatory monitoring actigraphs. Behavior Research Methods, 37(3), 492–497.

Tryon WW, Pinto LP, & Morrison DF (1991). Reliability assessment of pedometer activity measurements. Journal of Psychopathology and Behavioral Assessment, 13, 27–44.

Tsujii N, Okada A, Kaku R, Kuriki N, Hanada K, Matsuo J, … & Hitomi K (2007). Association between activity level and situational factors in children with attention deficit/hyperactivity disorder in elementary school. Psychiatry and Clinical Neurosciences, 61(2), 181–185.

Tsujii N, Okada A, Kaku R, Kuriki N, Hanada K, & Shirakawa O (2009). Differentiation between attention-deficit/hyperactivity disorder and pervasive developmental disorders with hyperactivity on objective activity levels using actigraphs. Psychiatry and clinical neurosciences, 63(3), 336–343.

Unsworth N, & Engle RW (2007). The nature of individual differences in working memory capacity: active maintenance in primary memory and controlled search from secondary memory. Psychological Review, 114, 104.

Valo S, & Tannock R (2010). Diagnostic instability of DSM–IV ADHD subtypes: Effects of informant source, instrumentation, and methods for combining symptom reports. Journal of clinical child & adolescent psychology, 39(6), 749–760.

Verret C, Guay MC, Berthiaume C, Gardiner P, & Béliveau L (2012). A physical activity program improves behavior and cognitive functions in children with ADHD: an exploratory study. Journal of attention disorders, 16(1), 71–80.

Walters CE (1965). Prediction of postnatal development from fetal activity. Child Development, 801–808.

Watkins MW (2017). Omega v.2 [Computer software]. Phoenix AZ: Ed & Psych Associates.

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

Werry JS (1968). Developmental hyperactivity. Pediatric Clinics of North America, 15,581–99.

Wehrmann T, & Müller JM (2015). An objective measure of hyperactivity aspects with compressed webcam video. Child and adolescent psychiatry and mental health, 9(1), 1–11.

Wechsler D (2014). Wechsler Intelligence Scale for Children-5th Ed. San Antonio: Pearson.

Whalen CK, Collins BE, Henker B, Alkus SR, Adams D, & Stapp J (1978). Behavior observations of hyperactive children and methylphenidate (ritalin) effects in systematically structured classroom environments: Now you see them, now you don'. Journal of Pediatric Psychology, 3(4), 177–187.

Wiebe SA, Sheffield T, Nelson JM, Clark CA, Chevalier N, & Espy KA (2011). The structure of executive function in 3-year-olds. Journal of experimental child psychology, 108(3), 436–452.

Willcutt EG, Nigg JT, Pennington BF, Solanto MV, Rohde LA, Tannock R, … & Lahey BB (2012). Validity of DSM-IV attention deficit/hyperactivity disorder symptom dimensions and subtypes. Journal of abnormal psychology, 121(4), 991.

Wilens TE, Biederman J, & Spencer TJ (2002). Attention deficit/hyperactivity disorder across the lifespan. Annual review of medicine, 53(1), 113–131.

Wood AC, Kuntsi J, Asherson P, & Saudino KJ (2008). Actigraph data are reliable, with functional reliability increasing with aggregation. Behavior research methods, 40, 873–878.

Zentall SS, & Zentall TR (1983). Optimal stimulation: a model of disordered activity and performance in normal and deviant children. Psychological bulletin, 94(3), 446.

Key Points

Question:

Is hyperactivity in children with ADHD a functional response to demands on specific executive functions or cognitive demands in general?

Findings:

Hyperactivity in ADHD reflects the outcome of at least two similarly important factors: a baseline elevated motor movement that is independent of environmental demands on their executive and non-executive cognitive abilities, and additional elevations attributable to demands placed on specific executive functions.

Importance:

Findings highlight the importance of functional links between children's excess movement and the demands placed on specific underdeveloped neurocognitive abilities, such as working memory and inhibitory control.

Next Steps:

Future work is needed to identify and understand additional neurocognitive and other factors that produce/exacerbate hyperactivity in children with and without ADHD.

