

## An Overview of the Advances in the Understanding of the Neuropsychology Attention-Deficit/Hyperactivity Disorder

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### Abstract

This article provides an overview of the significant advances that have been made in the understanding of the neuropsychology of Attention Deficit/Hyperactivity Disorder over the past 30 years. The major advances that are reviewed are: 1) context dependence, 2) etiological overlap between symptoms and neuropsychological deficits, 3) complexity and heterogeneity, and 4) developmental neuropsychological phenotypes. Each presentation is followed by a discussion of the implications of the advance for clinical practice.

### Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is the most common pediatric neurodevelopmental disorder (American Academy of Pediatrics, 2011) with a prevalence in the United States of 10.84% (14.10% males and 7.57% females) diagnosed in accordance with DSM-5 criteria (Vande Voort, He, Jameson, & Merikangas, 2014), which are the most scientifically validated criteria to date (Roberts, Milich, & Barkley, 2014). Hence, it is incumbent upon mental health providers, including pediatricians, to stay abreast of current advances on this disorder. However, because the scientific literature on ADHD has been increasing exponentially in the past decades with at least 800-1,000 journal articles published on an annual basis (Barkley, 2014a), it is a tremendous challenge for the average practitioner to stay current with this avalanche of literature that should inform their clinical practice. This avalanche is especially evident in the neuropsychology of ADHD (Barkley, 2014a). To help the practitioner stay informed on this topic, this article provides an overview of the major advances that have been made in the understanding of the neuropsychology of ADHD over the past 30 years (Sonuga-Barke & Coghill, 2014),<sup>1</sup> coupled with a discussion of the implications of the advances. This topic is especially apt given the recent decision of the National Institute of Mental Health to emphasize the importance of the neuropsychological underpinnings of mental disorders (Cuthbert & Kozak, 2013; Insel, 2014).

The major advances that are reviewed are those that have been identified by Sonuga-Barke and Coghill (2014). They are: 1) context dependence, 2) etiological overlap between symptoms and neuropsychological deficits, 3) complexity and heterogeneity, and 4) developmental neuropsychological phenotypes. Each presentation is followed by a discussion of the implications of the advance for clinical practice.

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<sup>1</sup> Sonuga-Barke and Coghill (2014) is a current authoritative review by two of the acknowledged experts on ADHD. Hence the validity of the advances they have identified, and which provide the foundation for the present overview, can be accepted with confidence. Also, the “advances” should be understood as applying equally to juveniles and adults.

### **Contextual Dependence**

A neuropsychological model in which ADHD was conceptualized to be a disorder like Down's syndrome in that it presented with a fixed deficit in all situations has been replaced with a model of ADHD in which neuropsychological deficits are seen as being highly context dependent (Nigg & Barkley, 2014; Songua-Barke & Coghill, 2014). Namely, it is now recognized that the deficits associated with ADHD are evident in some situations and not at all in others (Nigg & Barkley, 2014). Several contextual factors influence the extent to which the neuropsychological deficits are expressed. For example, performance is worse for those diagnosed with ADHD when engaging in more complex strategies requiring organization, under low levels of stimulation which they find boring, uninteresting, or in tasks in which there are long delays in reinforcement (Brown, 2013; Nigg & Barkley, 2014). However, they can typically do quite well in several different contexts such as: a) contexts that the individual finds quite interesting, intriguing, exciting, novel, etc., b) contexts in which the individual is faced with an imminent deadline and expects significant negative consequences to occur quite soon after the failure to meet the deadline, c) contexts in which the individual is receiving frequent rewards for appropriate behavior, or d) contexts in which the individual is under close supervision (American Psychiatric Association, APA, 2013; Brown, 2013).

### **Clinical Implications**

First, it is important for the clinician to recognize that marked fluctuation in performance for those with ADHD depending upon the context is not only not contraindicative of the disorder, but rather is of its essence (Brown, 2013). Second, the DSM-5 requirement that impairments from ADHD symptoms be present in two or more settings needs to be tempered by the realization that the "presence" might be markedly attenuated depending upon the characteristics of the setting. Hence an overly rigorous interpretation of this criterion should be avoided as it may falsely lead the clinician to conclude that the individual does not have ADHD. For example, with regard to a child, their ADHD might be much more apparent in a school setting which places far more emphasis on behavioral control, attention, and organization than in a home setting which has less stringent expectations in this regard. This variability is to be expected and is by no means contraindicative of an ADHD diagnosis.

