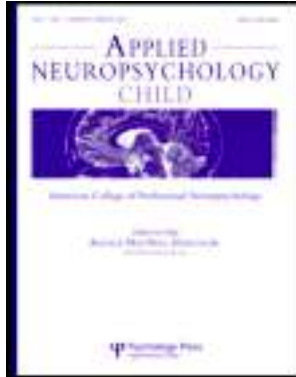


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Requiem for a Diagnosis: Attention-Deficit Hyperactivity Disorder

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GUEST EDITORIAL

Requiem for a Diagnosis: Attention-Deficit Hyperactivity Disorder

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This editorial discusses the diagnosis of ADHD from behavioral and neuropsychological viewpoints. The DSM and clinical neuropsychology offer two completely different nomenclatures while brain-behavior relationships do not easily “map” on to the symptom picture of ADHD. Neuropsychological evaluation offers specificity in identifying and treating individual ADHD presentations, avoiding the heterogeneity inherent in the DSM diagnosis of ADHD.

Key words: ADHD, attention-deficit hyperactivity disorder, diagnostic systems, executive functioning

Attention-deficit hyperactivity disorder (ADHD) is a behaviorally defined diagnosis. The *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association, 2000) lists 18 possible behavioral symptoms for diagnosing this condition. Nine observations concern criteria for inattention, 6 symptoms concern hyperactivity, and 3 observations pertain to impulsivity. This observational methodology allows for classifying three subtypes of ADHD (combined type, predominantly inattentive type, and predominantly hyperactive-impulsive type). However, children who present with a diagnosis of ADHD, regardless of behaviorally defined subtype, remain a highly heterogeneous population. In addition, the symptoms of ADHD are not unique to that diagnosis and can be seen in a variety of other behaviorally defined conditions. While ADHD can be considered an executive dysfunction disorder (Barkley, 1997; Brown, 2009; Miller, Gelfand, & Hinshaw, 2011), patients with a variety of other conditions

can demonstrate deficits in several elements of executive control, particularly when considering executive functioning (EF) includes aspects of both cognitive and visceral functions (Starkstein & Kremer, 2001). In fact, symptoms in various DSM categories can overlap to such a degree that differential diagnosis becomes extremely problematic and comorbidity is more rule than exception. In one investigation, children presenting for clinical evaluation met full DSM-Fourth Edition criteria for one to five diagnoses (Yaryura-Tobias, Rabinowitz, & Neziroglu, 2003). Nevertheless, proponents of the DSM system advocate that all that is necessary to make a differential diagnosis of ADHD is a set of observational rating scales (Barkley, 2006). Many defenders of this behaviorally defined system criticize, marginalize, and even negate the role that neuropsychological assessment can play in the process of differential diagnosis. Yet, it has been demonstrated that this behavioral method of diagnosing ADHD and its DSM subtypes is highly unstable and capricious, while influenced by the subjective perspectives of informants, by the informants who are chosen, by instrumentation or types of rating scale methodologies administered, and by the manner in which

information from multiple sources is aggregated (Valo & Tannock, 2010).

The diagnostic categories described in the DSM are not anatomically organized. Conditions in the DSM are actually driven by a variety of different brain networks, dependent upon the presenting symptom picture. The DSM implies that a “diagnosis” is a monolithic category, despite a variety of symptom presentations and the differing underlying anatomic networks that drive them. Obsessive-compulsive disorder (OCD) serves as just one example: Neuroimaging data have demonstrated that the symptom dimensions of washing, checking, and hoarding are all driven by different neural networks (Mataix-Cols et al., 2004; van den Heuvel et al., 2009). OCD is not a unitary nosologic entity, even though the DSM might mistakenly lead us to believe that it is. The diverse presentations and symptom dimensions of ADHD are also mediated by relatively distinct components of frontal–striatal–pallidal–thalamic circuits as well as prefrontal–cerebellar circuits implicated in cognitive, motivational, and emotional processing (Ashtari et al., 2005; Bledsoe, Semrud-Clikeman, & Pliszka, 2009; Depue et al., 2010; Mackie et al., 2007; Marsh, Maia, & Peterson, 2009; Sagvolden, Johansen, Aase, & Russell, 2005; Sonuga-Barke, 2003). There is no evidence to conclude that ADHD is subtyped correctly within the DSM when neuroanatomic substrates are investigated. Just as is the case with OCD, ADHD is not “one thing,” nor is the condition’s heterogeneity driven by a single neuroanatomy.