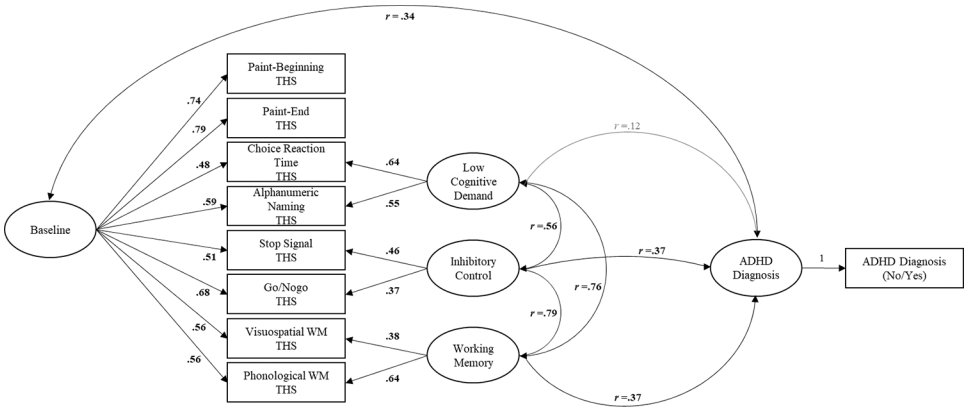

Figure 1.

Standardized structural model with ADHD status (0=no, 1=yes) correlated with the bifactor s-1 factor general factor (baseline hyperactivity) and specific factors (hyperactivity during low cognitive demand, inhibitory control, and working memory conditions). Standardized loadings are shown (all $p<.001$). Significant correlations ($p<.05$) with ADHD diagnosis and between specific factors are **bold**; nonsignificant correlations are **grey**. Age, sex, and SES are controlled but not depicted for clarity.

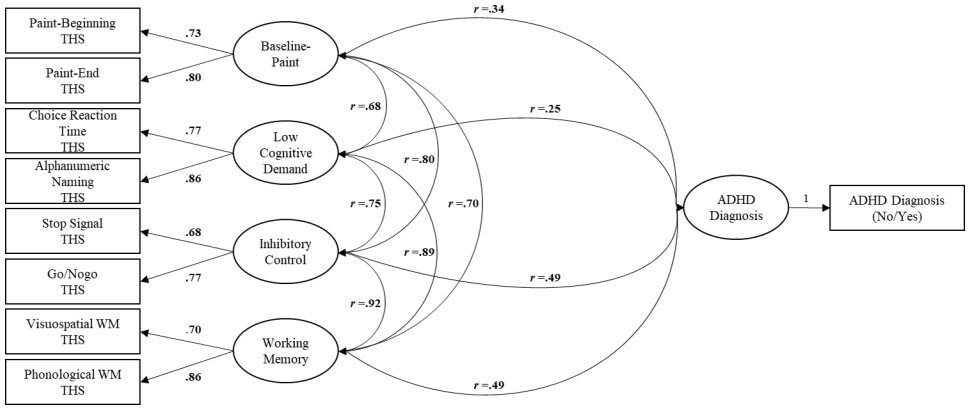

Figure 2.

Standardized structural model with ADHD status (0=no, 1=yes) correlated with the 4 cognitive conditions factors (hyperactivity during baseline paint, low cognitive demand, inhibitory control, and working memory). Standardized loadings are shown (all p<.001). All correlations with ADHD diagnosis and factors are significant (p<.05) and **bold**. Age, sex, and SES are controlled but not depicted for clarity.

Table 1.

Demographic characteristics.

