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WISC-IV and IVA+Plus Pattern Analysis: Assessment of Attention-Deficit /Hyperactivity Disorder

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WISC-IV AND IVA+PLUS PATTERN ANALYSIS: ASSESSMENT OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

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ABSTRACT

WISC-IV AND IVA+PLUS PATTERN ANALYSIS:
ASSESSMENT OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

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Diagnosis of attention-deficit/hyperactivity disorder (ADHD) is a challenging practice with no definitive diagnostic test. This study sought to evaluate the use of a pattern of performance method of inference in ADHD assessment that examines an individual's general intellectual ability in comparison to his or her own attention skills. WISC-IV scores, IVA+Plus quotients, and basic demographic information were collected from patient files of children previously evaluated by the Eastern Virginia Medical School Neuropsychology Clinic. Children with and without ADHD were compared by analyzing the following difference or delta score discrepancies: (a) WISC-IV General Ability Index (GAI) minus WISC-IV Working Memory Index, (b) WISC-IV GAI minus WISC-IV Processing Speed Index, (c) WISC-IV GAI minus IVA+Plus Full Scale Response Control Quotient, and (d) WISC-IV GAI minus IVA+Plus Full Scale Attention Quotient. Contrary to preliminary hypotheses, analyses demonstrated no significant utility for the use of these delta scores in distinguishing children referred for psychological testing with and without ADHD. Potential reasons for this lack of predicted discriminate ability are posited.

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CHAPTER I

INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) represents developmental deficits in attention and/or inhibition of behaviors. The disorder is primarily identified in childhood. While young children are often impulsive and highly energetic, they are expected to make developmental gains with age in the form of increased forethought, improved attentional abilities, and steady reductions in off-task behaviors and extraneous activity. Without these gains, children may experience significant difficulty managing their behaviors and learning at each successive stage of development.

Children with clinically significant problems in regulating attention, impulsivity, and hyperactivity across settings may meet criteria for the diagnosis of ADHD (American Psychiatric Association [APA], 2000). Studies suggest ADHD negatively impacts a broad range of functioning, including psychosocial, cognitive, and academic abilities (Barkley, Fischer, Smallish, & Fletcher, 2006; Biederman, Faraone, Monuteaux, Bober, & Cadogen, 2004). Academic progress may be hindered as children with significant attention-related difficulties or hyperactive behaviors fail to attend to important lessons or complete required schoolwork. Similarly, experiences necessary for appropriate development that are gained through interactions with peers and significant adults may be missed.

The *Publication Manual of the American Psychological Association* was used as the journal model.

While ADHD may cause clinically significant disruption across the lifespan, there are no definitive diagnostic tests for the disorder. Within the fields of clinical psychology and neuropsychology, ADHD assessment may still be improved by psychological and neuropsychological test methods of objective evaluation that refine the diagnostic process and have less susceptibility to responder bias than parent and teacher completed symptom checklists.

CHAPTER II

LITERATURE REVIEW

Current Diagnostic Criteria

Diagnosis of ADHD is defined by the standards established in the Diagnostic and Statistical Manual, Fourth Edition, Text Revision (DSM-IV-TR; APA, 2000). Defined criteria require clinically significant impairment in social, school, or work functioning with a persistent pattern of hyperactivity, impulsivity, or inattention before age seven. Furthermore, it is important that impairment from symptoms exists in more than one setting for more than six months. Three subtypes of ADHD are defined: Predominantly Inattentive Type, Predominantly Hyperactive/Impulsive Type, and Combined Type. Individuals meeting criteria for ADHD, Predominantly Inattentive Type are identified as having at least six of nine symptoms of developmentally inappropriate attention problems (APA, 2000). These behaviors include difficulty maintaining attention and avoiding tasks that require mental strain. Individuals with at least six of nine hyperactive-impulsive symptoms meet criteria for ADHD, Predominantly Hyperactive-Impulsive Type (APA, 2000). Those with a significant number of symptoms in both categories meet criteria for ADHD, Combined Type. A fourth category, ADHD Not Otherwise Specified, is defined by clinically significant impairments due to hyperactivity, impulsivity, or inattention failing to meet full criteria for a more specific ADHD diagnosis.

Prevalence

Studies have varied significantly in identifying prevalence rates for ADHD. Barkley (2003) attributed these differences to sampling and how ADHD is diagnosed.

Across studies, prevalence rates have ranged from as low as 2% to as high as 23% within the United States (Barkley, 2003). The national prevalence of ADHD is generally estimated to be between 3-7% (APA, 2000). Using DSM-III-R criteria and parent report, Breton et al. (1999) found a similar average prevalence of 4% among children 6 to 14 years old in Quebec. In a national study involving U.S. adults, 6.6% were identified as having met criteria for childhood ADHD with 2.6% continuing to meet criteria (Kessler et al., 2006). More recently, Faraone, Sergeant, Gillberg, and Biederman (2003) examined ADHD prevalence studies worldwide and found that, using DSM-IV criteria, between 2.4-19.8% of children worldwide meet criteria for the diagnosis of ADHD. Faraone et al. (2003) found this range to be very similar to the 11.4-16.1% found in U.S.-based studies examined in the same review. Few socioeconomic differences related to income and urban-rural setting appear to influence ADHD prevalence (Kessler et al, 2006; Szatmari et al., 1989).

Nonetheless, significant variation in incidence rates exists based on a number of factors, including how ADHD is assessed, what geographic region is involved, and what population is studied (Barkley, 2006c). For example, higher rates of ADHD occur when teacher ratings are used to identify dysfunction (Barkley, 2003; Breton et al., 1999). With advancing age, fewer individuals meet criteria for ADHD, though Barkley (2003) attributes this decline in incidence to limitations of DSM-IV criteria that do not adequately capture ADHD impairment into adulthood. Prevalence rates also appear to vary regionally. The Hampton Roads community of southeastern Virginia, for example, demonstrated a significantly higher prevalence of ADHD than the national average. Compared to an estimated 3-5% national prevalence rate, LeFever, Dawson, and Morrow

(1999) found 8-10% of students within Hampton Roads received ADHD medication during the school day. LeFever, Dawson, et al. (1999) suggest these regional differences may be indicative of differences in criteria clinically used for ADHD diagnosis. Specifically, conflicts between care providers and care practices were identified as major barriers to adequate assessment and treatment of ADHD in the Hampton Roads area (LeFever, Butterfoss, & Vislocky, 1999). Children with possible ADHD are commonly observed by different professionals (e.g. teachers, psychologists, primary care physicians, and psychiatrists) in a variety of settings, such as home and school. LeFever, Butterfoss, et al. (1999) note that within geographically large and complex communities, such as Hampton Roads, these various professionals are unlikely to coordinate efforts for consistent assessment of ADHD. Therefore, care providers may not agree on the diagnosis of ADHD for a particular child. When one professional relies solely on parent report while another attempts to collect information from a variety of sources including clinic test data, the accuracy and rates of ADHD diagnosis can be highly variable. When professionals disagree, it is also unclear which diagnostic decision will be applied for a particular child. For example, providing stimulant medication for a child when sleep disruption has not been adequately ruled out as a cause for attention-related difficulties may lead to further problems for the child and his or her teachers and parents.