NEUROPSYCHOLOGY AND ADHD

Neuropsychology’s nomenclature is descriptive. The main diagnostic goal of neuropsychological evaluation is to identify and characterize brain–behavior relationships. This represents an inherently different process than generating a diagnosis on the basis of behaviorally defined criteria. DSM reference-based criteria for applying particular neuropsychological test findings do not exist; a neuropsychologist has no way to specifically reference a DSM observation such as “has difficulty organizing tasks and activities” with a particular neuropsychological test result. Any attempt to make this judgment comprises a clinical inference based upon examiner experience rather than statistically based correspondence. We certainly acknowledge that studies have attempted to correlate the DSM diagnosis of ADHD with specific test profiles. However, this methodology essentially reveals the percentage of time the test results and the diagnosis co-occur. There is no specific neuropsychological test profile for ADHD other than frequent occurrence of EF deficits (Biederman et al., 2004; Brown, 2009). Moreover, neuropsychological test results have not been useful in identifying the subtypes of ADHD as defined by the DSM (Doyle, Biederman,

Seidman, Weber, & Faraone, 2000; Hinshaw, Carte, Sami, Treuting, & Zupan, 2002). The inconsistency in test results reflects ADHD’s heterogeneity as a syndrome. Proponents of behaviorally defined diagnostic systems use this heterogeneous data to conclude that neuropsychological tests are not useful in diagnosing ADHD. However, we believe this argument is very seriously flawed. A neuropsychological interpretation is more than compelling. These same studies can just as readily be interpreted to demonstrate the very heterogeneity of ADHD, the associated use and necessity of neuropsychological evaluation in identifying specific deficits from which to formulate treatment strategies, and that the category of ADHD itself is too heterogeneous to represent a meaningful diagnostic conceptualization as it currently stands.

The cognitive experimental data gathered during the last two decades demonstrate that the information-processing deficits demonstrated by children with ADHD rarely involve a unitary construct of disturbed “attention” (Denckla & Reiss, 1997; Douglas, 1988; Douglas, Barr, Amin, O’Neill, & Britton, 1988). Instead, the most common and consistent deficit found among the proposed subtypes of ADHD is characterized by a specific “EF” deficit in response inhibition (Aron & Poldrack, 2005; Voeller, 2004). Although it remains controversial, it is believed that psychostimulant medication specifically targets this deficit (Arnsten & Pliszka, 2011; Berridge & Devilbiss, 2011; Berridge et al., 2006; Wilens, 2008). This deficit belongs within the category of *intention* rather than within the domain of *attention* (Denckla & Reiss). In this regard, ADHD can perhaps be better understood as a manifestation of a deficit within the brain’s intention programs (Koziol & Budding, 2009).

Individuals diagnosed with ADHD do not consistently respond to stimulant medications. Further, within the group of those who do respond to stimulant medications, other EF deficits persist. For example, impairment in working memory, a very common finding in ADHD, can be notoriously persistent (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Martinussen & Tannock, 2006). Similarly, planning and organizational deficits and “slow personal tempo” (slow “processing speed”), which are other EF impairments, do not demonstrate predictable response to medication treatment approaches. ADHD is a collection of different symptoms as it is currently defined in the DSM. One can arrive at the diagnosis by different combinations of symptoms; it is not a pure “diagnosis” that carries with it a clear methodology for intervention.

ETIOLOGY

ADHD has multiple etiologies. Because the disorder is highly inheritable, considerable focus has been placed upon

examining genetic influences and multiple vulnerability genes have been identified (Miller et al., 2011). However, numerous other factors significantly increase the risk for a diagnosis of ADHD. These factors include elevated bilirubin levels, preterm birth, low birth weight, perinatal hypoxia and ischemic events, maternal metabolic disorders such as diabetes and phenylketonuria, as well as maternal alcohol use, smoking, and the use of certain medications during pregnancy (Gatzke-Kopp, 2011; Johnson & Bhutani, 2011; Thapar, Cooper, Jefferies, & Stergiakouli, 2011). Any abnormality within the frontal–thalamic circuitry system and/or the cerebro–cerebellar circuitry profile also increases the likelihood of the disorder (Voeller, 2004). It remains unknown how genetic risk factors and these other risk factors might interact to increase the likelihood of the diagnosis. We believe that these multiple etiologies speak to the heterogeneity of the disorder and its frequent comorbidity with other conditions.

