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Is hyperactivity ubiquitous in ADHD or dependent on environmental demands? Evidence from meta-analysis

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Abstract

Hyperactivity, or excess gross motor activity, is considered a core and ubiquitous characteristic of ADHD. Alternate models question this premise, and propose that hyperactive behavior reflects, to a large extent, purposeful behavior to cope with environmental demands that interact with underlying neurobiological vulnerabilities. The present review critically evaluates the ubiquity and environmental modifiability of hyperactivity in ADHD through meta-analysis of 63 studies of mechanically measured activity level in children, adolescents, and adults with ADHD relative to typically developing (TD) groups. Random effects models corrected for publication bias confirmed elevated gross motor activity in ADHD ($d = 0.86$); surprisingly, neither participant age (child vs. adult) nor the proportion of each ADHD sample diagnosed with the Inattentive subtype/presentation moderated this effect. In contrast, activity level assessed during high cognitive load conditions in general ($d = 1.14$) and high executive functioning demands in particular ($d = 1.39$) revealed significantly higher effect sizes than activity level during low cognitive load ($d = 0.36$) and in-class schoolwork ($d = 0.50$) settings. Low stimulation environments, more rigorous diagnostic practices, actigraph measurement of movement frequency and intensity, and ADHD samples that included fewer females were also associated with larger effects. Overall, the results are inconsistent with DSM-5 and ADHD models that a) describe hyperactivity as ubiquitous behavior, b) predict a developmental decline in hyperactivity, or c) differentiate subtypes/

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presentations according to perceived differences in hyperactive behavior. Instead, results suggest that the presence and magnitude of hyperactive behavior in ADHD may be influenced to a considerable extent by environmental factors in general, and cognitive/executive functioning demands in particular.

Attention-deficit/hyperactivity disorder (ADHD) is a complex, chronic, and heterogeneous neurodevelopmental disorder characterized by a triad of cardinal behavioral features that include inattention, impulsivity, and hyperactivity. Hyperactivity is a multifaceted construct that spans a broad range of verbal and physical behaviors, with excess gross motor movement forming a key component as evidenced by its explicit inclusion in 4 of the 6 DSM-5 ADHD hyperactivity symptoms (APA, 2013). This excess gross motor activity has been of longstanding interest and subjected to considerable empirical scrutiny using a broad range of methodologies (Tryon, 1991). While early approaches relied on rating scales (Werry, 1968), direct observations (Abikoff & Gittelman, 1984; Whalen et al., 1978), and floor grid-crossing counts (Milich et al., 1982), an expanding number of technologically sophisticated methods have followed, including pedometers (Plomin & Foch, 1981), ultrasonic sensors (Saxon et al., 1977), stabilometric cushions (Conners & Kronsberg, 1985), infrared motion analysis (Teicher et al., 1996), actigraphs (Halperin et al., 1992), and video compression algorithms (Wehrmann & Muller, 2015).

Subjective measures remain the most frequent indices of the hyperactivity construct (e.g., symptom ratings, clinical interviews), and suggest psychometrically distinct but temporally unstable ADHD subtypes/presentations definable by the quantity/severity of hyperactive symptom ratings (Nigg et al., 2010; Valo & Tannock, 2010). In contrast, mechanical methods consistently indicate elevated gross motor activity across all ADHD subtypes/presentations (Bauermeister et al., 2005; Dane et al., 2000; Miyahara et al., 2014), as well as longitudinally for both ADHD persisters and remitters (Cheung et al., 2015; Halperin et al., 2008). This discrepancy highlights the importance of objective methods for clarifying the role of excess gross motor activity in ADHD, and may reflect the modest agreement between subjective and direct measures of gross motor activity ($r = .32$ to $.58$; Rapport et al., 2006), informant reporting biases (Harris & Lahey, 1982), the superior reliability of mechanical measures ($r = .90$ to $.99$; Tryon, 1985), and/or difficulties psychometrically differentiating hyperactivity ratings from distinct behavioral dimensions such as impulsivity and inattention (DuPaul et al., 2015).

The converging evidence suggesting elevated gross motor activity across ADHD-Combined and ADHD-Inattentive subgroups (Bauermeister et al., 2005; Dane et al., 2000; Hartanto et al., 2015; Miyahara et al., 2014) appears to contradict the prevailing DSM-5 clinical view (APA, 2013), and suggests that hyperactivity may be a cross-subtype and relatively homogeneous feature of ADHD despite clear differences in subjective perceptions regarding its presence/severity. However, it appears premature to describe hyperactivity as a ubiquitous feature of ADHD due to substantial between-study differences in the presence and magnitude of excess motor movement relative to non-ADHD comparison groups. For example, studies employing mechanical technologies have characterized individuals with ADHD as less active (Plomin & Foch, 1981), minimally different (Bauermeister et al.,

2005), moderately more active (Halperin et al., 1992), or highly active relative to controls (Marks et al., 2005). Stated differently, mechanical measurement of hyperactive behavior suggests that the magnitude of the hyperactivity deficit is somewhere between -0.59 standard deviations (ADHD group less active than typically developing [TD] peers; Plomin & Foch, 1981) and $+3.45$ standard deviations (almost complete non-overlap of the ADHD-TD distributions; Marks et al., 2005). Although this variation does not appear attributable to between-study differences in ADHD subtypes/current presentations (Dane et al., 2000), additional methodological differences warrant scrutiny. In particular, this marked between-study heterogeneity may be related to vast differences in the tasks, tests, and activities in which participants were engaged while their motor activity was being measured—ranging from highly controlled laboratory sessions (Marks et al., 2005) and in-seat academic work (McGrath et al., 2004) to recess/physical education (Okada et al., 2013) and television watching (Porrino et al., 1983). As such, a unique contribution of the current meta-analysis is the systematic examination of demographic, methodological, and environmental factors associated with between-study differences in the magnitude of ADHD-related hyperactivity (Lipsey & Wilson, 2001).

Understanding the extent to which environmental factors provoke or rarefy hyperactive behavior in ADHD is critical for refining theoretical conceptualizations of ADHD and clarifying the disorder's etiology, course, and pathophysiology. Childhood hyperactive behavior predicts adult impairment (Mannuzza et al., 2002), and the contextual variability of ADHD-related behavior is increasingly being embraced (Dirks et al., 2012; Kofler et al., 2013) despite a lack of recognition among contemporary accounts of ADHD. Indeed, competing ADHD etiological models make disparate predictions regarding the underlying mechanisms and processes responsible for hyperactivity's ubiquity or contextual variability. As summarized in Table 1, many contemporary models of ADHD largely disregard the role of hyperactivity, envision it as corollary behavior that accompanies frequent attentional shifts (Sagvolden et al., 2005), or view it as ubiquitous behavior (APA, 2013; Barkley, 1997). Support for ubiquitous deficit models includes evidence that most, but not all, studies find significantly elevated gross motor activity in children with ADHD based on objective, mechanical measurement across a broad range of settings including home (Porrino et al., 1983), school (Imeraj et al., 2011), community (Baird et al., 2012), laboratory (Dane et al., 2000; Rapport, Bolden, et al., 2009), and clinic settings (Murillo et al., 2015; De Crescenzo et al., 2015).

In contrast to ubiquitous deficit models, alternate models describe hyperactivity as functional behavior driven in part by environmental factors that interact with underlying neurobiological vulnerabilities. These models describe hyperactive behavior as stimulation-seeking behavior secondary to chronic underarousal (Zentall & Zentall, 1983), or postulate that hyperactivity reflects compensatory behavior secondary to interactive effects of chronic cortical underarousal and environmental demands that overwhelm these individuals' underdeveloped working memory abilities (Rapport et al., 2001). The optimal stimulation model hypothesizes that hyperactive behavior occurs in response to low stimulation but not high stimulation environments (Zentall & Zentall, 1983). Functional working memory model predictions are consistent with this hypothesis, but further specify that hyperactive behavior reflects compensatory or escape/avoidance behavior in the face of environmental

demands that challenge or overwhelm, respectively, these children's underdeveloped neurocognitive abilities (Rapport, Bolden et al., 2009). Support for these stimulation/cognitive demand models includes demonstrations that children with ADHD are more motorically active than their peers during working memory but not control tasks (Rapport, Bolden et al., 2009), during academic but not non-academic activities (Porrino et al., 1983), and in low stimulation but not high stimulation environments (Antrop et al., 2005; Zentall & Zentall, 1983). However, relatively few ADHD studies have directly compared activity level during high and low cognitive demands, or under high versus low environmental stimulation conditions, which limits conclusions regarding the extent to which hyperactive behavior is ubiquitous or an outcome of external influences.

