**Abstract**

The article reviewed the major advances that have been made in the understanding of the neuropsychology of ADHD over the past 30 years in terms of context dependence, etiological overlap between symptoms and neuropsychological deficits, complexity and heterogeneity, and developmental neuropsychological phenotypes. The implications of each advance for clinical practice were also discussed.

Attention-Deficit/Hyperactivity Disorder (ADHD) is the most common neurodevelopmental disorder (American Academy of Pediatrics, 2011) with a prevalence in the United States of 10.84% (14.10% males and 7.57% females) based upon 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 to stay abreast of current advances on this disorder. However, since 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 huge challenge for the average practitioner to stay up to date with this avalanche of literature that should inform their clinical practice. One of the ways to meet this challenge is for the practitioner to access a current review of one of the many important ADHD-related topics. This article will provide a review of one such topic, i.e., the major advances that have been made in the understanding of the neuropsychology of ADHD over the past 30 years (Sonuga-Barke & Coghill, 2014). This focus 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).

The major advances that will be 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, 4) developmental neuropsychological phenotypes. Each presentation will be followed by a discussion of the implications of the advance for clinical practice.

**Context Dependence**

A neuropsychological model in which ADHD was conceptualized to be a disorder like Down 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 seen in ADHD are evident in some situations and not at all in others (Nigg & Barkley, 2014). Several contextual factors influence the extent to which the deficits are expressed. For example, performance is worse for those with ADHD in more complex activities in which organization strategies are required, 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, d) contexts in which the individual is under close supervision (American Psychological 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 rigorist interpretation of this criterion should be avoided as it may lead to a falsely lead the clinician to conclude that the child does not have ADHD.

**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 about .70 (Nigg & Barkley, 2014). However, although there is no doubt that 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, they may well be impaired in performing complex academic tasks as they may have subshreshold ADHD (Gau & Shang, 2010). For example, a recent nation wide (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 impairments were less severe than those of children who met full criteria, the study supported the clinical relevance of subthreshold ADHD. Parenthetically, it is interesting to also 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 which cause significant impairment are below the threshold of 6 criteria. 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 that 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 complexity, means that individuals with ADHD display markedly different profiles 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.

**Alterting/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 suggested, 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 symptom 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. Although the precise pathophysiology of this disorder has yet to established, Barkley (2014) suggests that it represents a dysfunction in the vigilance component of attention, i.e., the alterting/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. Thus commonly occurring symptoms in ADHD of low frustration tolerance, impatience, 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. In short, emotional impulsivity is as much a core component of ADHD as is behavioral impulsivity.

**Clinical Implications**

The clinical implications are fourfold. First, it would be simplistic to explain the etiology of ADHD to a client and their 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 neuropsychological tests (Roberts et al., 2014)[[1]](#footnote--1), 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 for 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).

**Developmental Neuropsychological Phenotypes**

 Although earlier it was believed that 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 indentified 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 indentifying neuropsychological factors involved in ADHD suggest 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 markedly increases the risk that ADHD will lead to the development of oppositional defiant disorder, followed by conduct disorder (Beauchaine, 2013).

**Clinical Implications**

Nonpharmacological treatment that targets specific neuropsychological impairments which result in different developmental phenotypes offers the promise of reducing the persistence of ADHD over the life span. Unfortunately, this prospect has yet to be convincingly realized. Two of the most conspicuous failures involve working memory training and neurofeedback. With regard to working memory, two comprehensive independent meta-analytic reviews of working memory training of individuals with and without ADHD (Melby-Lervag & Hulme, 2013; Shipstead, Redick, & Engle, 2012) plus a recent study (Chacko et al., 2014) converge in supporting the conclusion of Melby-Lervag and Hulme (2013, p. 4) that “…working memory training procedures cannot, based on the evidence to date, be recommended as suitable treatments for developmental disorders (such as ADHD or dyslexia).” With regard to neurofeedback, Vollebregt, van Dongen-Boomsma, Buitelaar, and Slatts-Willemse (2014) concluded on the basis of a systematic literature review combined a double-blind placebo-controlled study which they conducted to add to the literature“Overall, the existing literature and this study fail to support any systematic beneficial effect of EEG-neurofeedback on neurocognitive functioning in ADHD” (p.460). Thus the “promise” has yet to be realized and hence the development of effective nonpharmacological treatments to ameliorate both the symptoms and neuropsychological deficits associated with ADHD is very much a work in progress.

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1. The relationship between neuropsychological tests of executive functions and real-life adaptive functioning is very low for those with ADHD (Roberts, Milich, & Barkley, 2014). [↑](#footnote-ref--1)