CONCLUSIONS

ADHD is clearly a condition characterized by multiple etiologies, frequent diagnostic comorbidity, and a wide range of behavioral presentations. It can be considered a very general “EF” disorder most often characterized by defective response inhibition, which represents a disorder of intention rather than a disorder of attention. Concluding that neuropsychological evaluation is of limited or no value in making this diagnosis represents a process of faulty reasoning. ADHD is not a monolithic entity, while the only methodology for identifying the specific brain–behavior relationships that characterize each unique case presentation involves functional neuropsychological assessment. Through such comprehensive evaluation, the brain–behavior relationships that drive symptom presentations can be identified for the development of effective treatment directives and interventions. In this regard, the term “ADHD” has outgrown its usefulness as a diagnosis. Its “requiem” represents an opportunity for the advancement and promulgation of neuropsychological services.

REFERENCES

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Arnsten, A. F. T., & Pliszka, S. R. (2011). Catecholamine influences on prefrontal cortical function: Relevance to treatment of attention-deficit hyperactivity disorder and related disorders. *Pharmacology Biochemistry & Behavior*, *99*, 211–216.

Aron, A. R., & Poldrack, R. A. (2005). The cognitive neuroscience of response inhibition: Relevance for genetic research in attention-deficit hyperactivity disorder. *Biological Psychiatry*, *57*, 1285–1292.

Ashtari, M., Kumra, S., Bhaskar, S. L., Clarke, T., Thaden, E., Cervellione, K. L., . . . Ardekani, B. A. (2005). Attention-deficit

hyperactivity disorder: A preliminary diffusion tensor imaging study. *Biological Psychiatry*, *57*, 448–455.

Barkley, R. A. (1997). *ADHD and the nature of self-control*. New York, NY: Guilford.

Barkley, R. A. (2006). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (3rd ed.). New York, NY: Guilford.

Berridge, C. W., & Devilbiss, D. M. (2011). Psychostimulants as cognitive enhancers: The prefrontal cortex, catecholamines, and attention-deficit hyperactivity disorder. *Biological Psychiatry*, *69*, e101–e111.

Berridge, C. W., Devilbiss, D. M., Andrzejewski, M. E., Arnsten, A. F., Kelley, A. E., Schmeichel, B., . . . Spencer, R. C. (2006). Methylphenidate preferentially increases catecholamine neurotransmission within the prefrontal cortex at low doses that enhance cognitive function. *Biological Psychiatry*, *60*, 1111–1120.

Biederman, J., Monuteaux, M. C., Doyle, A. E., Seidman, L. J., Wilens, T. E., Ferrero, F., . . . Faraone, S. V. (2004). Impact of executive function deficits and attention-deficit hyperactivity disorder (ADHD) on academic outcomes in children. *Journal of Consulting and Clinical Psychology*, *72*, 10.

Bledsoe, J., Semrud-Clikeman, M., & Pliszka, S. R. (2009). An MRI study of the cerebellar vermis in chronically treated and treatment-naïve children with ADHD-combined type. *Biological Psychiatry*, *65*, 620.

Brown, T. E. (2009). ADD/ADHD and impaired executive function in clinical practice. *Current Attention Disorders Reports*, *1*, 37–41.

Denckla, M. B., & Reiss, A. L. (1997). Prefrontal–subcortical circuits in developmental disorders. In N. A. Krasnegor, G. R. Lyon, & P. S. Goldman-Rakic (Eds.), *Development of the prefrontal cortex: Evolution, neurobiology, and behavior* (pp. 283–294). Baltimore, MD: P. H. Brookes.

Depue, B. E., Burgess, G. C., Willcutt, E. G., Bidwell, L., Ruzic, L., & Banich, M. T. (2010). Symptom-correlated brain regions in young adults with combined-type ADHD: Their organization, variability, and relation to behavioral performance. *Psychiatry Research: Neuroimaging*, *182*, 96–102.

Douglas, V. I. (1988). Cognitive deficits in children with attention-deficit disorder with hyperactivity. In L. M. Bloomingdale & J. Sergeant (Eds.), *Attention-deficit disorder: Criteria, cognition, intervention* (pp. 65–81). Oxford, UK: Pergamon Press.

Douglas, V. I., Barr, R. G., Amin, K., O’Neill, M. E., & Britton, B. G. (1988). Dosage effects and individual responsivity to methylphenidate in attention-deficit disorder. *Journal of Child Psychology and Psychiatry*, *29*, 453–475.

Doyle, A. E., Biederman, J., Seidman, L. J., Weber, W., & Faraone, S. V. (2000). Diagnostic efficiency of neuropsychological test scores for discriminating boys with and without attention-deficit hyperactivity disorder. *Journal of Consulting and Clinical Psychology*, *68*, 477–488.