Need for a meta-analytic review

Although mechanically measured gross motor activity has been reported in 63 ADHD studies to date, conclusions regarding the ubiquity and underlying mechanisms of hyperactive behavior are limited because relatively few studies have systematically manipulated cognitive demands and/or varied environmental stimulation levels (within-study manipulations). In contrast, there currently exists a large body of evidence examining activity level across a wide range of contexts that can be compared systematically and quantitatively to address this critical issue (between-study comparisons). In addition, meta-analysis allows us to empirically investigate the role of competing explanations—namely, methodological and participant demographic differences across studies—that may parsimoniously explain the substantial differences in obtained results across studies. Two recent meta-analyses suggest greater activity level relative to non-ADHD comparison groups (De Crescenzo et al., 2015; Murillo et al., 2015). However, these reviews included only a small subset of available studies (13% and 29% of available studies, respectively), and were thus unable to address critical issues regarding the role of environmental factors (cognitive demands, stimulation) on objectively measured hyperactivity.

In summary, the current meta-analysis is a comprehensive review of 63 studies of mechanically measured activity level in preschoolers, children, adolescents, and adults with ADHD relative to typically developing comparison groups. Through meta-analytic synthesis, analysis, adequately powered moderator investigation, and best vs. worst case analysis (Lipsey & Wilson, 2001), the current review seeks to inform current debate regarding the nature and environmental modifiability of ADHD-related hyperactivity, with implications for the evaluation of etiological models, assessment practices, and treatment interventions for children and adults with ADHD.

Method

Literature Searches

A three-tier literature search was conducted using Medline, PubMed, PsycInfo, PsycArticles, PsycBooks, ERIC, Google Scholar, Dissertation Abstracts International, and Social Science Citation Index. Search terms included permutations of the ADHD diagnostic label (ADHD, ADD, attention deficit, attention problems, inattent*, hyperact*, hyperkinesis, minimal brain dysfunction/damage, MBD), mechanical, activity level, gross motor activity,

and mechanical technologies frequently used to objectively measure activity level (actometer, actigraph, accelerom*, infrared motion, QbTest, MMAT, stabilometer, stabilometric cushion, motionlogger). An asterisk following a root word instructs search engines to look for any derivative of the word that is followed by the asterisk. No search delimiters were selected to avoid missing studies due to database misclassification. To further expand the initial study base, the options “apply related words” and “also search within the full text of the articles” were selected across all databases. Searches were conducted independently by three, PhD-level authors (MJK, DES, JSR) and repeated until no new studies were located. Studies considered for inclusion were reviewed by at least two of these three authors; data was extracted by MJK and reviewed by DES and JSR. Disagreements were resolved via discussion after all three authors reviewed the study (100% consensus). After the initial searches, studies cited by articles reporting mechanical activity measurement in ADHD were examined (Phase II backward search), and a forward search (Phase III) was conducted using the Social Science Citation Index to locate studies citing those that reported mechanical activity measurement in ADHD. In addition, emails were sent to authors of studies published within the last 5 years that investigated mechanically measured activity level but did not report sufficient data for effect size calculation. These procedures generated 840 peer-reviewed studies, dissertations, and unpublished manuscripts written since 1959. All search processes were completed and study recruitment was closed on September 19, 2015.

Inclusion and Exclusion Criteria

Inclusion and exclusion criteria are described below, with the number of studies omitted for each criterion in parentheses. Several studies failed to meet multiple inclusion criteria; the counts below reflect the first failed criteria identified. The following served as inclusion criteria for the review: (a) English language (1) studies of (b) children, adolescents, and/or adults with a primary diagnosis of ADHD or related labels (e.g., hyperactive, attention problems) completing one or more activities during which activity level was mechanically assessed during a non-medication condition (baseline or placebo assessment condition) (494); (c) inclusion of a typically developing control group (85); and (d) mechanically measured activity level data reported, or statistics reported from which effect size can be estimated (103). Exclusion criteria included: (a) participants with gross neurological, sensory, or motor impairment, history of a seizure disorder, psychosis, autism, or intellectual disability, or estimated intelligence < 80 (6); (b) repeat data (e.g., study published in journal and as book chapter; follow-up longitudinal study) (14); and (c) mechanical activity assessment during sleep or fMRI only, or reporting behavioral coding/ratings by human (non-mechanical) observers (74).

For studies reporting repeat data with the same task(s) and instrument(s) (e.g., actigraphs), the newest study with the largest sample size was included. For repeat studies reporting different activity level instruments or different tasks with an overlapping sample, data from both studies were included but coded as part of the same study to provide data across as many tasks/conditions as possible while maintaining the independence assumption (Lipsey & Wilson, 2001). In all cases, decisions were made prior to effect size calculation to minimize experimenter bias.

A total of 63 studies from 1975 to 2015 met study criteria and were included in one or more sets of analyses. These 63 studies (60 published studies, 3 dissertations) provided 270 total effect sizes. Twelve of these 63 studies reported data for two or more independent subsamples (defined as ADHD samples with non-overlapping participants), resulting in a total study size of $k = 78$.

Coding of Moderators

Moderator variables were coded according to the characteristics reported in Table 6. Categorical variables were used to maintain consistency across moderators (Hedges & Pigott, 2004) because many studies reported data across multiple levels of the same potential moderator (e.g., movement frequency and intensity during the same task). Categorical variables were coded hierarchically, wherein higher values are associated with an addition to the variable in question (e.g., adding informants for diagnostics, including higher proportion of females).

Study characteristics—Activity measurement occurred in laboratory, school, and community settings (Setting) using actigraphs, actometers, infrared motion detection systems, pedometers, and ultrasonic sensors (Technology) that provided data on movement frequency, intensity, duration, and distance/area (Movement Type). Body Placement of the sensors was coded as ankle, wrist, trunk/shoulders/core, multiple (e.g., data collapsed across wrist and ankle actigraphs), and external (e.g., ultrasonic sensors). Gender composition was coded as greater or fewer than 25% females to create approximately equal cell sizes. Age was initially coded as preschool, child, adolescent, or adult based on the reported age range; analyses focused on child vs. adult samples given the relative paucity of preschool and adolescent studies. Diagnostic Method was coded as an index of study quality based on the recommendations for gold standard diagnosis of ADHD used to code study quality in previous meta-analytic reviews (Alderson et al., 2007; Kofler et al., 2008; Lipszyc & Schachar, 2010). Studies were classified into dichotomous groups, wherein higher values reflect more rigorous diagnostic procedures: 0 = single informant methods (referral or previous diagnosis only, single informant questionnaire and/or interview); 1 = multiple informants and settings (multiple informant report based on standardized and normed questionnaires, with or without gold standard semi-structured/structured clinical interview). ADHD Subtype/Presentation was coded as a continuous variable based on the percentage of participants diagnosed with the Inattentive relative to Combined and Hyperactive/Impulsive subtypes/presentations (% ADHD-I). Comorbidity was coded as the percentage of the ADHD sample with one or more comorbid diagnoses.

Situational moderators: Cognitive demands—Cognitive Demands during activity level measurement were coded as High (e.g., neurocognitive tests of attention, working memory, inhibition), Low (e.g., painting, recess, free play, television watching), Mixed, or Schoolwork. Sessions were coded as Mixed if the activity measurement was collapsed across a combination of tasks/activities that would be coded as high and low (e.g., activity data collapsed across a psychoeducational battery that included cognitive tests, questionnaires, a clinical interview, and break periods). Finally, in-class Schoolwork was coded separately to allow direct comparison to environments with known high and low

cognitive demands. High cognitive demands were further classified as Executive Function (EF; working memory, inhibition, set shifting) and Non-EF tasks (e.g., attention, reaction time) based on the influential Miyake et al. (2012) model of executive functions (Conway et al., 2005).

Situational moderators: Environmental stimulation—External environmental stimulation was similarly coded as High, Low, or Mixed/Unknown. Laboratory/clinic testing was coded as Low stimulation unless described otherwise by the study authors. Settings were coded as Mixed/Unknown if activity data were collapsed across multiple settings that would be coded High and Low, or if the setting was not reported (e.g., all waking hours). Activity measurement occurring in classroom settings was coded as High stimulation as recommended (Rapport, Kofler et al., 2009; Zentall & Zentall, 1983).

Moderator analyses were conducted progressively using a contingent approach, wherein basic demographic and methodological variables (e.g., age group) were analyzed first using the mixed effects, maximum likelihood Analog to ANOVA (categorical variables) and Meta-Regression (continuous variables) approaches recommended by Lipsey and Wilson (2001). Cognitive demands and environmental stimulation were then analyzed based on significant overall heterogeneity that was not explained more parsimoniously by these control variables.