Gender differences. Gender effects have also been noted in the diagnosis and expression of ADHD. The DSM-IV-TR reports male-to-female prevalence ratios ranging from 2:1 to 9:1. Szatmari, Offord, and Boyle (1989) found a 3:1 male to female ratio in a Canadian study. Meta-analysis by Gaub and Carlson (1997) found that while females met diagnostic criteria for ADHD less often than males, those identified with ADHD had

lower ratings of hyperactivity and problem behaviors, but also had lower intellectual abilities. Later meta-analysis conducted by Gershon (2002) supported the research of Gaub and Carlson (1997), demonstrating fewer symptoms of externalizing behaviors and less hyperactivity in females with ADHD compared to males with the disorder. Greater intellectual impairments in females with ADHD were also identified, though more recent research suggests this difference between males and females with ADHD may be marginal (Biederman et al., 2002). Gershon (2002) also found females to have greater internalizing problems and suggested comorbid disorders of depression and anxiety may be more prevalent for females with ADHD. Biederman et al. (2002) studied Caucasian boys and girls with and without ADHD, assessing various areas of functioning through an assessment battery and clinical interview. Girls with ADHD were more likely inattentive (meeting criteria for ADHD, Predominantly Inattentive Type more often than boys with ADHD). Fewer problem behaviors reported in school and fewer learning disabilities were identified in girls with ADHD compared to boys with ADHD. Biederman et al. (2002) also found that gender differences in most comorbid disorders were largely general differences found even among non-ADHD males and females. Substance use disorders, however, were identified as more prevalent among girls with ADHD.

In a study of adult males and females with ADHD, few gender differences were noted (Biederman et al., 2004). Symptoms of inattention, hyperactivity, and impulsivity all appeared similar for males and females. Gender differences related to ADHD may decline in adulthood. Nonetheless, adult males continue to have significantly higher rates of ADHD than adult females (Kessler et al., 2006).

Etiology

Researchers continue to pursue different explanations for the symptoms defined within ADHD. As with many psychiatric disorders, evidence suggests both biological and environmental factors likely contribute to the development of ADHD. However, Barkley (2003) notes that research currently demonstrates that ADHD is primarily influenced by biological factors. Specifically, family and twin, molecular genetic, and neurobiological studies have all provided strong support for neurological and molecular contributions to symptoms of hyperactivity, reduced inhibition, and inattentiveness.

Family and twin studies. ADHD research in the past decade has provided strong evidence that genetic factors impact the risk of developing the disorder (Stevenson et al., 2005). Biederman et al. (1992) found biological relatives of children with ADHD had a five times greater risk of also having the disorder compared to control group relatives. Biederman et al. (1995) found 84% of parents with ADHD had at least one child with the disorder and that 57% of children of adults with ADHD had the disorder. Even among studies specifically examining relatives of girl ADHD probands, relatives had a five times greater risk of having ADHD compared to controls (Faraone et al., 2000). More recently, research has studied relatives of boys referred for pediatric and psychiatric services. From this sample of patients, relatives of children with ADHD were found to have three times the risk of having ADHD when compared to relatives of non-ADHD children (Biederman et al., 2008). While the majority of ADHD research has focused on Caucasian populations, studies with other ethnic groups have shown similarly high familial correlates of ADHD (e.g. Samuel et al., 1999). As noted by Faraone et al. (2005), while significant findings in family studies are important, this type of research cannot

account for differences between genetic influences and environmental factors in the development of ADHD.

Twin studies provide useful information that allows for a closer comparison of genetic influences by attempting to account for some of the environmental factors that may impact ADHD outcomes. Comparing monozygotic twins (who possess almost all the same genes) with same-sex dizygotic twins (who share approximately 50 percent of genes) allows researchers to evaluate genetic differences between children who presumably develop in very similar environments, sharing home, gender, and age. Higher rates of concurrent disorders in monozygotic twins compared to same-sex dizygotic twins would support the argument for greater genetic influence. Many twin studies have been published demonstrating the strong influence of heritability in risk of ADHD (Waldman & Gizer, 2006). In an early ADHD twin study, Goodman and Stevenson (1989) found 51% of monozygotic twins shared significant levels of hyperactivity while only 33% of dizygotic twins both demonstrated hyperactivity. Sherman, Iacono, and McGue (1997) compared monozygotic and same-sex dizygotic twins on various ratings of ADHD symptoms. Their findings suggested environmental factors provided little influence on the development of ADHD and that monozygotic twins were significantly more likely than dizygotic twins to develop significant symptoms of ADHD. Even when twins exhibited a different category of significant ADHD symptoms (e.g., one twin with symptoms of inattention while the other was predominantly hyperactive-impulsive), monozygotic twins correlations were greater than for the dizygotic twins. Reviewing 18 twin studies, Biederman (2005) found a mean heritability of 77% for ADHD. Reviewing 20 twin studies, Faraone et al. (2005) reported a mean heritability 76% for ADHD. Smith,

Barkley, & Shapiro (2007) provide a similar estimate that at least 78% of the contribution to symptoms of hyperactivity-impulsivity are attributable to genetic factors. When one twin is diagnosed with ADHD, the other is significantly more likely to also develop diagnosable symptoms of the disorder.

Adoption studies provide another valuable method to examine the relationship between environmental and genetic factors contributing to the development of ADHD. Although efforts have been made to account for parent expectations that monozygotic twins will grow up more similarly (e.g. Goodman & Stevenson, 1989), Van den Oord, Boomsma, and Verhulst (1994) suggest twin studies provide limited generalizability to the general population and often fail to account for effects of interaction. Early adoption studies compared adoptive parents of children with ADHD with the children's biological parents. Studies such as Morrison and Stewart (1973) suggested biological parents were more likely to be hyperactive compared to the adoptive parents suggesting biological factors contributed more than environmental factors to the development of hyperactivity in children. Barkley (2003) notes, however, that these studies are retrospective. Later research comparing international, biologically related and unrelated adoptees demonstrated evidence of genetic heritability similar to previous twin studies of attention problems with a genetic heritability of 47% (Van den Oord et al., 1994). More recent research continues to provide evidence that biological relatives of individuals with ADHD are more likely than adoptive relatives to have ADHD or a related disorder (Sprich, Biederman, Crawford, Mundy, & Faraone, 2000). One review (Waldman & Rhee, 2002), summarizing a variety of genetic heritability studies, indicated genetic influences range from 60-90% across studies while non-shared environmental factors

account for only 10-40% of risk. Shared environmental factors provided negligible influence on the development of ADHD.

