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Reconsidering "Inattention" in Attention-Deficit Hyperactivity Disorder: Implications for Neuropsychological Assessment and Intervention

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Attention-deficit hyperactivity disorder (ADHD) does not exist. This explicit statement needs elucidation of course given ADHD is a common neurodevelopmental disorder, but it provides the reader with the impetus to reconsider long-held beliefs about this condition and its treatment. Surely, there is a disorder called ADHD from which this thesis is framed, but primary attention and hyperactivity-impulsivity problems are mediated by different albeit interrelated brain systems. Like many neurodevelopmental disorders (e.g., learning disabilities, autism spectrum disorder), the medical and psychological professions have used a single, large inclusive ADHD diagnostic category to represent children with different etiologies for their overt symptoms. Despite neurobiological differences among children diagnosed with ADHD, the clinical position that attention-deficit or primary attention problems are sufficient for ADHD identification undermines clinical practice. This commonly accepted dubious position not only

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undermines the diagnostic utility of our neuropsychological measures, but it attenuates treatment effects as well. Supported with evidence from our ongoing ADHD research program, this data-based review will support these contentions and provide implications for diagnosis and treatment of children with attention problems.

Key words: ADHD, differential diagnosis, frontal-subcortical circuits, treatment efficacy

ADHD: DOES IT EXIST?

Variable or inconsistent attention is a fact of life. As you read this treatise, your attention will either be indirectly or directly challenged. The response to distraction is under executive control for most individuals, but for some, sustained attention or vigilance is negatively affected by subcortical and/or cortical dysfunction that undermines volition. With so many causes of attention problems, one must ask whether a child has attention-deficit hyperactivity disorder (ADHD) or another disorder affecting attention. Unfortunately, this question is seldom asked in standard clinical practice. Overt behavioral criteria cannot help clinicians decipher the nature of the attention problem, yet most seasoned practitioners are comfortable concluding that a child has "ADHD" rather than spending the time or resources necessary for differential diagnosis of a child's attention problems. This diagnostic inclusiveness leads to heterogeneous ADHD samples in which overt behavioral criteria provide the clinician with the only coherent data pattern among those in the population. However, as we argue here, this longstanding and outdated approach not only ensures that our neuropsychological assessment instruments have limited specificity for differential diagnosis, but it also undermines the ecological and treatment validity of these measures (e.g., Hale, Reddy, Weissman, Lukie, & Schneider, 2013; Koziol & Budding, 2012), negatively impacting the children we are charged with serving.

Why continue a practice that undermines diagnostic accuracy and attenuates treatment effects? That is an important question addressed throughout this thesis as we attempt to deconstruct the disorder commonly referred to as ADHD. Despite the voluminous research conducted on what we call ADHD, we propose a certainly controversial position here that suggests the disorder actually does not exist from a neurobiological perspective. Instead of using a behavioral approach to ADHD diagnosis, we need new methods and approaches for understanding, diagnosing, and treating children with ADHD, with neurobiology—not behavior—serving as the conceptual anchor (e.g., Hale et al., 2013). With this caveat in mind, the extant ADHD database will be explored.

FOUNDATION FOR ADHD DECONSTRUCTION

Affecting approximately 5% to 7% of school-aged children, ADHD is a commonly diagnosed neurodevelopmental disorder with considerable neuropsychological heterogeneity (Fair, Bathula, Nikolas, & Nigg, 2012) due to its behavioral definition. Although traditionally considered a disruptive behavior disorder characterized by symptoms of inattention, hyperactivity, and impulsivity, children with ADHD often exhibit executive function (EF) impairments that manifest on neuropsychological measures and/or rating scales. EF constructs such as planning, organizing, strategizing, monitoring, shifting, and evaluating behavior, as well as working memory and response inhibition, may be impaired (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Children with ADHD also frequently present with externalizing (e.g., oppositional defiant disorder) or internalizing (e.g., anxiety) disorders and/or specific learning disabilities (Larson, Russ, Kahn, & Halfon, 2011), which leads one to consider that these disorders may be the *source* of inattention in some affected children. Therefore, the etiology of the attention problem is important for determining if the child has "true" ADHD or another disorder affecting attention—what we refer to as "pseudo" ADHD (Hale et al., 2009).