Computation of Effect Sizes

Means, *SDs*, and sample sizes for each group were used to compute Cohen's *d* effect sizes using Comprehensive Meta-Analysis version 2.2 (Biostat, Englewood, NJ, USA). When these data were unavailable, effect sizes were estimated using reported test statistics. For between-group comparisons, these statistics included each group's sample size and *t* or *p* values, each group's means and the comparison *p* value, or reported effect sizes converted to Cohen's *d*. Cohen's *d* effect sizes were corrected using the Hedges' *g* formula to correct for study sample size due to the upward bias in effect size magnitude of small *N* studies. Cohen's *d* effect sizes are in standard deviation units, such that an effect size of 1.0 indicates that two groups differ by one standard deviation (Zakzanis, 2001). An effect size of 0.2 is interpreted conventionally as small (detectable only through statistics), 0.5 is medium (detectable to a careful observer), and 0.8 is large (obvious to any observer; Cohen, 1988). Overall effect sizes were computed under a random effects model in which each study is weighted by its inverse variance weight ($1/SE^2$) to correct for study-level sampling error as recommended (Hunter & Schmidt, 2004; Lipsey & Wilson, 2001). No corrections to the group assignment variable were conducted given our goal of assessing diagnostic rigor as a potential moderator following previous ADHD meta-analyses (Alderson et al., 2007; Kofler et al., 2008; Lipszyc & Schachar, 2010).

Multiple effect sizes—Most studies reported data sufficient to calculate multiple effect sizes. The most common reasons included reporting activity level data across multiple tasks, reporting multiple activity level metrics, or both. Separate effect sizes were calculated for each task and metric to be comprehensive and allow studies to be included in as many analysis subsets as possible. To meet the independence assumption, only one effect size from each study was used to calculate each omnibus effect size (Lipsey & Wilson, 2001). Each

study's effect size reflected the average of all relevant comparisons from that study for that particular analysis.

Publication bias: The file drawer problem—Four studies did not provide data sufficient to calculate effect sizes for one or more comparisons, but reported no significant between-group differences. These studies were retained in the analysis and assigned an effect size of 0.00 for those comparisons because omitting them would artificially inflate overall effect size estimates due to publication bias (Rosenthal, 1995). In addition, three studies reported insufficient data for effect size calculation, but either published supplementary data online from which this data could be estimated (2), or their authors responded to email queries and provided data (1). Four tests of publication bias were used for each analysis subtest (Fail-safe N , Begg & Mazumdar's rank correlation test, Egger's test of the intercept, and Duval & Tweedie's trim-and-fill procedure; Lipsey & Wilson, 2001). For analyses where significant publication bias was detected, overall effect sizes were corrected using the methods described by Duval and Tweedie (2000).

Results

Overview

We initially report overall ('moderator-independent') effect sizes, followed by heterogeneity tests to determine whether moderator analyses are warranted. Subsequently, we analyze potential methodological and demographic moderators to inform inclusion criteria for subsequent analyses. Finally, we examine cognitive demands and environmental stimulation as potential moderators based on significant between-study heterogeneity that could not be accounted for by demographic or methodological factors. A histogram of obtained effect sizes is reported in Table 5. All analyses are based on random effects models; effect sizes are corrected for publication bias (Duval & Tweedie, 2000; Hunter & Schmidt, 2004) and summarized in Tables 2–5.

Activity Level: ADHD vs. Typically Developing Group Comparisons

A total of 63 studies ($k = 78$ independent subgroups) reporting data on 1,894 individuals with ADHD and 2,748 typically developing control participants were included in the analyses.

Moderator-independent activity level differences—As shown in Table 2, individuals with ADHD exhibited an overall large magnitude increase in mechanically-measured activity level relative to TD groups ($d = 0.86$, 95% CI = 0.73 to 0.99). The overall test of homogeneity was significant, suggesting that there is more between-study variance among effect sizes than would be expected based on study-level error alone, and supporting the analysis of potential moderators ($Q[77] = 248.98$, $p < .0001$).

Methodological and demographic moderators of between-study differences—Based on the tiered approach described above, the demographic and methodological variables (ADHD subtype/presentation, comorbidity, age, technology, movement type, body placement, setting, diagnostic method, and percent female) were examined initially to

inform inclusion criteria for subsequent analyses. Because many studies reported multiple metrics (e.g., reported separate results for frequency and intensity, or ankle and wrist placement), we elected to compute effect sizes separately for each moderator subgroup and conduct a series of planned comparisons to compare across moderator subgroups using the *z*-score test of equality (Patnoster et al., 1998). This method was selected for practical reasons as a compromise between meeting the independence assumption (Rosenthal, 1995) and including as many studies as possible in moderator analyses (Kofler et al., 2013).

Bias-corrected results indicated no significant differences among most comparisons (all $p > .05$ unless described below; Table 2). Specifically, effect size magnitude did not vary significantly based on the percentage of the ADHD group diagnosed as ADHD-Inattentive subtype/presentation ($p = .46$, $k = 54$) or the percentage of the sample with comorbid diagnoses ($p = .69$, $k = 27$). Effect sizes also did not vary across child and adult samples ($p = .11$), diagnostic method (single vs. multiple informants; $p = .08$), technology type (actigraph/actometer vs. infrared motion; $p = .07$) or body placement (ankle, wrist, trunk head; all $p > .07$). In contrast, effect sizes were moderately larger for movement frequency and distance relative to intensity and duration ($d = 0.82$ and 0.71 vs. 0.49 and 0.48 , respectively; all $p < .04$), lab settings relative to school/community settings ($d = 0.92$ vs. 0.27 ; $p < .0001$), and studies including a lower versus higher proportion of female participants with ADHD ($d = 0.87$ vs. 0.57 ; $p = .008$). In all cases, significant between-study heterogeneity remained ($p < .0005$), suggesting that additional moderator analyses were warranted.

Cognitive demands and environmental stimulation as moderators of between-study differences—Results revealed that Cognitive Demands exerted a significant impact on obtained effect sizes, with significantly larger effect sizes obtained during High cognitive demand conditions ($d = 1.14$) relative to Mixed demands ($d = 0.70$), which were in turn larger than Low demands ($d = 0.36$) and Classroom environments ($d = 0.50$; all $p < .007$). Low and Classroom environments did not differ ($p = .12$; $H > M > L = C$). No significant between-study heterogeneity remained for the Classroom condition ($p = .88$), whereas significant variation among High and Low cognitive load studies remained ($p < .001$). To further explore this heterogeneity, the high cognitive load tasks were categorized as Executive Functioning ($d = 1.39$) vs. Non-EF Cognitive Tasks ($d = 0.71$), which differed significantly ($p = .01$). Inspection of Table 3 suggests that the remaining heterogeneity ($p < .0005$) among EF tasks may be related to higher effect sizes during working memory ($d = 1.35$) relative to inhibition ($d = 0.49$) tasks ($p = .02$), but this conclusion must be considered tentative due to the low number of studies reporting activity level during tasks tapping each EF (Table 3).

The Analog to ANOVA for Environmental Stimulation was significant, with Low Stimulation ($d = 1.01$) environments associated with significantly higher effect sizes than High Stimulation environments ($d = 0.54$; $p < .0001$). Significant heterogeneity among Low ($p < .0005$) but not High ($p = .88$) stimulation environments remained. As expected, many but not all conditions coded as Low Stimulation were also coded as High Cognitive Load (above) (e.g., laboratory testing sessions). To test potential additive effects of cognitive demands and environmental stimulation, we compared High Cognitive/Low Stimulation ($d =$

1.15) to Mixed Cognitive/Low Stimulation environments ($d = 0.80$), which differed at $p = .02$.¹ This finding parallels the main effect of High vs. Mixed Cognitive Demands (above), and suggests that the larger effect sizes obtained during Low Stimulation conditions may be attributable at least partially to the High Cognitive Demand tasks that participants are performing in these (typically) low stimulation laboratory settings.

Significant heterogeneity remained among the 30 studies reporting activity data during High Cognitive/Low Stimulation settings ($p < .0005$). Moderator analysis of these studies were therefore repeated using the methodological and demographic moderators identified above (Table 4). The pattern of results was highly similar to the overall findings. During cognitive testing in low stimulation environments, participants with ADHD exhibited larger magnitude increases in activity level relative to their peers when studies used actigraphs ($d = 1.30$, $k = 17$) relative to infrared motion detection ($d = 0.70$, $k = 12$; $p = .003$); measured frequency ($d = 1.23$, $k = 18$) or intensity ($d = 0.97$, $k = 7$) relative to duration ($d = 0.50$, $k = 5$; both $p < .001$); and grouped participants based on multiple informants ($d = 1.31$, $k = 19$) relative to single informant diagnostic methods ($d = 0.68$, $k = 12$; $p < .0005$). Interestingly, participant age did not moderate the magnitude of mechanically measured hyperactivity for ADHD relative to non-ADHD groups ($p = .25$). Significant heterogeneity remained within at least one subgroup for each comparison, however, suggesting that additional, unmeasured factors may influence the magnitude of observed differences in activity level across studies.