The neurobiology of ADHD. Neuroscientific research has provided evidence of neurological causes for ADHD symptoms. Nonetheless, the complete neurobiology of ADHD is not currently understood. Early work implicated brain damage and seizures in the development of ADHD symptoms. More recently, researchers have focused on the frontal lobes (Barkley, 2003) and related subcortical regions (Biederman, 2005). The frontal lobe consists of structures in the most anterior region of the brain, including the premotor cortex, the primary motor cortex, Broca's area, medial cortex, and prefrontal cortex. Subcortical regions of interest include the reticular activating system (RAS), the limbic system, and the basal ganglia. Current research suggests that the interplay between cortical and subcortical structures and their associated pathways contributes to an individual's ability to maintain attention and inhibit behaviors. While deficits may appear related to frontal cortical areas, Biederman (2005) notes research cannot currently identify whether deficits are located within prefrontal abnormalities or within other subcortical areas of the brain connected to the prefrontal cortex.

Executive functioning, including planning, inhibition, and self-monitoring abilities, is primarily associated with the prefrontal cortex. The prefrontal cortex interacts directly and indirectly with subcortical brain structures such as the limbic system, RAS, thalamus, and hypothalamus. The RAS has been implicated in cortical arousal or "cortical tone" (Luria, 1973). Connecting to the frontal lobe, the RAS maintains alertness and the fundamental ability to respond to stimuli. The limbic system is also implicated in attention-related functioning, specifically the ability to selectively attend to the most

important stimuli in a given situation (Heilman, Watson, & Valenstein, 2003). The anterior cingulate cortex, which is closely associated with the limbic system, has been found to contribute to the motivational aspects of attention (Blumenfeld, 2002). Biederman (2005) notes that many of the pathways linking frontal lobe structures to subcortical structures are rich in catecholamines and that these pathways are thought to be involved in "the mechanism of action of stimulant medication used to treat this disorder" (p. 1218). The basal ganglia (composed of the globus pallidus, putamen, and caudate nucleus) is one such structure with catecholamine-rich connections to the frontal lobe. Durston (2003) implicated these connections in the regulation of cognitive functioning and motor control. Stimulant medications used in the treatment of ADHD inhibit the dopamine transporter, stimulate the release of catecholamines such as norepinephrine and dopamine, and inhibit catecholamine reuptake (Durston, 2003). Studies on individuals with unilateral infarcts of the caudate nucleus have also suggested that damage to that component of the basal ganglia may cause symptoms of ADHD (Caplan et al., 1990; Mendez, Adam, & Lewandowski, 1989).

Within the frontal lobe, research suggests different regions may be associated with different aspects of attention. Stuss, Binns, Murphy, and Alexander (2002) assessed brain injured individuals with injuries to different frontal regions on tests of attention functioning. Three brain areas were identified as involved in different areas of attention. The superior medial frontal regions of the brain were found associated with an overall readiness to respond to stimuli. The left dorsolateral frontal regions were associated with the brain's ability to set criteria for selective attention. This region appears to affect an individual's ability to respond more quickly to target versus non-target stimuli. Lastly,

the right dorsolateral frontal regions were implicated in the brain's ability to discriminate between target and non-target stimuli at all. Stuss et al. (2002) also noted that neuroimaging studies have suggested the anterior cingulated gyrus may be involved in attention, but that the region's specific role remains unknown.

Evaluating frontal lobe functioning through neuropsychological studies, behavioral inhibition has been shown to be largely evident in children and adults with ADHD (Nigg, 2001). Individuals with ADHD, Predominantly Inattentive Type, however, typically do not demonstrate difficulties in behavioral inhibition and may not be represented in this body of research (Quay, 1997). Other areas of frontal lobe functioning that have been utilized in evaluating ADHD include "nonverbal and verbal working memory, planning, verbal fluency, response perseveration, motor sequencing, sense of time, and other frontal lobe functions." Structural and functioning brain imaging studies have suggested the caudate, putamen, and globus pallidus are also involved in many of the above noted abilities (Biederman, 2005). These findings support the view of ADHD as both a behavioral disorder and a neurocognitive dysfunction.

Recent studies involving neuroimaging techniques have further contributed to the understanding of ADHD as a neurocognitive disorder. Early efforts to differentiate children with ADHD versus controls using psychophysiological measures, such as galvanic skin response, provided inconsistent research findings (Smith et al., 2007). Utilizing techniques such as quantitative electroencephalography (QEEG) and event-related potentials (ERP), single-photon emission computed tomography (SPECT), positron emission tomography (PET), magnetic resonance imaging (MRI), and functional magnetic resonance imaging (fMRI), researchers have demonstrated more reliable

correlations between differences in brain functioning and deficits in attention or behavioral inhibition.

Electroencephalography (EEG) measures electrical currents on the scalp resulting from conduction of currents within the brain. The level of brain electrical activity in different regions can thereby be assessed based on the amount of activity on scalp regions. As reviewed in Loo and Barkley (2005), specific waveforms (or frequency bands) have been identified as indicators of different types of brain activity. Delta waves are associated with sleep, theta waves with an unfocused state of mind, alpha waves with relaxation and closed eyes, and beta waves with active mental engagement. QEEG studies (Monastra et al., 1999; Monastra, Lubar, & Linden, 2001) have demonstrated specific indicators of ADHD based on a slowing of cortical activity within the prefrontal cortex. Tannock (1998) notes different ADHD subgroups may demonstrate different types of cortical underarousal or overarousal. Increased delta and theta wave activity and decreased beta wave activity are associated with ADHD Combined Type; increased alpha wave activity occurs more frequently in those with ADHD, Predominantly Inattentive Type (Loo & Barkley, 2005). ERPs are derived from EEG measurements and represent synchronized, often complex waveform patterns that change in response to study-related stimuli. Studies have suggested that ERP differences may differentiate controls from ADHD subjects with specific ERP differences associated with either inattentive or combined types of ADHD (Johnstone, Barry, & Anderson, 2001). While QEEG provides some insight into neurophysiological indicators of ADHD, it is not specific to any brain structures (Tannock, 1998). Therefore, other neuroimaging techniques have been studied.

SPECT studies use injected radioactive markers to monitor blood flow within the brain. Reviewing SPECT research comparing children with ADHD and control subjects, Hendren, De Backer, and Pandina (2000) found consistent evidence of less cranial blood flow in the basal ganglia and prefrontal cortex in children with ADHD compared to controls. Basal ganglia involvement appeared particularly related to impulsivity.

A similar radioactive marker is used in PET studies to monitor cerebral glucose metabolism. Within adults with childhood-onset ADHD, diminished glucose metabolism has been observed in the frontal lobe, specifically the premotor cortex and superior prefrontal cortex (Zametkin et al., 1990). Child PET studies appear more variable and do not consistently differentiate children with ADHD from controls (Tannock, 1998).

More recent brain imaging techniques have utilized MRI and fMRI technologies to obtain higher resolution images of brain structures. Studies have demonstrated lesser brain volume in children with ADHD compared to controls (Tannock, 1998). Filipek et al. (1997) found smaller volumes in specific brain areas, including the caudate nucleus, right frontal, and anterior-inferior regions. Another MRI study found smaller volumes in overall cortical gray matter, the anterior cingulated cortex, and prefrontal regions (Seidman, Valera et al., 2006). Advances in the use of fMRI have also identified specific brain differences associated with ADHD. Durston et al. (2003) demonstrated differences in striatal activation between children with ADHD and typically developing children, affecting the cortical-striatal-thalamic dopamine pathway. Studies suggest reduced striatal activity contributes to inattention and that stimulant medication appears to partly correct dopamine pathway activity (Swanson et al., 2007). Reviewing other fMRI studies,

Smith et al. (2007) note that abnormal activity has also been identified in the right prefrontal region, basal ganglia, and cerebellum.