### **Etiological Overlap between Symptoms and Neuropsychological Deficits**

It has long been known that ADHD runs in families with most of this similarity due to genetic factors as evidenced by a heritability of approximately 70% (Nigg & Barkley, 2014). Although ADHD is a highly heritable disorder, it has proven difficult to identify its precise molecular genetic basis (Sonuga & Barke, 2014). Hence an interest has developed in identifying endophenotypes to increase the ability to detect the genes involved in ADHD. An endophenotype is defined as a phenotype which can be measured at a cognitive or neurobiological level, which is more proximate to the biological etiology of a clinical disorder than the behavioral phenotype, and which is influenced by one or more of the same susceptibility genes as the condition" (Gau & Shang, 2010, p. 838). Recently, advances have been made in identifying such endophenotypes. For example, Gau and Shang (2010) found that unaffected siblings of children with ADHD performed poorly on a broad range of executive functions such as short-term spatial memory, verbal and spatial working memory, spatial planning and sustained attention compared to controls, with deficits similar to those of their ADHD siblings. Thus executive dysfunction appears to be a useful endophenotype for ADHD.

### **Clinical Implications**

Although the sibling(s) of a juvenile with ADHD may appear to be unaffected in terms of not meeting the full clinical diagnostic criteria for ADHD, they may demonstrate impairment when performing complex academic tasks as they may have subthreshold ADHD (Gau & Shang, 2010). For example, a recent nationwide (Korea) study of a community sample of 921 children (aged 8-11 years) found that children presenting with subthreshold ADHD symptoms (i.e., 3-5 symptoms) experienced significant functional impairments across multiple domains (academic, behavioral, emotional) in contrast to a control group (Hong et al., 2014). Although the functional impairments were less severe than those of children who met full criteria for a diagnosis of ADHD, the results of the study supported the clinical relevance of subthreshold ADHD. Parenthetically, it is interesting also to note that stimulant treatment has been found to be effective for subthreshold ADHD (Hinshaw & Scheffler, 2014). Indeed DSM-5 (APA, 2013) also obliquely acknowledges the clinical validity of subthreshold ADHD by providing a classification of “Other Specified Attention-Deficit/Hyperactivity Disorder.” This classification applies to presentations in which symptoms characteristic of ADHD that cause significant impairment and are below the threshold of 6 criteria for children. Hence, the apparently unaffected sibling(s) of an ADHD proband should be screened for subthreshold ADHD and interventions implemented if needed.

### **Complexity and Heterogeneity**

The idea there is a single core deficit shared by all individuals with ADHD has yielded to a consensus that ADHD is characterized by complexity and heterogeneity (Sonuga-Barke & Coghill, 2014). Complexity means that there are deficits in multiple neuropsychological processes and brain systems which even Russell Barkley, the most influential of the single core deficit theorists (Coghill, 2014), has come to acknowledge in the most recent iteration of his theory (Nigg & Barkley, 2014). Indeed, some theorists posit as many as 6 or 7 distinct neuropsychological deficits (Brown, 2013; Fair, Bathula, Nikolas, & Nigg, 2012). Heterogeneity, a consequence of the complexity, means that individuals with ADHD display markedly different profiles of neuropsychological deficits depending upon which neuropsychological functions are impaired. What remains unclear is exactly how many different independent neuropsychological deficits there are and how they relate to one another (Coghill, 2014). Two of the deficits warrant specific discussion as they have only recently been recognized and the evidence supporting their involvement in ADHD is impressive. They are deficits in alerting/arousal and emotional regulation.

### **Alerting/Arousal**

Attention is a multi-dimensional neuropsychological construct with at least three well-validated distinct networks, each with a discreet anatomical basis (Petersen & Posner, 2012; Roberts et al., 2014). What is termed the alerting/arousal network by Petersen and Posner is involved in producing and maintaining optimal vigilance and performance during tasks. More than two decades ago, Thomas Brown (1993) discovered a group of individuals who were not hyperactive but hypoactive and who demonstrated symptoms of inattention that were different from those typically found in ADHD. They were described as “often stares into space,” “daydreamy,” “often appears to be low in energy, sluggish, drowsy.” In the field trials of symptom utility for DSM-IV which was reported in 1994, two symptoms that were more