Best case estimation—Collectively, the current findings suggest that ADHD/non-ADHD differences in mechanically measured activity level may be maximally detected during tasks with high cognitive demands completed in relatively low stimulation environments. In particular, tasks with high executive functioning demands in general, and potentially working memory demands specifically, may maximally differentiate the groups. In addition, diagnosing participants based on multiple informants and using actigraphs to measure movement frequency and/or intensity were associated with larger effect sizes across studies. Of the 63 studies included in the meta-analysis, 6 studies reported this ‘best case’ combination of methodological, demographic, high cognitive load, and low environmental stimulation variables (Lipsey & Wilson, 2001). The bias-corrected, weighted mean effect size across these 6 studies was very large ($d = 1.47$, 95% CI = 0.59 to 2.35). Five of these studies included fewer than 25% females ($d = 1.63$) (Alderson et al., 2012; Hudec et al., 2015; Konrad et al., 2000; Marks et al., 2005; Rapport et al., 2009) and one included greater than 25% females ($d = 0.94$) (Hudec et al., 2014).

Worst case estimation: Is hyperactivity ubiquitous in ADHD?—Finally, we estimated the magnitude of ADHD/non-ADHD differences in mechanically measured activity level under conditions opposite those described for the best case. We identified 15 studies that included the combination of low cognitive demands or schoolwork, high stimulation, and school or community settings. The bias-corrected, weighted mean effect size across these 15 studies was small-to-medium but significant ($d = 0.44$, 95% CI = 0.29 to

¹We were unable to conduct the more direct comparison, High Cognitive/Low Stimulation vs. Low Cognitive/Low Stimulation, because no studies met criteria for the latter category. In addition, we were unable to compare High vs. Low Stimulation during High Cognitive Demands because all tasks with high cognitive demands occurred in low stimulation laboratory settings.

0.59; between-study heterogeneity *ns* at $p = .95$). We note that the above results also suggested smaller effect sizes when measuring movement duration and/or using mechanical technologies other than actigraphs; however, there were insufficient studies that used these methods and also met the cognitive demands/stimulation criteria. Collectively, these results suggest that elevated activity level remains present, albeit subdued, for individuals with ADHD even under 'ideal' conditions. Importantly, however, the magnitude of this elevation was modest; conventional effect size interpretation suggests that it may be difficult to detect without careful observation (Cohen, 1988).

Discussion

The current meta-analysis was the first to examine model-driven predictions regarding the role of cognitive demands and environmental stimulation on gross motor activity among children, adolescents, and adults with ADHD, while controlling for publication-bias, sampling error, and methodological differences across studies. The overall effect size of 0.86 indicated that individuals with ADHD exhibit large magnitude increases in gross motor activity relative to non-ADHD controls. This overall large magnitude effect was highly consistent with previous meta-analytic estimates, despite our control for publication bias and inclusion of more than three times the number of studies included in either previous meta-analysis ($d = 0.64$ to 0.92 ; De Crescenzo et al., 2015; Murillo et al., 2015). However, significant heterogeneity was detected, such that individuals with ADHD were characterized across studies as slightly less active (Okada et al., 2013), minimally different (Bauermeister et al., 2005), moderately more active (Halperin et al., 1992), or highly active relative to controls (Marks et al., 2005). A unique contribution of the current study was the systematic examination of demographic, methodological, and environmental factors associated with this between-study heterogeneity to inform debate regarding the role of hyperactivity in ADHD (Lipsey & Wilson, 2001).

The current review identified several demographic and methodological factors that were associated with obtained results across studies, with similar findings across the overall and high cognitive/low stimulation analyses. In particular, larger effects were obtained for studies defining their ADHD sample based on multiple informants ($d = 1.31$) relative to single informants ($d = 0.68$), particularly during high cognitive demand/low stimulation testing. This finding highlights the importance of comprehensive diagnostic procedures, and suggests that mono-informant methods may blunt detection of increased ADHD-related activity level. Interestingly, this finding appears at odds with previous meta-analytic reviews that found more comprehensive diagnostic procedures were associated with *smaller* effect sizes for laboratory tests of behavioral inhibition (Alderson et al., 2007; Lijffijt et al., 2005), delay aversion (Patros et al., 2015), and classroom observations of attentive behavior (Kofler et al., 2008). These authors hypothesized that the exaggeration of effects with less rigorous diagnostics was likely due to the inadvertent inclusion of non-ADHD children (with other forms of psychopathology) in the ADHD group, which interestingly decreased within-group variance when considering the high behavioral and cognitive performance variability associated with ADHD (Alderson et al., 2007). In contrast, the current results are congruent with previous ADHD subtype comparisons and indicate that actigraph-measured activity level may be similarly elevated across ADHD subtypes/presentations (Bauermeister et al.,

2005; Dane et al., 2000; Hartanto et al., 2015; Miyahara et al., 2014), despite clear differences in informant behavior ratings. In addition, actigraphy has been shown to differentiate between ADHD and clinical control groups (Matier-Sharma et al., 1995). Thus, diagnostic methods that better exclude false positive cases may exacerbate (current study), blunt (Alderson et al., 2007), or minimally affect (Kofler et al., 2013) between-group differences in ADHD studies depending on the degree of between- and inter-individual variability in the outcome of interest. In the case of mechanically assessed hyperactivity, it appears that more rigorous diagnostic procedures may result in a more homogeneous ADHD group, which would in turn increase effect size magnitude.

The current findings suggest that actigraphs ($d = 1.30$) may have improved sensitivity relative to infrared motion technologies ($d = 0.70$) for detecting hyperactivity during high cognitive/low stimulation conditions. Interestingly, the recent meta-analysis by Murillo and colleagues (2015) found similar performance between infrared motion detection ($d = 0.92$) and actigraphy ($d = 0.64$). Inspection of the studies and effect sizes reported in that meta-analysis suggests that this discrepancy may be associated with our correction for publication bias (Duval & Tweedie, 2001; Hunter & Schmidt, 2004), the increased number of studies employing each technology, and/or the use of infrared motion technology exclusively during continuous performance tests that were associated with lower effect sizes than lab-based executive functioning tests. Similarly, we found that activity frequency ($d = 1.23$) and intensity data ($d = 0.97$) were associated with larger effects than duration ($d = 0.50$) and distance data ($d = 0.71$), particularly under the high cognitive demand/low external stimulation conditions described below. This finding highlights the importance of examining the topography of movement in ADHD, particularly to understand the disconnect between objective measurement of activity level and behavioral ratings of hyperactivity (Sarver et al., 2015). For example, objective and subjective activity measures correlate only moderately (.32-.58; Rapport et al., 2006), and up to two out of three children rated as hyperactive by their teachers are motorically less active than at least one child rated as normally active (Tryon & Pinto, 1994). Similarly, ADHD subtypes/presentations differentiated based on hyperactivity ratings show similar, high levels of mechanically assessed activity level both cross-sectionally (Bauermeister et al., 2005; Dane et al., 2000; Hartanto et al., 2015; Miyahara et al., 2014) and longitudinally (Cheung et al., 2015; Halperin et al., 2008). Taken together, these findings suggest that hyperactivity in ADHD as perceived by parents, teachers, and other informants likely reflects more than raw activity level. The current findings suggest that movement topography (Teicher et al., 1996), along with other behaviors including task attention (Kofler et al., 2008; Sarver et al., 2015) and verbally intrusive behavior (Pelham et al., 2005), may be important for dissecting hyperactivity in ADHD and understanding the factors that influence whether or not high frequency/intensity movement is interpreted by observers as 'hyperactive'.

Interestingly, no subtype/presentation effects were found in the current meta-analysis, and highly similar effect sizes were obtained from child relative to adult samples. These findings were surprising given the prevailing DSM clinical model suggesting that hyperactivity in ADHD decreases across the lifespan, and appears inconsistent with the DSM-5 decision to relax the hyperactivity symptom quantity required for adult relative to child ADHD diagnosis (APA, 2013). In contrast, this finding was consistent with longitudinal studies

following children with ADHD into adulthood and finding no differences in actigraph-measured activity level between those who did and did not continue to meet ADHD diagnostic criteria despite clear separation in informant reports of hyperactive symptoms (Cheung et al., 2015; Halperin et al., 2008). This pattern of findings—that adults with ADHD are perceived as less ‘hyperactive’ than children with ADHD despite objective evidence to the contrary—suggests that the increased activity level associated with ADHD may change topographically across development, perhaps shifting into more socially acceptable or less overt forms of movement (e.g., decreased gross motor and increased fine motor movements). Alternatively, individuals with and without ADHD may show similar, curvilinear changes in activity level across development (Eaton, McKeen, & Campbell, 2001), such that the magnitude of between-group differences remains relatively constant over time despite both groups decreasing. Future studies would benefit from matching environmental contexts/task demands across child and adult samples to determine the extent to which the developmental continuity suggested herein may be attributable to differences in cognitive demands, maturation, or other modulators of activity level. Nevertheless, the clearly divergent developmental and subtype patterns identified via objective vs. subjective/informant-based methods provide strong empirical support for improving the science and technology of ADHD diagnostic methods toward instrumented tests for identifying hyperactivity.