Implications of molecular genetics. Having established evidence for the heritability of ADHD, researchers have more recently identified specific genes that may be related to the development of ADHD (Stevenson et al., 2005). Molecular genetic studies have identified a number of candidate genes that may provide a genetic marker indicating an increased risk for ADHD. The most prominently researched genes include those involved in neurotransmitter pathways (Stevenson et al., 2005). Reviewing studies of molecular genetics and factors that appear to contribute to ADHD, Faraone et al. (2005) identified the dopamine D4 receptor (DRD4), dopamine D5 receptor (DRD5), dopamine transporter (DAT1), serotonin transporter (5-HTT), and serotonin receptor (HTR1B) genes, dopamine beta hydroxylase enzyme (DβH), and synaptosome-associated protein (SNAP-25) as most significantly associated with ADHD across studies based on odds-ratio analysis. The number of candidate genes currently under consideration is reasonable given Waldman and Gizer's (2006) conclusion that the genetic influence on ADHD is mostly "comprised of a multitude of susceptibility genes, each contributing only a small magnitude of the overall risk for the disorder" (p. 399). As with many current efforts to identify specific gene influences on the development on psychiatric disorders, research to identify genetic markers for ADHD continued to provide both evidence for and against each candidate gene (Waldman & Gizer, 2006). For example, other suggested candidate genes, such as the dopamine D3 receptor (Muglia, Jain, & Kennedy, 2002), have not found strong support within the available literature (Faraone et al., 2005).

Diagnostic Comorbidity

Citing a number of studies, Barkley (2006b) notes that "as a group, children with ADHD are rated as having more symptoms of disruptive behavior (oppositional and conduct problems), anxiety, depression or dysthymia, and low self-esteem than other nondisabled children" (p. 185). Research suggests a high percentage of children with ADHD have either a mood or anxiety disorder (Biederman, Newcorn, & Sprich, 1991). Adolescents with ADHD demonstrate similar rates of comorbid anxiety and depression compared to children with ADHD but had higher rates of comorbid bipolar disorder (Faraone, Biederman, & Monuteaux, 2002). Studies of adults with ADHD continue to demonstrate significant risk for all DSM-IV mood and anxiety disorders, except obsessive-compulsive disorder (Kessler et al., 2006). Studies suggest nearly 50% of adults with ADHD also have a comorbid anxiety disorder (Biederman, 2005). Children with bipolar disorder have as much as 85% probability of meeting criteria for comorbid ADHD while as many as 22% of children with ADHD have a comorbid bipolar disorder (Singh, DelBello, Kowatch, & Strakowski, 2006). Neurological differences in combination with ADHD may also increase risk for mood disorders. Biederman et al. (1994) found children with ADHD and less common cerebral motor dominance characteristics to have significantly more risk for developing a comorbid major depressive disorder.

Children with ADHD are also at increased risk for the development of antisocial behaviors and drug use by young adulthood (Barkley, Fischer, Smallish, & Fletcher, 2004). Reviewing past studies, Barkley (2003) estimated that between 54% and 67% of children with ADHD meet criteria for a comorbid diagnosis of oppositional defiant

disorder (ODD) and that 20-50% of children and adolescents with ADHD will develop a comorbid conduct disorder. Even among girls, who generally demonstrate lower rates of conduct disorder compared to boys with and without ADHD (Biederman et al., 2002), ADHD presents a significant risk factor for lifetime conduct disorder (Monuteaux, Faraone, Gross, & Biederman, 2007). Recent research suggests the risk of drug use increases for adults with ADHD but the risk of alcohol use does not (Biederman et al., 2008; Kessler et al., 2006; Mannuza, Klein, Bessler, Malloy, & LaPadula, 1998). Not only are individuals with ADHD more likely to become drug dependent, Biederman et al. (2008) found relatives of adolescent ADHD probands were significantly more likely to have a diagnosable psychoactive substance use disorder or drug dependence than relatives of non-ADHD adolescents. While there has been obvious concern that the administration of stimulant medication to treat ADHD may be the basis for an increased risk of later substance abuse, some studies suggest that appropriate treatment of ADHD with such medication, specifically methylphenidate, may actually be protective against late substance use (Szobot & Bukstein, 2008). These findings further support the importance of early and accurate identification of ADHD. Additionally, young adults with ADHD have also been found to have more social problems, including smaller social groups and more difficulty maintaining friendships, and more work-related difficulty, such as maintaining a job (Barkley et al., 2006).

Overall, studies have suggested that individuals with ADHD often present with lower overall intellectual abilities (Frazier, Demaree, & Youngstrom, 2004). In addition to lower demonstrated cognitive ability, learning disabilities in areas of reading, writing, spelling, and arithmetic are commonly recognized in children with ADHD. While ADHD

alone produces significant neuropsychological impairment on assessment tasks, the presence of a comorbid learning disability produces even greater impairments as demonstrated in a study comparing girls with and without ADHD (Seidman, Biederman et al., 2006). Learning disability symptoms represent separate but overlapping or comorbid concerns. Studies have generally found that about 20% of children with ADHD also had a specific learning disability (Tannock, 1998). Mayes, Calhoun, and Crowell (2000) found as many as 70% of children with ADHD at a diagnostic clinic also had a significant learning disorder with writing disorders twice as likely as other types of learning disabilities. A study examining the relationship between processing speed, ADHD, and reading disorders found reduced processing speed a common component in both disorders (Shanahan et al., 2006). Studies following the progress of children with ADHD suggest worse academic performance than non-ADHD peers (Barkley et al., 2006).

Models of ADHD

Barkley's hybrid model. One of the most commonly cited and currently prominent models of ADHD is Barkley's 1997 "hybrid" model of human self-regulation (Barkley, 1997, 2003, 2006a). This model was developed through a merging of previous theories of prefrontal lobe functioning and ADHD with consideration of both neuropsychological and developmental factors. Within this model, Barkley describes behavioral inhibition as a central feature to the development of self-regulation. Behavioral inhibition includes the ability to inhibit a prepotent response (a response to an event when immediate reinforcement is available or the reinforcement is conditioned), the ability to interrupt an occurring or ongoing response to allow executive functions to change the behavior, and

the ability to protect the executive functions from other irrelevant distractions, such as unimportant stimuli (Barkley, 1997, 2003, 2006a). Inhibition of a prepotent response, ongoing response, or response to irrelevant stimuli allows an individual to delay action, allowing executive functions an opportunity to operate. With behavioral inhibition, discrete executive functions are thought to develop: (1) nonverbal working memory, (2) internalization of speech (verbal working memory), (3) self-regulation of affect/motivation/arousal, and (4) reconstitution (planning and generativity). Within Barkley's model of self-regulation, behavioral inhibition acts to protect and regulate the four executive functions.