diagnostic of attention problems than all but one of the symptoms included in the official DSM list were identified (i.e., “drowsy” and “daydreams”). Nevertheless, these symptoms never made it onto the list, nor are they in DSM-5 (APA, 2013; Frick et al., 1994). What this finding supported, though it was not recognized at that time, was that there was a type of attention disorder that was different from ADHD. In the intervening two decades substantial evidence has accumulated that has provided strong support for this theory in that there are individuals who exhibit a cluster of symptoms that are related to but distinct from the inattentive symptom cluster in ADHD. These individuals are described as having difficulty regulating alertness and sustaining effort and exhibiting symptoms of daydreaming and cognitive sluggishness (Barkley, 2014b; Brown, 2013). Barkley (2014b) has persuasively argued that this symptom cluster, initially designated as “sluggish cognitive tempo,” represents a new attention disorder which he has renamed “concentration deficit disorder” and which he suggests characterizes 30-40% who previously had been diagnosed with DSM-IV predominantly inattentive type of ADHD.<sup>2</sup> Although the precise pathophysiology of this disorder has yet to be established, Barkley (2014b) suggests that it represents a dysfunction in the vigilance component of attention, i.e., the alerting/arousal network identified by Petersen and Posner.

### **Emotional Regulation**

Although a deficit in emotional regulation has a long history of being a core component of ADHD in its clinical descriptions, the DSM in its various iterations has consistently relegated it to a lesser status of an associated feature that occurs some of the time (Barkley, 2014c). Recently, Barkley (2014c) has marshaled an impressive array of compelling evidence that convincingly establishes deficient emotional regulation as a core component of ADHD that is comparable in centrality to the universally accepted components of hyperactivity/impulsivity and inattention. The commonly occurring symptoms in ADHD of low frustration tolerance, impatience, and anger are best understood as being primarily a direct consequence of ADHD rather than being a manifestation of a comorbid disorder such as oppositional defiant disorder (ODD). This raises the question of how the emotional dysregulation symptoms of ADHD might be distinguished from ODD symptoms. The answer resides in the emerging consensus that ODD symptoms are best understood in terms of a bifactor model (Burke et al., 2014). One factor, termed *irritability*, is comprised of the symptoms: *temper*, *touchy*, *angry*. The second factor, termed *oppositional behavior*, is comprised of the symptoms: *argue*, *defies*, *annoys*, *blames*, *spiteful*. It is this latter factor that distinguishes ODD from ADHD.

### **Clinical Implications**

The clinical implications are fourfold. First, it would be simplistic to explain the etiology of ADHD to a client and his or her family in terms of a single neuropsychological deficit (Coghill, 2014; Sonuga-Barke & Coghill, 2014). Second, the neuropsychological heterogeneity of ADHD results in a low level of sensitivity thereby calling into question the common practice of using neuropsychological tests to diagnose ADHD (Coghill, 2014; Sonuga-Barke & Coghill, 2014). When this serious limitation is added to the dubious ecological validity of

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<sup>2</sup> The reader who is interested in the differential diagnosis between ADHD and concentration deficit disorder should consult Barkley (2014b) for an excellent discussion.

neuropsychological tests (Roberts et al., 2014)<sup>3</sup>, there is even further reason to question their utility in diagnosing ADHD. Third, when the presenting complaints seem to involve attention problems, an evaluation for ADHD with predominantly inattentive presentation should include a careful exploration of symptoms of a “concentration deficit disorder.” Barkley’s (2014b) superb chapter on this disorder is enormously helpful for conducting such a thorough evaluation. Fourth, understanding that deficient emotional regulation is a core component of ADHD will reduce the likelihood of attributing all emotional problems in ADHD as being entirely due to comorbidity (Barkley, 2014c). Of course, although not all emotional problems are simply a direct manifestation of emotional dysregulation (e.g., see Pliszka, 2014 for a review on comorbidity), this deficit clearly is an important risk factor for such problems (Barkley, 2014c).

### **Developmental Neuropsychological Phenotypes**

Although earlier it was believed that a diagnosis of ADHD was outgrown by adolescence, it is now clear that there is a relatively high rate of persistence of ADHD from childhood to adolescence (50-80%) and into adulthood (35-65%) [Owens, Cardoos, & Hinshaw, 2014]. Among the many factors that have commonly been identified as affecting persistence across development (e.g., symptom severity, disorder co-morbidity, family history of ADHD) [Owens, Cardoos, & Hinshaw, 2014], the previously discussed advances that have been made in identifying neuropsychological factors involved in ADHD support that these different factors may result in different developmental phenotypes, i.e. a different pattern of emergence, persistence, and outcome (Sonuga-Barke & Coghill, 2014; Sonuga-Barke & Halperin, 2010). For example, a phenotype characterized by both behavioral and emotional impulsivity markedly increases the risk that ADHD will lead to the development of oppositional defiant disorder, followed by conduct disorder (Beauchaine, Hinshaw, & Pang, 2010).