The current findings suggest gender differences in objectively-measured activity level. This conclusion is based on the finding that larger effects were generally found in studies that included a lower percentage of female participants, and is generally consistent with the replicated findings that males are diagnosed with ADHD at higher rates than females in children (Biederman et al., 2005) and adults (Williamson & Johnston, 2015), and have higher mean hyperactivity/impulsivity symptom rating scores (Arnett et al., 2015). Interestingly, recent evidence suggests that gender differences in symptom severity may be at least partially attributable to gender differences in cognitive functioning to the extent that males with ADHD are more cognitively impaired than females with ADHD (Arnett et al., 2015; Seymour et al., 2015).

Of primary interest in the current study was the extent to which environmental factors—particularly cognitive demands and environmental stimulation—provocate or rarefy the presence of excess activity level (hyperactivity) in ADHD. Competing etiological models of ADHD diverge in their predictions regarding the underlying mechanisms and processes responsible for ADHD-related hyperactivity, such that hyperactivity is described as a ubiquitous deficit that is unrelated to environmental demands, a byproduct of attention deficits or variability, an outcome of deficits in one or more neurocognitive functions, secondary to trait underarousal, or the result of an interaction between neurocognitive deficits, chronic cortical underarousal, and environmental demands that challenge or overwhelm these underlying impairments (Table 1). In the current meta-analysis, high cognitive demands were associated with very large effects ($d = 1.14$), particularly during tasks that placed heavy demands on executive functioning in general ($d = 1.39$) and working memory in particular ($d = 1.35$). Low stimulation environments appear to exert a similar effect ($d = 1.01$); however, the significant difference in effect sizes between high ($d = 1.15$) and mixed ($d = 0.80$) cognitive demands within low stimulation environments suggests that this effect may be attributable at least partially to cognitive load. None of the reviewed

studies, however, tested executive functioning in high stimulation environments (Lawrence et al., 2002), which limits firm conclusions regarding the interactive role of environmental stimulation (Antrop et al., 2005; Zentall & Zentall, 1983) and cognitive demands (Rapport, Bolden, et al., 2009). In addition, the larger effects obtained during working memory relative to inhibition tasks must be considered preliminary due to the small number of studies reporting these data.

The large and significant difference in effect sizes between high cognitive/low stimulation environments ($d = 1.47$) and low cognitive/high stimulation environments ($d = 0.44$) based on best vs. worst case analyses (Lipsey & Wilson, 2001) runs contrary to etiological models that describe hyperactivity as ubiquitous, non-goal-directed activity, and suggests that environmental factors may be a primary modulator of hyperactive behavior in ADHD. This pattern of results is broadly consistent with the optimal stimulation view of hyperactive behavior (Zentall & Zentall, 1983), as well as previous experimental manipulations showing increased hyperactivity in low but not high stimulation environments (Antrop et al., 2005; Zentall & Zentall, 1976). The results appear broadly consistent also with functional working memory (Rapport et al., 2001; Rapport, Bolden, et al., 2009) accounts of hyperactivity as a compensatory behavior intended to facilitate cognitive performance by augmenting cortical underarousal. Interestingly, recent within-subject observations appear consistent with this view, and suggest that higher activity level may be associated with better performance on working memory and cognitive control tasks for children with ADHD (but not non-ADHD children; Hartanto et al., 2015; Sarver et al., 2015). To our knowledge, however, there have been no controlled provocation and rarefaction studies necessary to draw causal inferences regarding the role of hyperactivity in augmenting cognitive performance for children with ADHD.

Importantly, activity level in ADHD remained significantly elevated, albeit subdued, even during high stimulation/low cognitive load conditions ($d = 0.44$). This finding may be interpreted as partial support for models describing individuals with ADHD as ubiquitously more active than their peers; however, conventional interpretation of this small-to-medium magnitude effect size suggests that it may be difficult to detect without careful observation (Cohen, 1988). Stated differently, an effect size of 0.44 is associated with approximately 70% overlap between ADHD and non-ADHD samples, suggesting that only 30% of individuals with ADHD would be considered 'hyperactive' in these settings based on activity level that fell outside the non-ADHD range (Zakzanis, 2001)². In contrast, the 1.47 effect size associated with activity level during high cognitive/low stimulation contexts corresponds to approximately 30% overlap. In this case, approximately 70% of participants with ADHD obtained activity level scores that were not obtained by healthy controls. Thus, activity level appears to be significantly subdued but not eliminated under low cognitive demand conditions. The idea that 'one cannot not think' (Rapport et al., 2001) may provide a parsimonious explanation for this finding—that is, even 'low' cognitive demand conditions

²This value corresponds to 100% negative predictive power (NPP) but only 30% positive predictive power (PPP) if the diagnostic cut-off score is set at the edge of the Typically Developing range, indicating that mechanically-assessed activity level during low cognitive demand tasks is not likely to be useful diagnostically. Changing the cut-off score can increase PPP at the cost of decreased NPP (i.e., more true positives but also more false positives; Zakzanis, 2001).

likely require at least some cognitive processing. To the extent that motor activity functions to compensate for chronic cortical underarousal (Andreassi 1995; Barry et al. 2005) and/or underdeveloped neurocognitive abilities (Kasper et al., 2012) as hypothesized, we might expect activity level to vary systematically across a continuous dimension of cognitive demands (Hudec et al., 2014, 2015). Alternatively, third variable explanations cannot be ruled out due to the non-experimental nature of meta-analysis; it remains possible that studies coded as high and low cognitive demands also vary on one or more unmeasured variables that could account for the obtained results.

Limitations

The current meta-analysis was the first to test model-driven predictions regarding the ubiquity and underlying mechanisms of hyperactivity in ADHD, and systematically examine methodological, demographic, and environmental factors associated with mechanically assessed hyperactivity in ADHD. Several caveats require consideration when interpreting the present findings despite these and other methodological refinements (e.g., publication bias and sampling error correction, random effect models, best case estimation). We were unable to directly compare ADHD subtypes/presentations because the majority of studies included mixed groups and collapsed activity data across subtypes. However, effect sizes did not vary systematically based on the proportion of ADHD participants with the Inattentive subtype, and the few studies that have examined this directly have consistently documented minimal differences in mechanically measured activity level between Inattentive and Combined presentations of the disorder (Bauermeister et al., 2005; Dane et al., 2000; Hartanto et al., 2015; Miyahara et al., 2014), or between ADHD-persisters and ADHD-remitters defined in part by hyperactivity symptom ratings (Cheung et al., 2015; Halperin et al., 2008). In addition, significant publication bias was detected, suggesting that additional studies failing to find elevated activity level in ADHD may have been conducted but not published. Although we were able to statistically correct for this trend when computing overall effect sizes, missing studies by necessity were not included in moderator analyses, where they may have impacted the significance and magnitude of examined moderators. Finally, a limited number of studies were available to examine the extent to which the relation between cognitive demands and hyperactive behavior in ADHD was attributable to specific cognitive functions.

Summary and clinical implications

Collectively, the present meta-analysis of over 1,800 children, adolescents, and adults with ADHD confirmed that individuals with ADHD are objectively more hyperactive than their unaffected peers, and identified specific environmental, methodological, and demographic factors that systematically influenced the extent to which this hyperactivity is detectable across studies. In particular, substantially elevated activity level was apparent in studies that employed actigraphy to measure movement intensity and/or frequency during working memory and related tasks with high executive functioning demands completed in relatively low stimulation environments ($d = 1.47$). In contrast, detecting ADHD-related hyperactivity was considerably more challenging when cognitive demands were relatively low, external stimulation was high, and the assessment occurred in school or community settings ($d = 0.44$). These results are consistent with ADHD etiological models that describe hyperactivity

as situation-dependent, as well as models conceptualizing hyperactivity as compensatory behavior and highlighting the interaction between neurobiological vulnerabilities and environmental demands.

For clinical practice, these results suggest that we should consider carefully the settings in which informants are observing children suspected of ADHD. In particular, the current findings suggest that most individuals with ADHD may not appear ‘hyperactive’ in environments with high stimulation/low cognitive demands; thus, a documented lack of hyperactivity in these settings may not rule out ADHD or inform subtype/current presentation. Notably, however, the unexpectedly modest effect sizes for school-based studies ($d = 0.55$) may be due in part to insufficient sampling durations. That is, a majority of school-based studies were limited to a few hours or single day of behavioral sampling; given the high within- and between-day variability in ADHD children’s classroom behavior (Kofler et al., 2008; Rapport, Kofler et al., 2009), hyperactivity differences are likely to become increasingly large when presented as a cumulative record over a longer period (e.g., 2 weeks; Licht & Tryon, 2009). These cost efficient, minimally intrusive, objective, and ecological valid records may become clinically useful for diagnosing hyperactivity, monitoring treatment effects, and advancing a ‘precision medicine’ agenda—particularly if clinicians collect locally representative, normative data.