Barkley (2006a) defines nonverbal working memory, one of the four discrete executive functions, as "the capacity to maintain internally represented information in mind or online that will be used to control a subsequent response" (p. 307). Nonverbal working memory includes the ability to recall past events (recent or distant) relevant to a given situation and reimagine these events as sensory experiences. These remembered sensory events can then be processed and manipulated to help determine future actions. Barkley (2006a) theorizes that through this executive function, humans are able imitate complex behaviors, have hindsight through processing of sensory representations, prepare for future actions, prime motor actions, and gain a sense of self-awareness. Nonverbal working memory is also theorized to contribute to an individual's sense of time (in relation to past events), the increased ability to organize behaviors in the distant future, and the capacity for mental representations to act as rules that govern behavior (Barkley, 2006a).

Barkley (2006a) describes the internalization of speech and verbal working memory as another discrete form of executive functioning. The ability to remember verbal speech is theorized to allow for an internal verbal dialogue. In addition to providing an individual the verbal ability to reflect and describe a given event, self-restraint is also reinforced through self-directed speech. For example, the internalization of verbal guidance by a caregiver may be utilized later in life to regulate behavior. Barkley (2006a) theorizes that self-directed speech also allows for self-questioning about events and contributes to problem-solving through verbal self-dialogue. This aspect of verbal functioning also allows an individual to develop a system of organization for rules and rule-governed behavior. Reading comprehension and moral reasoning are also thought possible due to verbal working memory (Barkley, 2006a).

Self-regulation of affect, motivation, and arousal is another theorized executive function in the 1997 hybrid model. Barkley (2006a) theorized self-directed visual images and speech (derived from nonverbal working memory and internalization of speech) also allowed for internal motivation and emotions to occur. Furthermore, the delay created when an individual delays a prepotent response (a product of behavioral inhibition), emotional reactions are also inhibited. This delay allows the individual to reflect on the events using internal visualizations and speech to regulate affect and decide how to respond. Barkley (2006a) notes, "impulsive prepotent responses are often charged with far more raw emotion than those responses that are emitted after a delay or a period of self-regulation" (p. 313). Therefore, this delay provides the opportunity for social perspective taking and more goal-directed responding.

The fourth and final theorized executive function relates to reconstitution or planning (Barkley, 2006a). This executive function involves the ability to study each unit within a behavioral sequence and then recombine the behavioral units into novel sequences of behavior. For example, an individual is able to break down the functional units of speech and recombine these units into a variety of new sentences. Similarly, analyzing nonverbal sequences allows an individual to develop a flexible, unique repertoire of goal-directed behavior responses (Barkley, 2006a).

The inhibition of behaviors, nonverbal working memory, internalization of speech, self-regulation of affect/motivation/arousal, and reconstitution all contribute to motor control. Executive functioning allows behavioral control to be future-oriented, flexible, and deliberate. When these aspects of behavioral inhibition and executive functioning are working well, motor responses that are irrelevant to a given goal are inhibited, goal-directed responses are executed (often through complex or novel motor sequences), and internal motivation allows the individual to act in a persistent manner. Barkley (2006a) also notes that aspects of nonverbal and verbal working memory allow an individual to reflect on responses and therefore to develop a sensitivity to errors. Working memory, self-regulation, and reconstitution also allow an individual to be interrupted, respond to the interruption, than continue with the goal-directed behavior because the individual is able to retain the motor sequence in memory then reinitiate at the point of interruption. Therefore, Barkley (2006a) describes ADHD as a "disorder of performance" in which an individual with ADHD will "know what they should do or should have done before, but knowing provides little consolation to them, little influence over their behavior, and often much irritation to others" (p. 324). Individuals with ADHD

make preparations and take action much closer to the given event and therefore are often spending their efforts reacting with little forethought.

Barkley's hybrid model accounts for symptoms of ADHD that develop related specifically to the disorders of ADHD, Combined and Predominantly Hyperactive-Impulsive Types. Barkley (2003, 2006a) and others (e.g. Quay, 1997) suggest ADHD, Predominantly Inattentive Type may actually represent a significantly heterogeneous disorder. Children with ADHD, Predominantly Inattentive Type demonstrate less aggression and social difficulties but are at higher risk of developing anxious or depressive symptoms (Barkley, 2003). Based on this heterogeneity, Barkley (2006a) does not attempt to account for children with predominantly inattentive symptoms in his model of self-regulation.

While Barkley's model of human self-regulation is the most comprehensively described theory of the interaction between cognitive executive functioning deficits and ADHD, other models have been developed that contributed to Barkley's understanding of ADHD or that proposed alternative explanations. One such model, the stop-signal paradigm (Logan & Cowan, 1984) theorizes a "race" between two processes, one that initiates a response behavior and another that inhibits the behavior. When the initiation behavior "wins" the race, the behavior occurs and when the inhibition response occurs faster, the behavior is inhibited. Schachar and Logan (1990) describe the outcome of the race to be largely based on probability. Factors affecting this probability include when each of the two processes is started, how often each process is called upon during an activity, and the speed of the process. Therefore, when the inhibitory signal is sent later,

is only called upon sometimes (and therefore less readily triggered), and acts slowly, a child is much less likely to inhibit a behavior during a given activity.

Quay's model and Gray's Behavioral Inhibition System. Somewhat similar to Barkley's model, Quay (1997) proposed a model focusing on the impact of inhibitory dysfunction using Gray's Behavioral Inhibition System (BIS). This system is theorized to respond to conditioned stimuli for both positive and negative punishment. When individuals learn that a behavior leads to an aversive response, the behavioral responses should then be inhibited. Quay (1997) theorized that children with ADHD are less responsive to conditioned stimuli and therefore fail to inhibit behavioral responses. This deficit is specifically linked to the septo-hippocampal circuit (involving the septum, amygdala, hippocampus, and fornix) and connections between the circuit and the frontal cortex. As with Barkley's model, Quay (1997) identifies children with ADHD, Predominantly Inattentive Type as distinctly different from other children with ADHD and therefore does not attempt to account for these children within this model.

Sonuga-Barke's model. Sonuga-Barke (2002) describes a somewhat different model of ADHD in which impulsivity may represent difficulties in modulating inhibition of behavior to meet the demands of the situation. "State regulation deficits" represent the inability or lack of desire to delay behavior to meet the demands of a given task (Sonuga-Barke, 2002; Sonuga-Barke, Houlberg, & Hall, 1994). Impulsivity is therefore theorized to be task dependent and either related to inability or lack of desire to meet inhibition demands. Sonuga-Barke (2002) also suggests children may learn the need to utilize inhibition strategies within a given setting, but may fail to generalize the need to modulate such behavior in a new situation.