### **Clinical Implications**

Because different developmental ADHD phenotypes are rooted in different neuropsychological impairments, this generates a significant implication for treatment. Namely, nonpharmacological treatments targeting the specific neuropsychological impairment associated with the phenotype offer the promise of an effective treatment for the phenotype. Unfortunately, this prospect has yet to be convincingly realized. For example, recent meta-analytic reviews of cognitive training programs, as well as neurofeedback, have not been found to be effective for the various neuropsychological impairments associated with ADHD such as attention, working memory, and behavior (Melby-Lervag & Hulme, 2013; Orban, Rapport, Friedman, & Kofler, 2014; Shipstead, Redick, & Engle, 2012; Vollebregt, van Dongen-Boomsma, Buitelaar, and Slatts-Willemsse, 2014). Thus the development of effective non-pharmacological treatments to treat the neuropsychological impairments that undergird different developmental ADHD phenotypes is very much a work in progress.

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<sup>3</sup> The relationship between neuropsychological tests of executive functions and real-life adaptive functioning is very low for those with ADHD (Roberts, Milich, & Barkley, 2014).

## Conclusion

This overview has identified four of the most significant advances in understanding the neuropsychology of ADHD that have been made in the recent decades. Research on this aspect of ADHD can be expected to continue to be abundant in the succeeding years, especially with regard to attempts to develop a neuropsychologically-based classification of ADHD (Barkley, 2014a; Rommelse & Zeeuw, 2014), as this goal is particularly congruent with National Institute of Mental Health's emphasis on the importance of the neuropsychological underpinnings of mental disorders (Cuthbert & Kozak, 2013; Insel, 2014). Hence it behooves the practicing clinician who is interested in maintaining a competence in the assessment and treatment of ADHD to stay especially informed on advances in this domain.

## References

- American Academy of Pediatrics (2011). ADHD: Clinical Practice Guideline for the Diagnosis, Evaluation, and Treatment of Attention-Deficit/Hyperactivity Disorder in Children and Adolescents. *Pediatrics*, 128, 1007-1022.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Barkley, R. (2014). *Attention-deficit hyperactivity disorder*, 4<sup>th</sup> ed. New York: Guilford Press.
- Barkley, R. (2014a). Preface. In R. Barkley (Ed.), *Attention-deficit hyperactivity disorder*, 4<sup>th</sup> ed. (pp. ix-x). New York: Guilford Press.
- Barkley, R. (2014b). Concentration deficit disorder (Sluggish cognitive tempo). In R. Barkley (Ed.), *Attention-deficit hyperactivity disorder*, 4<sup>th</sup> ed. (pp. 435-454). New York: Guilford Press.
- Barkley, R. (2014c). Emotional dysregulation is a core component of ADHD, In R. Barkley (Ed.), *Attention-deficit hyperactivity disorder*, 4<sup>th</sup> ed. (pp. 81-115). New York: Guilford Press.
- Beauchaine, T., Hinshaw, S., & Pang, K. (2010). Comorbidity of attention-deficit/hyperactivity disorder and early-onset conduct disorder. *Clinical Psychology: Science and Practice*, 17, 327-336.
- Brown, T. (1993). Attention deficit disorders without hyperactivity. *Chadder*, Spring/Summer, 7-10.
- Brown, T. (2013). *A new understanding of ADHD in children and adults: Executive function impairments*. New York, NY: Routledge.
- Burke, J., Rowe, R., Boylan, K., Duku, E., Waldman, I., Stepp, S., ... Hipwell, A. (2014). Identifying the irritability dimension of ODD: Application of a modified bifactor model across five large community samples of children. *Journal of Abnormal Child Psychology*, 123, 841-851.
- Chacko, A., Bedard, A., Marks, D., Feirsen, N., Uderman, J., Chimiklis, A., ... Ramon, M. (2014). A randomized clinical trial of Cogmed Working Memory Training in school-age children with ADHD: A replication in a diverse sample using a control condition. *Journal of Child Psychology and Psychiatry*, 55, 247-255.
- Coghill, D. (2014). Editorial: Acknowledging complexity and heterogeneity in causality — implications of recent insights into neuropsychology of childhood disorders for clinical practice. *Journal of Child Psychology and Psychiatry*, 55, 737-740.