Inattention is a common feature of most neurodevelopmental and neuropsychiatric disorders. As a result, it is often difficult to determine whether children presenting with attention problems have ADHD or another disorder affecting attention. Despite evidence that ADHD is caused by dopaminergic dysregulation and hypoactivity in the frontal-subcortical circuits (FSC; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006), current diagnostic practice only requires affirmative behavioral criteria gained through subjective, summative informant reports. This practice, although commonly accepted, is in essence clinically a non sequitur. The question remains as to why this overt behavioral approach remains the mainstay of current ADHD diagnostic practice given the wealth of literature highlighting the neurobiological deficits associated with this disorder.

The explanation for this seemingly incongruent practice can be found in the medical profession guidelines and research. The American Academy of Pediatrics (2011) guidelines for ADHD diagnosis include parent-reported (e.g., *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition [DSM-5]; American Psychiatric Association, 2013) criteria, symptoms present at both home and school, and ''evaluation'' of potential coexisting conditions. Direct measurement of a child's attention or EF is seldom required or even considered for ADHD diagnoses unless there are other concerns about academic achievement. Thus, professional tradition or dogma ensures the preeminence of behavioral diagnostic criteria as the ''gold standard'' for diagnosis of this neurodevelopmental disorder. In apparent support of this position, some have argued against the use of neuropsychological tests in differential diagnosis of ADHD, and instead, they suggest that rating scales are sufficient ADHD diagnostic tools (e.g., Brown & LaRosa, 2002; Doyle, Biederman, Seidman, Weber, & Faraone, 2000). Such behavior-rating advocates use the limited-to-poor correlations between neuropsychological EF tests and ADHD behavior ratings as evidence to support their positions that behavior ratings are more useful in ADHD diagnosis (e.g., Barkley & Murphy, 2010). We have also found these limited correlations in our own carefully diagnosed children with ADHD (physician and independent psychologist verification using informant report and rating scale criteria) who have been referred for double-blind placebo-controlled trials of methylphenidate (MPH; e.g., Hale et al., 2011; Kubas, Backenson, Wilcox, Piercy, & Hale, 2012; see Table 1).

If DSM-5 criteria are used for ADHD diagnosis and the correlations between the neuropsychological measures and behavioral criteria are poor, why bother with costly and time-consuming neuropsychological testing for ADHD? The problem herein is a fundamental error in how previous ADHD research has been conducted—an error we argue negates many of, if not all, the ADHD empirical efforts to date. If the disorder is defined on the basis of informant (parent) report and then subsequently validated with rating scales from the same informant, there is an inherent circularity in the research design (e.g., Hale et al., 2009).

To summarize, attention problems are ubiquitous in clinical practice and are characteristic of most neurodevelopmental and neuropsychiatric disorders. Attention is not typically measured directly in ADHD diagnostic practice, and instead, the use of indirect, summative, informant reports of attention problems is all that is needed according to professional standards. However, previous research suggesting that behavioral reports are better for ADHD diagnosis than neuropsychological testing is based on circular research designs, which negate the conclusions drawn from these studies.

Neuropsychological measures may be more useful in distinguishing between ADHD and other disorders impacting attention, as well as when monitoring treatment response, but to realize this possibility, we must move away from strict behavioral diagnostic methods and incorporate neuropsychological measures in practice to help differentiate ADHD from other disorders of attention. After all, ADHD is a neurodevelopmental disorder, not a behavioral one.

TABLE 1
Zero-Order Correlations Between Neuropsychological Measures and DSM-IV-TR Criteria

	DSM-IV-TR Criteria		
Neuropsychological Measures	*Inattentive r (r^2)*	*Hyperactive-Impulsive r (r^2)*	*Total Symptoms r (r^2)*
SCWT Raw	−.17 (.030)	−.31 (.096)*	−.37 (.138)*
SCWT Errors	.01 (.000)	.15 (.022)	.13 (.018)
TMTB Time	.33 (.106)*	.19 (.036)	.35 (.125)*
TMTB Errors	.41 (.170)*	.31 (.096)*	.51 (.258)*
Digits Backward	.18 (.031)	−.28 (.076)*	−.15 (.021)
SRTM Consistent Retrieval	.03 (.001)	−.32 (.104)*	−.27 (.072)*
HDCT Correct	−.15 (.021)	−.08 (.006)	−.15 (.023)
Go–No Go	−.08 (.006)	−.22 (.048)*	−.24 (.058)*
CPT-II Omissions	.17 (.030)	.13 (.016)	.21 (.044)*
CPT-II Commissions	.13 (.018)	−.06 (.004)	.02 (.000)
CPT-II Block Change	.19 (.035)	.20 (.038)*	.28 (.078)*