The cognitive-energetic model. The cognitive-energetic model has also been applied to understand ADHD (Sergeant, 2000). This model connects an individual's ability to process information, state factors, and management factors with the efficiency with which information is processed and behaviors exhibited. Processing (computational mechanisms) includes the individual's ability to encode information, search for relevant associated factors, make decisions, and organize a motor response. State factors include available effort (energy to meet task demands), arousal (temporal responding to stimuli), and activation (physiological increase in activity; Sergeant, 2000). The third level of the cognitive-energetic model relates to factors commonly associated with executive functioning: planning, monitoring, detection of errors, and correction. Reviewing this model, Sergeant, Geurts, Huijbregts, Scheres, and Oosterlaan (2003) describe the cognitive-energetic model as accounting for both top-down and bottom-up processing. Therefore, ADHD is not defined as a specific deficit in executive functioning, but determined by functioning on the cognitive (processing) and energetic (state) levels. Sergeant et al. (2003) criticize models such as Barkley's hybrid model and the BIS model as only accounting for top-down processing with inhibition as the core deficit. Sergeant et al. (2003) go on to differentiate working memory from executive functioning. Working memory is defined as "selective activation of long-term memory, which requires executive functioning" (p. 587).

Current measures of ADHD

Reviewing the current practice of assessment and diagnosis of ADHD, Smith et al. (2007) note that no "gold standard" exists in which an instrument can provide firm proof that an individual has or does not have the disorder. Instead, diagnosis of ADHD as

defined by the DSM-IV-TR (APA, 2000) relies largely on reported or observed behaviors. In an effort to obtain a maximally accurate picture of a child's symptom presentation, experts have generally recommended a multi-modal approach to assessment of ADHD. For example, more recently, Smith et al. (2007) recommend a test battery involving multiple parent-completed report measures, teacher-completed rating measures, self-report measures complete by the child, and a semistructured interview. Pursuing a higher standard in the assessment of ADHD, a wide variety of clinical tests, procedures, interviews, and behavior rating scales have been utilized.

Parent/teacher questionnaires. Child behavior rating scales are often considered vital in assessing a child's behaviors across settings (e.g., Barkley & Edwards, 2006; Pelham, Fabiano, & Massetti, 2005; Smith et al, 2007). Parents and teachers can be mailed these assessment measures prior to a clinic visit, allowing the clinician the opportunity to review symptoms or areas of difficulty before meeting with the child and parents. These behavior rating scales provide useful information about observed symptoms of ADHD and other behaviors that may suggest other psychological disorders, thereby allowing consideration of differential diagnoses. The use of both broad-band and narrow-band scales is commonly recommended (e.g., Barkley & Edwards, 2006; Smith et al., 2007).

Broad-band scales gather information about a range of psychopathologies and areas of difficulty seen in childhood. Common broadband ratings instruments available for parents to complete include the Behavior Assessment System for Children, Second Edition (BASC-2; Reynolds & Kamphaus, 2004), the Child Behavior Checklist for Ages 6-18 (CBCL/6-18; Achenbach & Rescorla, 2001), the Conners' Parent Rating Scales –

Revised: Long Version (CPRS-R:L; Conners, 1997), and the Personality Inventory for Children, Second Edition (PIC-2; Lachar & Gruber, 2001). These scales differ somewhat in development and scope. For example, CBCL items are empirically derived while BASC-2 scales were originally derived rationally using common behavioral descriptions (Pelham et al., 2005). The CPRS-R:L provides a good initial screening for a variety of childhood psychopathologies but does not provide as much depth as the other measures (Barkley & Edwards, 2006). Teacher rating scales such as the Teacher Report Form (Achenbach & Rescorla, 2001) and teacher version of the BASC-2 and Conners' scales are also available. Narrow-band scales provide the opportunity to collect information about behaviors specifically associated with ADHD. Such scales include the Disruptive Behavior Rating Scale (Erford, 1993), the ADHD Rating Scale-IV: Home Version (DuPaul, Power, Anastopoulous, & Reid, 1998), and Conners' Parent Rating Scales – Revised: Short Version (CPRS-R:S; Conners, 1997).

Structured and semistructured interviews are also commonly recommended in multi-modal assessment of ADHD. Such clinical interviews reduce variability in questions asked and can be used to assess for differential diagnoses. Smith et al. (2007) recommend the use of a comprehensive semistructured interview occurring over three sessions as part of "The ADHD Checkup." Pelham et al. (2005) found the Diagnostic Interview for Children and Adolescents – Revised (Reich, 2000) and Diagnostic Interview Schedule for Children (Schaffer, Fisher, Lucus, Dulcan, & Schwab-Stone, 2000) to be reliable and consistent measures. Barkley and Edwards (2006) recommend that when considering ADHD, a parent interview should include subjects such as demographic information, developmental progress, history, and psychosocial functioning.

Clinical interview with child and teachers may also be considered (Barkley & Edwards, 2006).

Despite strong support for the use of rating scales and structured clinical interviews in ADHD assessment, these methods are not entirely perfect. Even after collecting such data, a clinician may still question the diagnosis of ADHD and seek more information about other alternative explanations for reported behaviors. The accuracy of specific parent ratings may be questionable at times. Research by Chi and Hinshaw (2002) suggests maternal depression is more common in mothers of children with ADHD, possibly contributing to exaggerated parent ratings scores. Smith et al. (2007) acknowledge that teachers' and parents' ratings may vary based on a number of factors that must be considered when considering checklist data. Psychological testing provides more objective data that may be considered in the diagnosis of ADHD. Quinn (2003) found one psychological test, the Intermediate Visual and Auditory Continuous Performance Test (IVA; Sandford & Turner, 1995), to be less prone to the effects of "faking bad" than behavior rating scales. Furthermore, a clinician may require more information to evaluate the severity and possible contributing factors to demonstrated disruptive behaviors. Gordon, Barkley, & Lovett (2006) identify three potential benefits offered by direct psychological testing: clarification of an ADHD diagnosis, considering alternative diagnoses, and assessing for comorbid disorders that also require treatment. Reviewing commonly used tests, Gordon et al. (2006) identified four specific types of testing commonly conducted: "(1) intelligence/achievement tests, (2) general neuropsychological batteries, (3) individual neuropsychological tests, and (4) projective/personality tests" (p. 372).

Intelligence/achievement tests. Intellectual assessment can often be important in understanding an individual's cognitive resources or limitations. Lower scores on tests of intellectual functioning have previously been theorized to differentiate between children with ADHD and non-ADHD controls. Recent studies highlight the disagreement in the field and limited utility of using IQ scores to identify children with ADHD. A meta-analysis conducted by Frazier et al. (2004) found that overall cognitive ability significantly differentiated between ADHD and non-ADHD groups. Nonetheless, the effect size of this difference was relatively small, suggesting limited diagnostic application. More importantly, overall cognitive deficits may be indicative of a variety of other disorders that may interfere with aspects of learning and test performance.