In addition, the current findings raise questions about commonly recommended classroom accommodations for reduced distraction / low stimulation settings—that is, the current results suggest that this approach, while face valid, may exacerbate rather than control hyperactive behaviors for students with ADHD (Zentall & Zentall, 1976). Finally, we speculate that the current findings may suggest problems for animal models of ADHD—that is, to the extent that animal models should mimic the symptomatology of ADHD (Sagvolden et al., 2005), the applicability of animal models based on selective breeding for spontaneous hyperactivity warrants scrutiny if indeed hyperactivity in ADHD is an outcome of environmental stimulation/cognitive demands rather than ubiquitous behavior (Rapport, Bolden, et al., 2009; Zentall & Zentall, 1983).

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Highlights

- Excess motor activity in ADHD depends to considerable extent on environmental demands
- High executive function demands and low stimulation environments predict largest activity effect sizes
- Diagnostic method, movement technology and topography, and gender also moderated activity magnitude
- Children & adults with ADHD exhibit similar, high levels of excess motor activity
- ADHD subtypes/presentations exhibit similar, high levels of excess motor activity

Table 1

Description of attention-deficit/hyperactivity disorder (ADHD) etiological models with predictions regarding hyperactivity

Model	Model description of ADHD	Model account of hyperactivity	Representative publications
Behavioral Inhibition	A core deficit model wherein deficits in BI (stopping pre- potent/ongoing responses and interference control) result in four areas of executive dysfunction that collectively result in ADHD behavioral symptoms	Ubiquitous, non-goal directed behavior attributable to the direct effects of BI dysfunction and indirect effects of BI deficits through executive dysfunction resulting in sustained attention/vigilance deficits	Barkley (1997, 2001)
Cognitive Neuroenergetic	Decreased ATP production and inadequate lactate supply from deficient astrocyte functioning causes the behavioral features of inefficient and inconsistent performance in individuals with ADHD.	Attentional lapse model; Model does not predict hyperactivity per authors; post hoc predictions consistent with Working Memory Model	Killeen et al. (2013); Russell et al. (2006); Sergeant (2005)
Default Mode Network	A multiple pathway model that hypothesizes that disruptions in cortico-striato-thalamo-cortical neuroanatomical circuitry--consisting of 'hot' and 'cool' regions--contribute to functional behavioral and cognitive differences in ADHD	Attention lapse model; Predictable oscillations in default mode (resting state) neural networks interfere with task-oriented neural processing, producing periodic lapses of attention.	Castellanos et al., (2005); Castellanos & Tannock (2002); Sonuga-Barke & Castellanos (2007)
DSM-5 Clinical Model	Core hyperactivity deficit in ADHD	Hyperactivity as ubiquitous, non-goal directed behavior	DSM-5 (APA, 2014)
Dynamic Developmental	A core deficit model that hypothesizes that reduced dopaminergic functioning causes narrower reinforcement gradients and altered extinction processes in normal behavior-consequence relationships. These deficient dual processes contribute to core ADHD symptoms and behavioral variability, which vary based on context, task, and function	Behavioral manifestation of pattern of inconsistent behavior-response associations affected by deficient reinforcement/ extinction mechanisms, which in turn, disrupt the accumulation of simple behavioral response units into more complex and functional response chains	Sagvolden et al. (2005)
Functional Working Memory	A core deficit model that views ADHD symptoms as phenotypic/behavioral expressions of interaction between neurobiological vulnerability & environmental demands that overwhelm impaired working memory. Associated features of ADHD arise through direct effects of impaired WM, or indirect effects of impaired WM through its impact on core behavioral symptoms	Attributable to the direct effects of CE dysfunction and chronic cortical underarousal. Increased motor activity reflects compensatory behavior to either (a) augment cortical arousal during cognitively challenging activities, or (b) escape/avoid tasks that overwhelm these children's underdeveloped working memory systems.	Rappoport et al. (2001, 2009); Sarver et al. (2015)
Optimal Stimulation	Hyperactive children are chronically underarousaed due to inadequate neurotransmission and/or a shift in the level of stimulation these children find to be optimal. A feedback model based on the assumption that response output functions homeostatically to regulate the level of stimulus input.	Increased activity in low stimulation environments functions to increase visual and kinesthetic input to compensate for underarousal; this increased activity may take the form of locomotor activity, looking around, and increased verbalizations	Zentall & Zentall (1983)
Subcortical Deficit	A developmental model that hypothesizes that ADHD is caused by subcortical neural dysfunction that manifests early in ontogeny, remains relatively static throughout life, and is not associated with the remission of symptomatology. Executive dysfunction	Reflects unconsciously (i.e., non-prefrontally) mediated deficits in arousal and activation similar to those described by the Cognitive Energetic Model	Halperin & Schulz (2006); Halperin et al. (2008); Trampush et al. (2014)

Model	Model description of ADHD	Model account of hyperactivity	Representative publications
	does not cause ADHD symptoms, but developmental growth in executive functions facilitates recovery.		
Tripartite Pathway	A multiple pathway/equifinality model in which ADHD symptoms are caused by deficits in one or more dissociable cognitive (behavioral inhibition, temporal processing) and/or motivational (delay aversion) processes	Heterogeneity model; ADHD symptoms including hyperactivity attributable to inhibition, delay, and/or temporal processing deficits, each affecting some ADHD patients	Sonuga-Barke (2010)
Variability Trait	Childhood Hyperactivity attributed to excessive variability, both in rate and magnitude of change, in arousal level and reactivity; excessively inconsistent arousal and reactivity result in problems in sustained attention, performance, and social behavior	Excessive variability in autonomic, electrocortical and behavioral response underlies impairments in attention, performance, and social behavior	Hicks et al. (1989)

Note. BI = behavioral inhibition; CE = central executive; WM = working memory

Table 2

Methodological and demographic moderators of activity level in ADHD

	<i>k</i>	Cohen's <i>d</i>	95% CI	Contrasts
Overall Cohen's <i>d</i> effect size	78	0.86	0.73 to 0.99	
Moderated Cohen's <i>d</i> effect sizes: Methodological & Demographic Factors				
Age Group				Child = Adult = Pre = Adol; Pre/Adol > Ch
Preschool	6	1.26	0.48 to 2.03	
Child	53	0.46	0.33 to 0.60	
Adolescent	7	1.11	0.43 to 1.78	
Adult	13	0.61	0.42 to 0.80	
Technology				Actigraph = Infrared
Actigraph/actometer	61	0.89	0.74 to 1.04	
Infrared motion	12	0.70	0.50 to 0.91	
Other	5	0.23	-0.47 to 0.93, <i>ns</i>	
Movement Type				Freq = Dist > Int = Dur
Frequency	57	0.82	0.66 to 0.98	
Duration	7	0.48	0.28 to 0.68	
Intensity	15	0.49	0.33 to 0.64	
Distance/area	9	0.71	0.53 to 0.88	
Body Placement				Ank = Tr = Wr = Hd > Mult
Ankle(s)	20	0.87	0.62 to 1.12	
Wrist	36	0.96	0.75 to 1.16	
Trunk/shoulders	16	1.05	0.64 to 1.45	
Head	12	0.72	0.49 to 0.94	
Multiple	5	0.39	0.08 to 0.69	
Setting				Lab > School = Comm.
Laboratory/clinic	53	0.92	0.76 to 1.07	
School and/or Community	25	0.27	0.07 to 0.46	
School	13	0.55	0.36 to 0.73	
Community	10	0.34	-0.01 to 0.67, <i>ns</i>	
Diagnostic Method				Multiple = Single
Single Informant	35	0.75	0.54 to 0.95	
Multiple Informants	42	0.94	0.77 to 1.11	
Percent Female				Low Pct > High Pct
< 25% Female	43	0.87	0.69 to 1.05	
25% Female	29	0.57	0.39 to 0.74	

Note. All Cohen's *d* effect sizes are corrected for sample size using the Hedges' *g* formula due to the upward bias of small *N* studies. Effect sizes are considered significantly different from 0.0 (statistically significant at $p < .05$) if their 95% confidence interval does not include 0.0. All effect sizes are corrected for sampling error and publication bias. Positive values indicate increased activity level for ADHD groups relative to typically developing groups.

k = number of included studies; *ns* = non-significant (95% confidence interval includes 0.0; $p > .05$).