DSM-IV-TR = *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision; SCWT = Stroop Color-Word Test; TMTB = Trail-Making Test-Part B; SRTM = Selective Reminding Test of Memory; HDCT = Hale-Denckla Cancellation Test; CPT-II = Conners' Continuous Performance Test-Second Edition.

*$p < .05$.

MOVING TOWARD A NEUROPSYCHOLOGICAL APPROACH TO UNDERSTANDING ADHD

If behavioral models lead to neuropsychological heterogeneity in ADHD, including those with and without EF dysfunction, perhaps redefining ADHD as a neurobiological disorder of executive control of attention makes more sense. The movement away from behavioral diagnosis alone when determining mental disorders is now under way, according to the U.S. National Institute of Mental Health (NIMH; Insel, 2013). Recognizing the arguments presented herein, one purpose of the NIMH *Research Domain Criteria* (RDoC) is to transform diagnosis away from behavioral categorical approaches to multimethod approaches that combine neurobiological and genetic markers of disorders that can enhance diagnostic and treatment practices. The NIMH RDoC approach is based on several assumptions:

1. A diagnostic approach based on the neurobiology of symptoms is needed and cannot be constrained by DSM-5 categories;
2. mental disorders are brain disorders that affect cognition, emotion and/or behavior;
3. levels of analyses should be considered using a dimensional approach; and
4. determining the neurobiological, genetic, and cognitive markers of mental disorders will likely lead to more targeted and efficacious treatment outcomes (Insel, 2013).

A neuropsychological approach to ADHD assessment and treatment is premised on the fact that structural and functional differences in the FSC are common in children with ADHD compared to typical children and those with other neuropsychiatric conditions. This research suggests poor attention *control* in ADHD is the result of catecholamine dysregulation that leads to FSC hypoactivity (Arnsten & Pliszka, 2011). As Denckla (1996) lucidly argued some time ago, primary attention is *not* impaired in ADHD; instead, it is the child's *intention* to regulate attention—a function of FSCs—that is impaired. This explains why children with ADHD can focus attention quite well when EF is "online" and in sync with environmental demands, such as during fast-paced, exciting activities (e.g., video games, "thrill" sports). Specifically, the dorsal (e.g., dorsolateral-dorsal cingulate) and ventral-medial (e.g., orbital-nucleus accumbens-ventral cingulate) cortices and associated FSC structures (e.g., basal ganglia, thalamus) are often implicated in children with ADHD (Semrud-Clikeman, Walkowiak, Wilkinson, & Butcher, 2010).

It is not enough to say that children with ADHD have FSC hypoactivity and EF deficits. Instead, we need to begin to parcel out FSCs in relation to EF if we are to separate ADHD from other disorders that affect attention. Given that FSCs, EF, attention control, and primary attention are highly related (e.g., Steinlin, 2008), the differentiation of EF and primary posterior-subcortical attention is not particularly easy, and even then, most neuropsychiatric disorders also have FSC impairment and attention problems (Hale et al., 2013). Different assessment tools that can assess these interrelated FSCs are needed so that we can begin to differentiate the FSC patterns characteristic of the neurodevelopmental and neuropsychiatric disorders. Once different FSC functions are considered, we can begin to sort out the deficits that are more related to ADHD and those of other disorders affecting attention.