Specific composites/indices within intelligence tests have also been theorized to detect symptoms of ADHD. These too, however, have demonstrated limited utility and sensitivity to ADHD diagnosis when the average or mean skill levels of children with and without ADHD have been compared (Gordon et al., 2006). The Freedom from Distractibility Index (FDI) on the Wechsler Intelligence Scale for Children—Third Edition (WISC-III; Wechsler, 1991) was one combination of subtests designed to measure attention, concentration, and mental control in children (Sattler, 2001). This index consisted of Arithmetic and Digit Span subtests (Wechsler, 1991). Reviewing literature on the utility of the FDI, Gordon et al. (2006) conclude there is no clear indication that poor performance on the index was due to attention-related deficits. Additionally, with the development of the Wechsler Intelligence Scale for Children—Fourth Edition (WISC-IV; Wechsler, 2003a), the FDI was no longer utilized and instead factor-loadings suggested two new composites, the Processing Speed Index and the

Working Memory Index. The Arithmetic subtest was made optional and Digit Span was combined with Letter-Number Sequence to calculate the Working Memory Index (Wechsler, 2003b). The current edition of the Wechsler Intelligence Scale for Children contains four indices theorized to represent specific aspects of intellectual ability. The Verbal Comprehension Index (VCI) consists of the Vocabulary, Comprehension, and Similarities subtests. This index measures the child's verbal knowledge and ability to express information obtained through previous "informal and formal education" (Sattler & Dumont, 2004, p. 21). The Perceptual Reasoning Index (PRI) is derived from the Block Design, Matrix Reasoning, and Picture Concepts subtests. This index examines nonverbal problem solving abilities, novel spatial reasoning, and hypothesis testing with a reduced emphasis on motor speed (Williams, Weiss, & Rolfhus, 2003; Sattler & Dumont, 2004). The Working Memory Index (WMI) is calculated using Digit Span and Letter-Number Sequencing subtests. Working Memory assesses the child's ability to retain verbally administered information in memory, perform simple mental manipulations, and then provide a resulting response. Aspects of executive functioning, including attention and concentration are evaluated by this index (Sattler & Dumont, 2004). Lastly, the Processing Speed Index (PSI) consists of the Coding and Symbol Search subtests. PSI evaluates the child's ability to quickly utilize visual information to complete a task. This construct includes concentration and hand-eye coordination (Sattler & Dumont, 2004).

Neuropsychological batteries and tests. Theories of ADHD and research continue to identify deficits in executive functioning as major characteristic in the disorder (Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008). Attempting to assess for such deficits

to improve the diagnosis of ADHD, some clinicians have used a number of neuropsychological batteries and instruments that are designed to evaluate frontal lobe functioning. The Halstead-Reitan battery is one example of a core battery designed to evaluate a broad range of neuropsychological functioning. Gordon et al. (2006) argue against the use of such batteries, however, due to limited validity, specificity, and sensitivity as to what neuropsychological processes are being assessed. Similarly, reviewing available literature regarding the use of common tests of executive functioning, Gordon et al. (2006) do not recommend the Wisconsin Card Sort Test, Stroop Word-Color Test, Hand Movements Test, Rey-Osterrieth Complex Figure Drawing, and Trail Making Test due to limited accuracy or utility in ADHD diagnosis. Woods, Lovejoy and Ball (2002), however, reviewed the use of a variety of neuropsychological measures of this type for assessing frontal lobe cortical dysfunction in adults with ADHD and suggested that there may yet be diagnostic utility with some of these measures when they are used together, in proper context, and with consideration of intra-individual difference scores.

One common neuropsychological test that has demonstrated utility in the assessment of ADHD among both children and adults is the continuous performance test (CPT). A number of different versions of this type of test exist, but CPTs generally require the examinee to provide some type of response to specific visual and/or auditory stimuli. For example, on the Integrated Visual and Auditory Continuous Performance Test (IVA+Plus; Sandford & Turner, 2004a), individuals must click the mouse button when presented with a target visual or auditory stimulus and ignore non-target stimulus. Scores generated from CPT administration generally include the number of correct

responses, the number of missed target responses (omission errors), and the number of responses to non-target stimuli. Omission errors are believed to be related to attentive vigilance while responses to non-targets are thought to represent impulsivity and inattention. Other CPT data may include speed of responding and amount of mouse movement while completing the test (Sandford & Turner, 2004b). Reviews of CPT literature suggest such tests are particularly sensitive to the core symptoms of ADHD, including inattention and impulsivity, but that the symptoms being evaluated may not be specific to ADHD (Gordon et al., 2006; Riccio, Reynolds, Lowe, & Moore, 2002). Some research has raised concerns about whether the test measures an ecologically valid and important aspect of attention (Barkley, 1991). Nonetheless, studies provide strong evidence that CPT measures are able to differentiate individuals with ADHD from controls when comparing level of vigilance (Corkum & Siegel, 1993). Gordon et al. (2006) describe the instrument as the "only psychological measure that seems to directly assess the core symptoms of the disorder—namely, impulsivity and attention" (p. 377). More recently, CPT tests have also been found to demonstrate the greatest effect size in distinguishing between individuals with ADHD and controls when compared to measures of overall intellectual abilities and other neuropsychological tests (Frazier et al., 2004).

Projective measures. Reviewing literature on the use of projective measures with children with ADHD, Gordon et al. (2006) note the lack of available studies examining predictive validity of such tests. Such measures include the Thematic Apperception Test and the Rorschach inkblot test. While some studies may indicate that children with ADHD are more impulsive, lonely, dependent, avoidant, and socially challenged, Gordon et al. (2006) regard this method of evaluation as neither efficient nor well validated. They

do, however, recommend the use of such measures to rule out thought or emotional disturbances.

Pattern of performance. While level of performance and comparisons with normative data have been the primary method of interpreting psychological tests (Woods, Lovejoy, Stutts, Ball, & Fals-Stewart, 2002), ipsative comparison can control for factors such as an individual's level of intellectual functioning and can identify relationships between abilities (Mayes & Calhoun, 2002; Woods, Lovejoy, Stutts, et al., 2002). In general, pattern analysis between overall intellectual ability and a level of performance in certain neuropsychological domains is supported as a means to identify brain dysfunction (Ivnik et al., 2000). A number of studies have examined the relationship between IQ and specific subtests or configurations of subtests on the WISC-III (e.g., Brinkman, 2005; Mayes & Calhoun, 2002; Mayes, Calhoun, & Crowell, 1998; Nevin, 2003). These studies compared ADHD subjects with non-ADHD controls referred to a diagnostic clinic but results have not been consistent across studies. For example, Brinkman (2005) found no significant differences when comparing groups on IQ minus FDI comparisons while Mayes and Calhoun (2002) suggested that a positive difference between IQ and FDI might accurately predict ADHD in over 70% of cases. Brinkman's (2005) findings, however, demonstrated significant differences between ADHD and non-ADHD subjects when using Extended FDI scores. Similar research has also been conducted comparing IQ to CPT scores, specifically the Gordon Diagnostic System (Brinkman, 2005; Mayes & Calhoun, 2002; Mayes, Calhoun, & Crowell, 2001).