Table 3

Cognitive demands and environmental stimulation as moderators of activity level in ADHD

	<i>k</i>	Cohen's <i>d</i>	95% CI	Contrasts
Overall Cohen's <i>d</i> effect size	78	0.86	0.73 to 0.99	
Moderated Cohen's <i>d</i> effect sizes: Cognitive Demands				
Cognitive Demands				High > Mixed > Low = School
High	31	1.14	0.93 to 1.35	
Mixed	25	0.70	0.50 to 0.90	
Low	29	0.36	0.18 to 0.54	
In-Class Schoolwork	15	0.50	0.34 to 0.67	
High Cognitive Demands				EF > Non-EF WM > Inhibition
Executive Functions	12	1.39	0.80 to 1.97	
Working Memory	3	1.35	0.56 to 2.15	
Inhibition	5	0.49	0.20 to 0.78	
Set Shifting	0	--	--	
Non-Executive Functions	22	0.71	0.54 to 0.88	
CPT, Choice RT	19	0.69	0.52 to 0.86	
IQ	3	0.48	0.10 to 0.86	
Moderated Cohen's <i>d</i> effect sizes: Environmental Stimulation				
Environmental Stimulation				Low > High
High	27	0.54	0.40 to 0.67	
Low	45	1.01	0.83 to 1.19	
Unknown/Mixed	18	0.40	0.13 to 0.68	
Cognitive Demand x Environmental Stimulation: Interaction Analysis				
Low Envir. Stimulation (LES) and				LES+HC > LES+MC
High Cognitive	30	1.15	0.94 to 1.36	
Mixed Cognitive	17	0.80	0.54 to 1.05	
Low Cognitive	0	--	--	

Note. All Cohen's *d* effect sizes are corrected for sample size using the Hedges' *g* formula due to the upward bias of small *N* studies. Effect sizes are considered significantly different from 0.0 (statistically significant at $p < .05$) if their 95% confidence interval does not include 0.0. All effect sizes are corrected for sampling error and publication bias. Positive values indicate increased activity level for ADHD groups relative to typically developing groups.

k = number of included studies; *ns* = non-significant (95% confidence interval includes 0.0; $p > .05$).

Table 4

Methodological and demographic moderators of heterogeneity among high cognitive + low stimulation conditions

	<i>k</i>	Cohen's <i>d</i>	95% CI	Contrasts
Overall Cohen's <i>d</i> effect size	30	1.15	0.94 to 1.36	
Moderated Cohen's <i>d</i> effect sizes: Methodological & Demographic Factors				
Age Group				Child/Adolescent = Adult
Child/Adolescent	20	0.82	0.60 to 1.03	
Adult	9	0.72	0.53 to 0.92	
Technology				Actigraph > Infrared
Actigraph/actometer	17	1.30	0.93 to 1.66	
Infrared motion	12	0.70	0.50 to 0.91	
Movement Type				Frequency = Intensity > Duration = Distance
Frequency	18	1.23	0.91 to 1.52	
Intensity	7	0.97	0.64 to 1.30	
Duration	5	0.50	0.27 to 0.73	
Distance/area	9	0.71	0.53 to 0.88	
Body Placement				Ankle = Mult > Head; Wrist, Trunk = All
Ankle(s)	12	1.12	0.73 to 1.52	
Wrist	6	1.34	0.60 to 2.08	
Trunk/shoulders	3	1.84	0.29 to 3.38	
Head	12	0.72	0.49 to 0.94	
Multiple	4	1.42	0.81 to 2.04	
Setting				--
Laboratory/clinic	30	1.15	0.94 to 1.36	
School/Community	0	--	--	
Diagnostic Method				Multiple > Single
Single Informant	12	0.68	0.49 to 0.87	
Multiple Informants	19	1.31	0.98 to 1.63	
Percent Female				Lower Percent > Higher Percent
< 25% Female	17	1.23	0.89 to 1.57	
25% Female	11	0.73	0.54 to 0.92	

Note. All Cohen's *d* effect sizes are corrected for sample size using the Hedges' *g* formula due to the upward bias of small *N* studies. Effect sizes are considered significantly different from 0.0 (statistically significant at $p < .05$) if their 95% confidence interval does not include 0.0. All effect sizes are corrected for sampling error and publication bias. Positive values indicate increased activity level for ADHD groups relative to typically developing groups.

k = number of included studies; *ns* = non-significant (95% confidence interval includes 0.0; $p > .05$).

Table 5

Histogram of Cohen’s *d* Effect Sizes

Stem	Effect Size										
3	3.45										
...											
2	2.28	<u>2.74</u>									
1.9											
1.8											
1.7	1.70	1.71									
1.6											
1.5	<u>1.57</u>										
1.4	1.47	<u>1.48</u>	<u>1.49</u>								
1.3	1.30	1.34									
1.2	1.21										
1.1	<i>1.11</i>	1.11	1.12	1.18							
1	1.06	<u>1.07</u>	<i>1.07</i>								
0.9	<i>0.90</i>	<u>0.92</u>	<u>0.93</u>	<i>0.95</i>	0.95	0.97	0.98				
0.8	<i>0.81</i>	0.81	0.83	0.84	<i>0.84</i>	0.87	0.87	<i>0.87</i>	<u>0.89</u>		
0.7	<u>0.70</u>	0.70	0.71	<u>0.73</u>	0.74	<u>0.75</u>	<u>0.75</u>	0.76	0.76	<i>0.77</i>	
0.6	0.62	<i>0.62</i>	0.62	0.63	0.65	<u>0.67</u>	0.67				
0.5	<u>0.50</u>	0.54	<u>0.54</u>	<i>0.55</i>	0.55	<i>0.56</i>	0.59	0.59			
0.4	0.44	0.45	0.45	0.46	0.46	<i>0.47</i>	0.48	0.49			
0.3	<i>0.30</i>	<u>0.30</u>	<i>0.31</i>	<u>0.32</u>	<i>0.32</i>	<u>0.34</u>	<u>0.36</u>	0.36			
0.2	<i>0.23</i>	<i>0.24</i>	<u>0.25</u>	<i>0.26</i>	<u>0.27</u>	<i>0.28</i>	<u>0.29</u>				
0.1	<u>0.11</u>	<i>0.17</i>	<i>0.18</i>	0.18	<i>0.19</i>						
0	0.00	<i>0.00</i>	<i>0.00</i>	0.04	<u>0.06</u>						
-0	<i>-0.06</i>	<i>-0.07</i>									
-0.1	<i>-0.16</i>	<i>-0.17</i>									
-0.2											
-0.3	<u>-0.37</u>										
-0.4											
-0.5	<i>-0.59</i>										
-0.6	<i>-0.65</i>										

Note. Histogram of effect sizes across studies. The left column reflects the ones and tenths digits, and the right columns reflect each obtained effect size. Each entry indicates one unique effect size. **Bold** font represents effect sizes for activity level during High Cognitive Demand conditions, *italicized* font reflects Low Cognitive Demand conditions, underlined font indicates Mixed Cognitive Demand conditions, and regular font notates In-Class Schoolwork conditions. Positive values indicate increased activity level for ADHD groups relative to typically developing groups.

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Table 6

Study characteristics.

Study	ADHD N	TD N	SUB- GRP	TASK(S)	TECH- NOLOGY	MOVEMENT TYPE	BODY PLACE.	SETTING	AGE	% MALE	% ADHD-I
Abaya (1997)	14	17	ADHD-C	5 Days	Actometer	Frequency	Trunk	Community	Child	1.0	0
Alderson et al. (2012)	8	17	ADHD-I	5 Days	Actometer	Frequency	Trunk	Community	Child	1.0	1.0
	12	11		Stop Signal Choice RT(2) Painting(2)	Actigraph	Intensity	Wrist + Ankle	Lab	Child	1.0	0
Anderson (2011)	62	61	Child	CPT	Infrared Motion	Multiple	Head	Lab	Child	.79	.40
	19	30	Adult	CPT	Infrared Motion	Multiple	Head	Lab	Adult	.63	.22
Baerg et al. (2011)	16	30	Male	7 Days	Actigraph	Frequency	Waist	Community/School	Adol	1.0	NR
	14	18	Female	7 Days	Actigraph	Frequency	Waist	Community/School	Adol	0	NR
Baird et al. (2012)	13	19		Daily Activity	Actigraph	Frequency	Wrist	Community	Adult	.63	NR
Barkley & Cunningham (1979)	14	14		Playroom	Actometer	Frequency	Wrist + Ankle	Playroom	Child	1.0	0
Barkley & Ullman (1975)	16	16		Free Play Go/No-Go	Actometer Pedometer	Frequency	Wrist	Lab	Child	1.0	0
Barkley et al. (1990)	42	34	ADD+H	CPT Math P-C Int.	Actometer	Frequency	Wrist Ankle	Lab	Child	.93	Nr
	48	34	ADD-H	CPT Math P-C Int.	Actometer	Frequency	Wrist Ankle	Lab	Child	.90	Nr
Bauermeister et al. (2005)	44	29		CPT Math Task	Actigraph	Frequency	Trunk	Lab	Child	.59	.36
Bijlenga et al. (2013)	12	12		5 Days	Actigraph	NR	Wrist	Community	Adult	.50	NR
Boonstra et al. (2007)	33	39		Daily Activity	Actigraph	Frequency	Wrist	Community	Adult	.53	0
Casaseca De La Higuera et al. (2012)	31	32		Daily Activity	Actigraph	Frequency	Wrist	Community	Child	NR	0
Cunningham & Barkley (1979)	20	20		Mother-Child Interaction	Actometer	Frequency	Wrist Ankle	Lab	Child	1.0	NR
Dane et al. (2000)	22	22	ADHD-C	Psychoed. Eval.	Actigraph	Frequency	Wrist	Lab	Child	.77	0
Edebol et al. (2013)	25	114	Men	CPT	Infrared Motion	Distance	Head	Lab	Adult	1.0	NR
	30	88	Women	CPT	Infrared Motion	Distance	Head	Lab	Adult	0	NR
Firestone (1977)	11	15		RT	Stabilometric Chair	Frequency	Chair	Lab	Child	NR	0
Glass et al. (2014)	16	22		Neuropsych. Battery	Actigraph	Intensity	Wrist	Lab	Child	NR	NR