There are likely both dorsal and ventral circuits involved in ADHD, with dorsal systems affecting attention and *cognitive* inhibitory control and ventral-medial systems regulating motivation, affect (Arnsten & Rubia, 2012), and *emotional* inhibitory control. First, *cognitive* aspects of EF, such as sustained attention, planning, flexibility, and executive working memory (EWM), are associated with "cool" dorsolateral-dorsal cingulate functioning (Castellanos et al., 2006). Cool EFs maintain information in working memory to facilitate adaptive responses in complex environments and are also involved in memory encoding and retrieval; thus, they likely have a large influence on academic achievement (Hale et al., 2013). Neuropsychological and neuroimaging studies have provided support for cool circuit involvement in ADHD during working memory, response inhibition, and executive attention tasks (e.g., Rubia et al., 2009).

"Hot" EFs, which refer to *affective* control mediated by the orbital and ventral-medial circuitry, are even more complex and difficult to measure. These "hot" EFs govern behavioral self-regulation (SR), emotional response inhibition, and response to reward (Castellanos et al., 2006; Rubia et al., 2009), functions for which there are few neuropsychological measures available. Neuroimaging research has shown these circuits to be more highly activated during reward-processing tasks (Kelly, Scheres, Sonuga-Barke, & Castellanos, 2007), which could account for those children with ADHD who experience difficulty with delayed gratification or delay aversion (Sonuga-Barke, 2005) and also those who experience poor social functioning due to deficits in perspective taking or theory of mind (Brüne & Brüne-Cohrs, 2006).

Recognizing the value of an RDoC perspective in our studies of ADHD and medication response, we have previously explored FSC functions in ADHD using structural equation modeling (SEM) and have recently added functional magnetic resonance imaging and diffusion tensor imaging to validate findings. Although

there are no "dorsal" or "ventral" EF neuropsychological tests, we used SEM to create regression-based factor scores for cool dorsal and hot ventral circuit functions for use in subsequent analyses. The Hale, Fiorello, and Brown (2005) SEM results using the neuropsychological measures reported in Table 1 revealed nonsignificant chi-square values and adequate goodness-of-fit indexes for the EWM and SR factors (LISREL goodness-of-fit indexes of .94 and .66; root mean square residuals of .05 and .12, respectively), indicating an adequate fit of the data, especially for the dorsal factor (Hale et al., 2005). Not surprisingly, given the interrelationship of the FSCs, the EWM and SR factors were highly correlated ($r = -.76$).

Using this FSC model, we have begun to explore its relationship with ADHD diagnoses and symptoms. To determine the level of baseline neuropsychological impairment, participants' individual scores on each of the neuropsychological measures were weighted based on factor loadings, with summative values added and subsequently converted to z scores. Participants were categorized into one of four groups based on FSC factor impairment level. Participants whose z score was +1.0 or greater from the mean were classified as having no apparent impairment. Participants whose scores were 0.0 to +0.9 were considered to have low impairment levels. Those with a score of −0.10 to −0.9 were classified as having moderate impairment. Finally, individuals with a score of −1.0 or less were considered to have high levels of impairment.

Table 2 presents findings from this research that examined the link between neuropsychological impairment and *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision (DSM-IV-TR) ratings. Notably, we found correlations between both hot (orbital-ventral cingulate) and cool (dorsolateral-dorsal cingulate) circuit impairment and hyperactive-impulsive symptoms ($r = .33$, $p < .05$; $r = .50$, $p < .01$, respectively); however, correlations were not significant for these executive circuits and inattention ratings ($r = .18$, $p = .25$; $r = .14$, $p = .39$, respectively). This suggests that neuropsychological impairment observed in children with ADHD is more associated with hyperactive-impulsive symptoms than with inattention, further supporting the notion that inattention is not the hallmark feature of this disorder (Barkley, 1997) and suggesting that ADHD is again a problem of response inhibition that interferes with executive control of attention (Hale et al., 2013; Koziol & Budding, 2012).

Two important caveats are worth mentioning here. First, the conclusion that attention problems are not as relevant for ADHD determination as response inhibition and EF deficits is in part dependent on how one defines ADHD. If one makes the determination on the basis of delay aversion (e.g., Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008) or cognitive-energetic (Sergeant, 2005) models—constructs that are not measured directly—then relationships with inattentive symptoms would be stronger and expected, because these problems appear to be more subcortical in nature (e.g., Paloyelis, Asherson, & Kuntsi, 2009). Clearly, there are differences between children diagnosed with ADHD with and without EF deficits, with delay aversion being found in those without EF deficits (Lambek et al., 2010), as may be the case with those appearing to have ADHD who have what has been termed slow or sluggish cognitive tempo (Barkley, 2006). This suggests there may be inherent value in parsing out these individuals (Sonuga-Barke et al., 2008) and recognizing them as having other disorders that mimic ADHD.