Gatzke-Kopp, L. M. (2011). The canary in the coalmine: The sensitivity of mesolimbic dopamine to environmental adversity during development. *Neuroscience & Biobehavioral Reviews*, *35*, 794–803.

Hinshaw, S. P., Carte, E. T., Sami, N., Treuting, J. J., & Zupan, B. A. (2002). Preadolescent girls with attention-deficit hyperactivity disorder: II. Neuropsychological performance in relation to subtypes and individual classification. *Journal of Consulting and Clinical Psychology*, *70*, 1099–1111.

Johnson, L., & Bhutani, V. K. (2011). The clinical syndrome of bilirubin-induced neurologic dysfunction. *Seminars in Perinatology*, *35*, 101–113.

Koziol, L. F., & Budding, D. E. (2009). *Subcortical structures and cognition: Implications for neuropsychological assessment*. New York, NY: Springer.

Mackie, S., Shaw, P., Lenroot, R., Pierson, R., Greenstein, D. K., Nugent, T. F., III, . . . Rapoport, J. L. (2007). Cerebellar development and clinical outcome in attention-deficit hyperactivity disorder. *American Journal of Psychiatry*, *164*, 647–655.

Marsh, R., Maia, T. V., & Peterson, B. S. (2009). Functional disturbances within frontostriatal circuits across multiple childhood psychopathologies. *American Journal of Psychiatry*, *166*, 664–674.

Martinussen, R., Hayden, J., Hogg-Johnson, S., & Tannock, R. (2005). A meta-analysis of working memory impairments in children with

- attention-deficit hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 44, 377–384.
- Martinussen, R., & Tannock, R. (2006). Working memory impairments in children with attention-deficit hyperactivity disorder with and without comorbid language-learning disorders. *Journal of Clinical and Experimental Neuropsychology*, 28, 1073–1094.
- Mataix-Cols, D., Wooderson, S., Lawrence, N., Brammer, M. J., Speckens, A., & Phillips, M. L. (2004). Distinct neural correlates of washing, checking, and hoarding symptom dimensions in obsessive-compulsive disorder. *Archives of General Psychiatry*, 61, 564.
- Miller, M., Gelfand, J., & Hinshaw, S. (2011). Attention-deficit hyperactivity disorder. In A. S. Davis (Ed.), *Handbook of pediatric neuropsychology* (pp. 565–580). New York, NY: Springer.
- Sagvolden, T., Johansen, E. B., Aase, H., & Russell, V. A. (2005). A dynamic developmental theory of attention-deficit hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Sciences*, 28, 397–419.
- Sonuga-Barke, E. J. (2003). The dual pathway model of ADHD: An elaboration of neurodevelopmental characteristics. *Neuroscience and Biobehavioral Reviews*, 27, 593–604.
- Starkstein, S. E., & Kremer, J. (2001). The disinhibition syndrome and frontal-subcortical circuits. In D. Lichter & J. Cummings (Eds.), *Frontal-subcortical circuits in neurological and psychiatric disorders* (pp. 163–176). New York, NY: Guilford.
- Thapar, A., Cooper, M., Jefferies, R., & Stergiakouli, E. (2011). What causes attention-deficit hyperactivity disorder? *Archives of Disease in Childhood*. Advance online publication. doi:10.1136/archdischild-2011-300482
- Valo, S., & Tannock, R. (2010). Diagnostic instability of DSM-IV ADHD subtypes: Effects of informant source, instrumentation, and methods for combining symptom reports. *Journal of Clinical Child and Adolescent Psychology*, 39, 749–760.
- van den Heuvel, O. A., Remijnse, P. L., Mataix-Cols, D., Vrenken, H., Groenewegen, H. J., Uylings, H. B., . . . Veltman, D. J. (2009). The major symptom dimensions of obsessive-compulsive disorder are mediated by partially distinct neural systems. *Brain: A Journal of Neurology*, 132, 853.
- Voeller, K. K. (2004). Attention-deficit hyperactivity disorder (ADHD). *Journal of Child Neurology*, 19, 798–814.
- Wilens, T. E. (2008). Effects of methylphenidate on the catecholaminergic system in attention-deficit hyperactivity disorder. *Journal of Clinical Psychopharmacology*, 28, S46–S53.
- Yaryura-Tobias, J. A., Rabinowitz, D. C., & Neziroglu, F. (2003). Possible basal ganglia pathology in children with complex symptoms. *Journal of Clinical Psychiatry*, 64, 1495–1501.