Study	ADHD N	TD N	SUB- GRP	TASK(S)	TECH- NOLOGY	MOVEMENT TYPE	BODY PLACE.	SETTING	AGE	% MALE	% ADHD-I
Gunther et al. (2012)	18	20	ADHD1	CPT	Infrared Motion	Distance	Head	Lab	Child	.72	Nr
	18	20	ADHD2	CPT	Infrared Motion	Distance	Head	Lab	Child	.89	Nr
Halperin et al. (1992)	31	25		Neuropsych. Battery	Actigraph	Frequency	Trunk	Lab	Child	.77	NR
Halperin et al. (2008)	98	85		Neuropsych. Battery	Actigraph	Frequency	Ankle Trunk	Lab	Adult	.88	1.0
Hartanto et al. (2015)	26	18		Flanker	Actigraph	Intensity	Ankle	Lab	Child/Adol	.46	NR
Ho et al. (1996)	59	73	ADHD1	CPT IQ	Actometer	Frequency	Ankle	Lab	Child	1.0	0
	19	73	ADHD2	CPT IQ	Actometer	Frequency	Ankle	Lab	Child	1.0	0
	17	73	ADHD3	CPT IQ	Actometer	Frequency	Ankle	Lab	Child	1.0	0
Hudec et al. (2014)	20	15		WM(2) Painting(2)	Actigraph	Intensity	Wrist + Ankles	Lab	Adult	.50	NR
Hudec et al. (2015)	19	18		N-Back(2) RT(2) Painting(2)	Actigraph	Intensity	Ankles	Lab	Child	1.0	.10
Kam et al. (2010)	10	132		Playtime Class	Actigraph	Intensity	Wrist	School	Child	.50	NR
Kam et al. (2011)	10	7		Class(3)	Actigraph	Intensity	Wrist	School	Child	.80	NR
Kercood et al. (2004)	29	23		Math(3)	Actometer	Frequency	Wrist	Lab	Child	.69	NR
Konrad et al. (2000)	31	26		Stop Signal Delay Task	Actigraph	Intensity	Wrist	Lab	Child	.90	.29
Lee & Zentall (2006)	12	17		CPT(2)	Actometer	Frequency	Ankle	Lab	Adult	.27	NR
Licht & Tryon (2009)	9	9		12 Days School Home	Actigraph	Intensity	Trunk	School Community	Child	.89	0
Lin et al. (2013)	20	20		7 Days School Home	Actigraph	Frequency	Trunk	Community/School	Child	1.0	NR
Lis et al. (2010)	20	20		N-Back	Infrared Motion	Frequency	Head	Lab	Adult	.65	.35
Madan-Swain (1990)	25	25		Dyad Play	Actometer	Frequency	Wrist	Lab	Child	.64	NR
Marks et al. (2005)	22	50		Flanker(2) STM(2)	Actigraph	Frequency	Ankle Trunk	Lab	Pre-School	.81	NR
Martin-Martinez et al. (2011)	31	32		Morning Afternoon	Actigraph	Intensity	Wrist	Community	Child	NR	0
Matier-Sharma (1995)	40	18		CPT Psychoed. Battery	Actigraph	Frequency	Trunk	Lab	Child	.74	NR
Mcgrath et al. (2004)	18	22	ADHD1	School Days(3)	Actigraph	Intensity	Wrist	School	Adol	.73	.28

Study	ADHD N	TD N	SUB- GRP	TASK(S)	TECH- NOLOGY	MOVEMENT TYPE	BODY PLACE.	SETTING	AGE	% MALE	% ADHD-I
Meyer et al. (1995)	17	22	ADHD2	School Days(3)	Actigraph	Intensity	Wrist	School	Adol	.73	.29
Miyahara et al. (2014)	39	76	ADHD-C	Solitary & Dyad Play Neuropsych. Battery	Actometer Actigraph	Frequency Frequency	Wrist Ankle Trunk	Lab Lab	Child Pre-School	1.0 .71	NR .10
Murillo et al. (2015)	62	76	ADHD-I ADHD-H Child	Neuropsych. Battery Neuropsych. Battery CPT	Actigraph Actigraph Infrared Motion	Frequency Frequency Duration Frequency Distance Area	Ankle Trunk Ankle Trunk Head	Lab Lab Lab	Pre-School Pre-School Child/Adol	.71 .71 .79	.10 .10 .40
Nevin (2002)	23	26	Adult	Neuropsych. Battery	Actigraph	Frequency	Head	Lab	Adult	.63	.22
Ohashi et al. (2010)	62	62		CPT	Infrared Motion	Multiple	Head	Lab	Child	1.0	0
Okada et al. (2014)	15	18		Classwork Recess	Actigraph	Frequency	Wrist	School	Child	1.0	0
Pinto & Tryon (1996)	22	31		2 Weeks Classwork Recess Home	Pedometer	Frequency	Wrist	School Community	Child	1.0	0
Plomin & Foch (1981)	18	216		1.0 Week	Pedometer	Frequency	Trunk	NR	Child	.67	0
Pollock (2014)	15	17		CPT Paint(2)	Actigraph	Intensity	Wrist + Ankles	Lab	Adult	.67	NR
Porrino et al. (1983)	12	12		Classwork Recess PE Home	Actometer	Frequency	Wrist	School Community	Child	1.0	0
Rapport et al. (1980)	15	14		Neuropsych. Battery	Actometer	Frequency	Trunk	Lab	Child	1.0	0
Rapport et al. (2009)	12	11		WM(2) Paint(2)	Actigraph	Intensity	Wrist + Ankles	Lab	Child	1.0	0
Reh et al. (2014)	45	45		CPT	Infrared Motion	Duration Frequency Distance Area	Head	Lab	Child	.78	.09
Saxon et al. (1977)	5	5	ADHD1	Playroom(4)	Ultrasonic	Duration		Lab	Pre/Child	.50	0
Shoryer & Zentall (1986)	12	15	ADHD2	Playroom(4) Story Listening(4)	Ultrasonic Actometer	Duration Frequency	Wrist	Lab School	Pre/Child Child	.50 .96	0 NR
Söderström et al. (2014)	41	20		CPT	Infrared Motion	Composite	Head	Lab	Adult	.44	.27

Study	ADHD N	TD N	SUB- GRP	TASK(S)	TECH- NOLOGY	MOVEMENT TYPE	BODY PLACE.	SETTING	AGE	% MALE	% ADHD-I
Teicher et al. (1996)	18	11		CPT	Infrared Motion	Duration Frequency Distance Area	Head Shoulder Elbow	Lab	Child	1.0	NR
Teicher et al. (2012)	40	60		Go/No-Go	Infrared Motion	Duration Frequency Distance Area	Head Ankle	Lab	Adult	.58	NR
Tripp & Luk (1997)	23	17	ADHD1	CPT	Actometer	Frequency	Wrist Ankle	Lab	Child	.83	0
	19	17	ADHD2	CPT	Actometer	Frequency	Wrist Ankle	Lab	Child	.90	0
	15	17	ADHD3	CPT	Actometer	Frequency	Wrist Ankle	Lab	Child	.67	0
Tsujii et al. (2007)	16	20		Classwork PE/Recess	Actigraph	Frequency	Wrist	School	Child	.81	0
Wood et al. (2009)	116	218		Psychoed. Battery	Actigraph	Intensity Frequency	Trunk + Leg	Lab	Child	.90	0
Zahn et al. (1980)	14	13		Multiple	Actometer	Frequency	Trunk	Lab	Child	1.0	0
Zentall & Dwyer (1989)	12	12		Visual Match(2)	Actometer	Frequency	Wrist	Lab	Child	NR	0
Zentall & Leib (1976)	15	16		Drawing(2)	Actometer	Frequency	Wrist	Lab	Child		0
Zentall & Shaw (1980)	24	24		Math(2)	Actometer	Frequency	Wrist	School	Child	.88	0
Zentall (1985)	35	35		Visual Search	Actometer	Frequency	Wrist	Lab	Child	.54	NR
Zentall et al. (1978)	4	4		Spelling	Actometer	Frequency	Wrist	School	Child	NR	0
Zentall (1989)	20	26		Spelling(2)	Actometer	Frequency	Wrist	School	Child	1.0	NR