A second consideration is that response inhibition and other EF deficits may be *sensitive* for diagnosing "true" ADHD (e.g., Hale et al., 2009; Willcutt et al., 2005), but unfortunately, they are not *specific* to ADHD, as many disorders also have these deficits (Castellanos et al., 2006; Hale et al., 2013). Research needs to determine the types of attention deficits that are characteristic of the different disorders and how this relates to typical EF development. This focus of research and subsequent shift from using a behavioral approach alone in the diagnosis and treatment of ADHD will be critical for the future of these children, both for assessment and intervention purposes.

TABLE 2
Frontal-Subcortical Factors and DSM-IV-TR Behavioral Criteria

	DSM-IV-TR Criteria			
	Inattentive		*Hyperactive-Impulsive*	
Factor Scores	*R*	*p*	*R*	*p*
Dorsal Impairment	.136	.389	.502	.001
Ventral Impairment	.178	.253	.327	.032
Total Impairment	.170	.282	.448	.003

Note. Ventral factor recoded so lower factor scores = greater impairment. DSM-IV-TR = *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision.

EXAMINING MEDICATION RESPONSE IN ADHD

Given the complexity of issues presented in this article, perhaps the most critical issue to consider is treatment validity of the arguments presented. As suggested by those who advocate behavioral approaches, perhaps neuropsychological testing has no relevance for treatment of attention disorders including ADHD. However, if parsing ADHD into subtypes or endophenotypes leads to different treatment outcomes, then further empirical efforts are needed to discover the biological markers necessary to predict treatment response. Unfortunately, using behavioral criteria alone in ADHD diagnoses leads to substantial neuropsychological heterogeneity, and this could explain why stimulant medication response is not uniform across treated children with ADHD (Hale et al., 2011). Children with ADHD who have significant EF impairments tend to demonstrate stronger response to medication than do children without EF deficits (Kubas et al., 2012). It is possible that many children diagnosed with ADHD who do not respond to medication may not have the EF impairments that are affected positively by stimulants—despite the presence of clear behavioral symptoms of ADHD such as inattention.

It is important to consider how the most commonly used stimulant medication, methylphenidate (MPH), affects the brain. MPH is thought to increase dopamine (DA) availability in subcortical and prefrontal regions by inhibiting DA reuptake in the striatum (Arnsten & Pliszka, 2011). MPH can be effective in improving EFs such as planning, inhibition, attention, vigilance, and mental flexibility (e.g., Carrey, MacMaster, Gaudet, & Schmidt, 2007) by normalizing FSC dysregulation in ADHD (Rubia et al., 2009); however, MPH dose–response effects are not uniform, with low-dose stimulants preferentially affecting catecholamine functioning in the prefrontal regions most often associated with cool cognitive EFs (Schmeichel & Berridge, 2013). Because cool dorsolateral EFs are important for academic functioning (Hale et al., 2013; Koziol, Budding, & Hale, 2013), this low-dose preference in the dorsal EF system is important to consider given that the long-term academic treatment efficacy of stimulants has been limited (Molina et al., 2009).

NEUROPSYCHOLOGICAL IMPAIRMENT LEVEL IN DETERMINING MEDICATION RESPONSE

Given the weak correlations between neuropsychological impairment and informant-reported symptoms of ADHD described earlier, the question remains whether neuropsychological or behavioral markers are more important in predicting medication response and if this varies by subtype or cause of attention problem. We found that a majority of the children with FSC factor impairment had ADHD Combined type, but findings were not definitive (e.g., some with Inattentive type had FSC impairment, some with Combined type did not). As a result, behavior ratings alone cannot provide us with sufficient information regarding whether a child has FSC impairment and will respond to treatment (Hale et al., 2013).