	ADHD		Non-ADHD			
Variable	***M***	***SD***	***M***	***SD***	***Cohen's d***	***p***
N (Girls/Boys)	36/83		25/40		--	.33
Ethnicity/Race (A/B/H/M/W)	0/18/6/8/87		1/6/7/9/42		--	.11
VCI	103.81	14.29	106.13	11.38	0.17	.26
Age	10.24	1.48	10.70	1.49	0.31	.04
SES	47.83	11.15	49.71	11.43	0.17	.29
Maternal Education Level (HS/A/B/G)	14/23/49/33		5/7/27/26		--	.21
BASC-2/3: Attention Problems (Raw Scores)						
Parent	14.27	3.02	10.95	4.91	0.87	<.001
Teacher	16.51	4.11	11.17	6.32	1.07	<.001
BASC-2/3: Hyperactivity/Impulsivity (Raw Scores)						
Parent	16.33	6.14	10.90	5.72	0.91	<.001
Teacher	15.47	9.13	7.60	7.40	0.92	<.001
Actigraph Data (THS)						
Paint-Beginning	37.16	29.95	25.61	24.53	0.41	.01
Paint-End	59.06	47.77	38.30	28.46	0.49	<.001
Alphanumeric Naming	178.15	142.16	133.12	97.69	0.35	.03
Choice Reaction Time	103.89	77.61	66.96	52.63	0.53	<.001
Go/No-go	127.48	92.08	81.41	63.84	0.55	<.001
Stop Signal	98.34	90.94	52.86	51.28	0.57	<.001
Global-Local	151.40	110.15	89.79	73.30	0.62	<.001
Number-Color	125.87	95.32	78.42	58.45	0.56	<.001
Visuospatial Working Memory	172.41	105.90	106.58	78.25	0.68	<.001
Phonological Working Memory	246.41	151.81	157.30	101.25	0.66	<.001

Note: Ethnicity: A = Asian, B = Black, H = Hispanic, M = Multiracial, W = White Non-Hispanic. VCI = Verbal Comprehension Index. Maternal Education Levels: HS = High School diploma or equivalent, A = at least 1 year of college, Associate's degree or specialized training, B = Bachelor's/4-year college degree, G = Graduate degree, BASC = Behavior Assessment System for Children. THS = Total Hyperactivity Score.

Table 2.

Measurement Models with Covariates of Age, Sex, and SES

Model	CFI	TLI	RMSEA (90%CI)	SRMR	AIC	BIC	General factor loadings	Specific factor loadings
Initial Single Factor Model (10 THS indicators)	0.92	0.90	.07 (.05–.09)	0.05	19514.13	19619.68	.67–84, all *p*<.001	--
Revised Single Factor Model (8 THS indicators)	0.95	0.93	.06 (.03–.08)	0.04	15459.31	15545.67	.63–.81, all *p*<.001	--
Revised 3-Correlated Factor Model	0.97	0.95	.05 (.02–.07)	0.04	19485.22	19619.55	--	.66–.84, all p<.001
Revised 4-Correlated Factor Model	1.00	1.00	.00 (.00–.06)	0.03	15438.36	15572.70	--	.66–.85, all p<.001
Revised 4-Factor Bifactor (s-1) Model (1 general factor, 3 specific factors)	1.00	0.99	.02 (.00–.07)	0.02	15441.03	15584.97	.54–.76, all p<.001	.36–.62, all p<.001

Note. Modified Single Factor Model omitted THS indicators from both of the set shifting tasks. AIC = Akaike Information Criteria, BIC = Bayesian Information Criteria, CFI = comparative fit index, RMSEA = root mean square error of approximation, SRMR = standardized root mean square residual, TLI = Tucker-Lewis index.

Table 3.

Structural Models: Associations between ADHD diagnosis and hyperactivity (correlated bifactor s-1 model) with covariates

Model	CFI	TLI	RMSEA (90%CI)	General Factor Loadings	Specific Factor Loadings
Revised 4-Factor Bi-factor (s-1) Model	.99	.97	.04 (.00–.08)	Baseline: .48–.79, all p<.001	LowCog: .55–.64, all p<.001
					IC: .37–.46, all p<.001
					WM: .38–.64, all p<.001
Revised 4-Correlated Factor Model	.95	.89	.09 (.07–.12)	--	Paint: .73–.80, all p<.001
					LowCog: .77–.86, all p<.001
					IC: .68–.77, all p<.001
					WM: .70–.86, all p<.001

Note. AIC = Akaike information criteria, BIC = Bayesian information criteria, CFI = comparative fit index, RMSEA = root mean square error of approximation, SRMR = standardized root mean square residual, TLI = Tucker-Lewis index.