- Cuthbert B. & Kozak M. (2013). Constructing constructs for psychopathology: The NIMH research domain criteria. *Journal of Abnormal Psychology, 122*, 928-937.
- Fair, D., Bathula, D., Nikolas, M., & Nigg, J. (2012). Distinct neuropsychological subgroups in typically developing youth inform heterogeneity in children with ADHD. *PNAS, 109*, 6769-6774.
- Frick, P., Lahey, B., Applegate, B., Kerdyck, L., Ollendick, T., Hynd, G., ... Barkley, R. (1994). DSM-IV field trials for disruptive behavior disorders: Symptom utility estimates. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*, 529-539.
- Gau, S., & Shang, C. (2010). Executive functions as endophenotypes in ADHD: evidence from the Cambridge neuropsychological test battery (CANTAB). *Journal of Child Psychology and Psychiatry, 51*, 838-849.
- Hinshaw, S., & Scheffler, R. (2014) *The ADHD explosion*. New York, NY: Oxford University Press.
- Hong, S., Dwyer, D., Kim, J., Park, K., Shin, M., Kim, B., Yoo, H., ... Cho, S. (2014). Subthreshold attention-deficit/hyperactivity disorder is associated with functional impairments across domains: a comprehensive analysis in a large scale community study. *European Child and Adolescent Psychiatry, 23*, 627-636.
- Insel, T. (2014). The NIMH research domain criteria (RDoC) project: Precision medicine for psychiatry. *American Journal of Psychiatry, 171*, 395-397.
- Melby-Lervag, M., & Hulme, C. (2013). Can working memory training ameliorate ADHD and other learning disorders? A systematic review. *ADHD Report, 21*, 1-5.
- Nigg, J. & Barkley, R. (2014). Attention-Deficit/Hyperactivity Disorder. In E. Mash & R. Barkley (Eds.), *Child psychopathology* (3<sup>rd</sup> ed., pp. 75-144). New York: Guilford Press.
- Orban, S., Rapport, M., Friedman, L., & Kofler, M. (2014). Executive function/cognitive training for children with ADHD: Do results warrant the hype and cost? *ADHD Report, 22*, 8-14.
- Owens, E., Cardoos, S., & Hinshaw, S. (2014). Developmental progression and gender differences among individuals with ADHD. In R. Barkley (Ed.), *Attention-deficit hyperactivity disorder*, 4<sup>th</sup> ed. (pp. 223-255). New York: Guilford Press.
- Petersen, S., & Posner, M. (2012). The attention system of the human brain: 20 years after. *Annual Review of Neuroscience, 35*, 73-89.
- Pliszka, S. (2014). Comorbid psychiatric disorders in children with ADHD. In R. Barkley (Ed.), *Attention-deficit hyperactivity disorder*, 4<sup>th</sup> ed. (pp.140-168). New York: Guilford Press.
- Roberts, W., Milich, R., & Barkley, R. (2014). Primary symptoms, diagnostic criteria, subtyping, and prevalence of ADHD. In R. Barkley (Ed.), *Attention-deficit hyperactivity disorder*, 4<sup>th</sup> ed. (pp. 50-80). New York: Guilford Press.
- Rommelse, N., & Zeeuw, P., (2014). Neurobiological measures to classify ADHD: A critical appraisal. *European Journal of Child and Adolescent Psychiatry, 23*, 1151-1164.
- Shipstead, Z., Redick, T., & Engle, R. (2012). Is working memory training effective? *Psychological Bulletin, 138*, 628-654.

- Sonuga-Barke, E., & Coghill, D. (2014). Editorial perspective: Laying the foundations for next generation models of neuropsychology. *Journal of Child Psychology and Psychiatry*, *58*, 1215-1217.
- Sonuga-Barke, E., & Halperin, J. (2010). Developmental phenotypes and causal pathways in attention-deficit/hyperactivity disorder: potential targets for early intervention? *Journal of Child Psychology and Psychiatry*, *51*, 368-389.
- Vande Voort, J., He, J., Jameson, N., & Merikangas, K. (2014). Impact of the DSM-5 Attention-Deficit/Hyperactivity Disorder age-of-onset criterion in the US adolescent population. *Journal of the American Academy of Child and Adolescent Psychiatry*, *53*, 736-744.
- Vollebregt, M., van Dongen-Boomsma, M., Buitelaar, J., & Slaats-Willemse, D. (2014). Does EEG-neurofeedback improve neurocognitive functioning in children with Attention-Deficit/Hyperactivity Disorder? A systematic review and a double-blind placebo controlled study. *Journal of Child Psychology and Psychiatry*, *55*, 460-472.