As described earlier (Hale et al., 2005), we used neuropsychological impairment on the FSC factor scores to examine MPH response in children with behaviorally diagnosed ADHD. In addition to the neuropsychological measures in Table 1, we assessed behavioral response using teacher ratings on the Conners' Teacher Rating Scales - Revised: Long Form (CTRS-R:L), *Academic Performance Rating Scale*, and *School Situations Questionnaire-Revised*; parent ratings on the Conners' Parent Rating Scales - Revised: Long Form (CPRS-R:L) and *Home Situations Questionnaire-Revised*; and classroom observations, which included coding children using the categories from *Restricted Academic Task*.

Each MPH trial lasted approximately 4 weeks, with baseline assessment always being the 1st week, followed by a randomized order of placebo, low-MPH-dose, and high-MPH dose conditions during Weeks 2 to 4. To determine cognitive and behavioral MPH response, each variable was rank-ordered across each of the four conditions, with the best score given a rank of 1 and the poorest score given a rank of 4. Separate ranks for neuropsychological and behavioral MPH response were determined for each child using the NPstat nonparametric randomization test for ranks (May, Masson, Hunter, & Wells, 1990). For separate cognitive and behavioral analyses, for which the NPstat Friedman test was significant ($p < .05$), Wilcoxin post-hoc analyses were used to determine MPH response for each participant. If either (low- or high-dose) or both MPH conditions were significantly different than the placebo condition, participants were assigned a score of 1, while those who did not show an MPH response received a score of 0.

As depicted in Table 3, Spearman correlations of behavioral medication response were found to be unrelated to baseline DSM-IV-TR Inattentive criteria ($r = .03$, $r^2 < .000$, *ns*) and Hyperactive-Impulsive-type criteria ($r = .25$, $r^2 = .06$, *ns*). Correlations of cognitive MPH response were also found to be unrelated to baseline informant reported Inattentive symptoms ($r = .09$, $r^2 = .01$, *ns*), and while baseline Hyperactive-Impulsive-symptoms showed a modest correlation with cognitive MPH response ($r = .30$, $r^2 = .09$, $p = .050$), more than twice the variance was accounted for when measures

TABLE 3
Neuropsychological Tests and DSM-IV-TR Criteria in Determining Stimulant Response

Measure	*Cognitive Response r (r^2)*	*Behavioral Response r (r^2)*
DSM-IV-TR Inattention Ratings	.09 (.01)	.03 (.00)
DSM-IV-TR Hyperactivity-Impulsivity Ratings	.30* (.09)	.25 (.06)
"Cool" Dorsal Factor Score	.44** (.19)	.33* (.11)
"Hot" Ventral Factor Score	.45** (.20)	.31* (.10)

DSM-IV-TR = *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision.
*$p < .05$. **$p < .01$.

of cool dorsolateral-dorsal cingulate and hot orbital-ventral cingulate EF were considered (r range .31 to .45, r^2 range .10 to .20, p range .049 to <.01).

Repeated-measures multivariate analyses of variance reported in Hale et al. (2011) confirmed the importance of examining FSC impairment when discriminating between MPH responders and nonresponders. With impairment groups as the independent variable in these analyses, we found that participants in the group with no apparent impairment had a relatively poor, nonsignificant MPH response, whereas those with moderate or high FSC factor impairment at baseline demonstrated a robust response to medication, with all but one child determined to be a responder using NPStat single-subject analyses in these two impairment groups. This suggests MPH is more effective for individuals with ADHD who exhibit neuropsychological impairment on cool dorsolateral-dorsal cingulate and hot orbital-ventral cingulate factors. Interestingly, children who were good MPH responders also showed differential dose–response relationships not seen in the low impairment groups, with the best dose for neuropsychological functioning often lower than the best dose for behavior, consistent with basic science findings regarding dopamine agonist effects on the FSC (Arnsten & Pliszka, 2011; Schmeichel & Berridge, 2013).

EVALUATING DIFFERENCES IN ADHD SUBTYPES AND MPH RESPONSE

Although the ADHD Combined type was more likely than the Inattentive type to show FSC factor impairment, it was important to examine if impairment and subtype were related to MPH response. Children diagnosed with ADHD-Combined type ($n = 33$) comprised the largest group, followed by those diagnosed with Inattentive type ($n = 19$) and those diagnosed with Hyperactive-Impulsive type ($n = 4$; too few Ss to analyze further). Displayed in Figure 1, the results illustrate that most children diagnosed with Inattentive type were MPH nonresponders, whereas most children diagnosed with Combined type demonstrated significant MPH response, but this trend was not definitive. When response patterns were evaluated in terms of baseline FSC factor impairment level, a much more prominent finding emerged. For both ADHD groups, it appeared as if FSC factor impairment—not behavioral diagnosis—was the defining feature in determining MPH response.

IMPLICATIONS FOR RESEARCH AND PRACTICE

We began this data-based review with the premise that ADHD does not exist. There are many disorders of attention, some cortical and some subcortical, and these brain systems are certainly interrelated (Koziol et al., 2013), but are they inseparable? It is clear that behavioral diagnosis, which includes all children with attention problems under a heterogeneous ADHD umbrella, minimizes the diagnostic utility and treatment validity of neuropsychological measures. The *NIMH* (Insel, 2013) RDoC approach is a monumental step in the right direction to help understand the disorders of attention, how ADHD is similar or different from these disorders, and how best to treat it. A majority of the evidence suggests that what we currently call "ADHD" is an FSC disorder that leads to significant EF impairments, particularly cognitive and emotional response inhibition, as well as many academic and behavioral problems (e.g., Fair et al., 2012; Semrud-Clikeman et al., 2010). Because

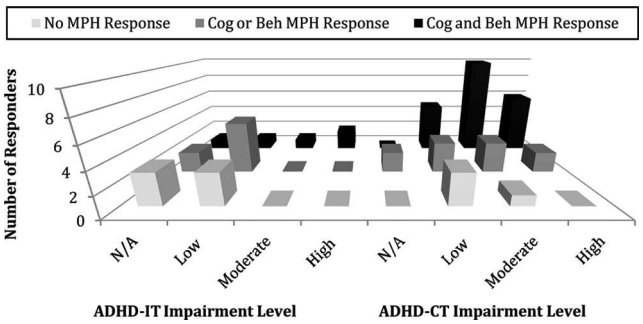

FIGURE 1 Cognitive and behavioral methylphenidate (MPH) response by attention-deficit hyperactivity disorder (ADHD) subtype and impairment level.

neuropsychological measures are sensitive but not specific to ADHD, research is needed to identify the genetic determinants, neurobiological markers, and neuropsychological tests that will help improve diagnostic and treatment validity. Our findings suggest that attention deficit is not as relevant for diagnosing ADHD and determining MPH treatment response as is response inhibition, which appears to be the primary deficit in the disorder (Barkley, 1997). Perhaps it is time to rethink the ADHD label altogether (e.g., Koziol & Budding, 2012), because it is not a disorder of primary attention or attention deficit, but rather one of *intention* (Denckla, 1996) that leads to poor executive control of attentional resources (Hale et al., 2013).

Of the utmost importance is the treatment validity of the position presented here. Because MPH response is not uniform in ADHD (Kubas et al., 2012), which can be explained by differential effects of stimulants on FSC function (Schmeichel & Berridge, 2013), determining the children most likely to benefit from MPH treatment is essential for practitioners and parents alike. Results suggest that while children with behaviorally defined ADHD Combined type are more likely to respond to MPH than those with the Inattentive type, it was the cool dorsal and hot ventral neuropsychological test results that were the strongest indicators of MPH responsive. Although the use of ADHD behavioral criteria remains the current standard-of-care diagnostic method and can provide relevant information regarding the child's behavior, practitioners may also benefit from administering a test battery sensitive to ADHD neuropsychological symptoms. Attending to both neuropsychological and behavioral MPH response could guide MPH titration and other intervention practices, which in turn could lead to improved cognitive, academic, and behavioral outcomes for children with ADHD, especially given that the best dose for cognition appears to be lower than the best dose for behavior in good MPH responders (Hale et al., 2011; Kubas et al., 2012). Given the profound costs that ADHD can have on academic (e.g., Barkley, 2006) and social (e.g., Miller & Hinshaw, 2010) functioning, the potential added benefit of using more thorough neuropsychological and behavioral evaluation of ADHD and MPH treatment response outweighs the costs, especially if both academic and behavioral long-term treatment efficacy is realized.

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