Since these studies were conducted, the WISC-III has been updated. As previously discussed, subtest configurations were reconceptualized for the WISC-IV and

the utility of FDI as a measure of distractibility was reconsidered. Therefore, the WISC-IV composite Index scores have changed to capture new theoretical sub-domains of intellectual functioning. While some studies have suggested that ipsative comparisons have some utility in the diagnosis of ADHD, few studies have been conducted using the current WISC-IV. One recent study (Mayes & Calhoun, 2006), found that children with ADHD tended to score significantly lower on the WISC-IV composites of the WMI and PSI compared to performance on the VCI and PRI. Mean WMI and PSI scores were 21 points lower than VCI and 24 points lower than PRI. This research demonstrated significant ipsative comparisons for ADHD subjects, but a review of literature found no WISC-IV studies that compare patterns of performance for different groups of ADHD versus control subjects, drawn from within the same broad clinical sample. Similarly, no known studies have examined the relationship between WISC-IV performance and CPT scores.

CHAPTER III

HYPOTHESES

The purpose of the present study was to evaluate the utility of examining intra-test and inter-test discrepancies or pattern analyses using WISC-IV composite scores and IVA+Plus quotient scores in the diagnosis of ADHD. The present study sought to replicate and extend prior findings that intra-individual skill discrepancies can be useful in making ADHD diagnoses in children (Mayes and Calhoun, 2006). Since there is particular utility in the factor structure of the WISC-IV for this purpose, this study employed WISC-IV (vs. WISC-III) profile analyses and utilized a newer CPT (IVA+Plus) that is commonly used in clinic settings. The study compared discrepancy or difference (Delta, Δ) scores derived from the WISC-IV and IVA+Plus to attempt to distinguish ADHD and control groups. Specifically, the WISC-IV General Ability Index (GAI), a central measure of a child's reasoning potential (comprised jointly of Verbal Comprehension and Perceptual Reasoning subtest scores), was compared to two WISC-IV cognitive efficiency measures, (a) Working Memory Index (WMI) and (b) Processing Speed Index (WMI) and to two IVA+Plus Quotients, (a) Response Control and (b) Attention. The GAI is "a summary score that is less sensitive to the influence of working memory and processing speed" (Raiford, Weiss, Rolfhus, & Coalson, 2005, p. 2). Raiford et al. (2005) note the GAI does not represent a more valid estimation of intellectual functioning, but interprets intellectual functioning based on three Verbal and three Performance subtests. As noted above, children with ADHD have been found to have significantly lower PSI and WMI scores compared to VCI and PRI (Mayes & Calhoun,

2006). Therefore, the GAI should provide an appropriate central measure of intellectual abilities that are not influenced by cognitive efficiency tasks in the WISC-IV (tasks that might be particularly sensitive to ADHD). Delta scores were derived by subtracting each of four theoretically ADHD-influenced scores (two from the WISC-IV, WMI and PSI, and two from the IVA+Plus, Response Quotient and Attention Quotient) from the WISC-IV GAI in order to derive ipsative discrepancy scores that were expected to be useful in improving diagnostic accuracy. Discrepancy or Delta scores derived as described above were used to compare subjects in the ADHD and control groups. Delta scores were compared to simple level of performance scores in order to evaluate the expected improvements from an ipsative approach to diagnosis. Furthermore, the use of a combination of two or more Delta scores were compared to the use of a single Delta score to determine whether a cluster of these Delta scores reflecting ipsative or intraindividual skill discrepancies might have greater diagnostic utility than a single Delta score.

For the present study, the following hypotheses were offered:

1. The ipsative discrepancy comparison between WISC-IV GAI and WISC-IV WMI will differ significantly between ADHD and control groups; subjects in the ADHD group are hypothesized to have a significantly larger discrepancy between global cognitive ability and working memory skills, as reflected by these two scores.
2. The ipsative discrepancy comparison between WISC-IV GAI and WISC-IV PSI will differ significantly between ADHD and control groups; subjects in the ADHD group are hypothesized to have a significantly larger discrepancy between

global cognitive ability and processing speed skills, as reflected by these two scores.

3. The ipsative discrepancy comparison between WISC-IV GAI and IVA+Plus Full Scale Response Control Quotient will differ significantly between ADHD and control groups; subjects in the ADHD group are hypothesized to have a significantly larger discrepancy between global cognitive ability and inhibition skills, as reflected by these two scores.
4. The ipsative discrepancy comparison between WISC-IV GAI and IVA+Plus Full Scale Attention Quotient will differ significantly between ADHD and control groups; subjects in the ADHD group are hypothesized to have a significantly larger discrepancy between global cognitive ability and sustained attention skills, as reflected by these two scores.
5. Pattern of performance differences between subjects with and without ADHD will be significantly greater than level of performance differences (i.e., comparing mean difference or Delta scores between a global ability measure [GAI] and a measure of cognitive inefficiency [WMI, PSI, Response Quotient, Attention Quotient] in groups of ADHD and non-ADHD subjects will better separate these groups than mean differences between them on the subject's level of performance on these four measures of cognitive efficiency.
6. The combination of two or more different patterns of performance (e.g., WISC-IV GAI minus WISC-IV WMI *and* WISC-IV GAI minus IVA+Plus Full Scale Response Control) will provide better diagnostic accuracy than the use of any single discrepancy comparison (e.g., WISC-IV GAI minus WISC-IV WMI).

CHAPTER IV

METHODS

Participants

This study utilized data collected between 2005 and June 2008 at the Eastern Virginia Medical School Neuropsychology Center. This time span represented the first years that the WISC-IV was used in the clinic. Subject data was collected from all patient records of children between ages 6 and 16 who had completed neuropsychological evaluations at the clinic in which the test measures of interest to this research (WISC-IV [Wechsler, 2003a], IVA+Plus [Sandford & Turner, 2004a], and the CPRS-R:S [Conners, 1997]) were included within the evaluation. All patients' evaluations were completed with both direct involvement and under the direct supervision of a licensed clinical psychologist. Study participants were required to have an IQ over 80 as established by Full Scale IQ (FSIQ) or, when VCI and PRI are significantly discrepant, the highest of either VCI or PRI.

Subjects were separated into two groups consisting of children with ADHD and those without ADHD. All subjects included in the ADHD group met two criteria: (1) must have been diagnosed with ADHD by the licensed clinical psychologist conducting the assessment, and (2) must show an elevation on the Conners' ADHD Index (Conners, 1997) that is 1.5 standard deviations above the norm for the child's age group (65T or greater). Subjects with ADHD were excluded based on the following criteria: were previously diagnosed with a psychiatric disorder (other than ADHD), were previously diagnosed with a neurological disease (other than ADHD), experienced a brain injury

Table 1

Demographic Characteristics of Included Subjects (n = 148)

Variable	All subjects		non-ADHD		ADHD	
	n	%	*n*	%	*n*	%
Gender						
Male	89	60.1	55	65.5	34	53.1
Female	59	39.9	29	34.5	30	46.9
Age						
6	9	6.1	7	8.3	2	3.1
7	20	13.5	12	14.3	8	12.5
8	12	8.1	6	7.1	6	9.4
9	21	14.2	12	14.3	9	14.1
10	16	10.8	9	10.7	7	10.9
11	11	7.4	8	9.5	3	4.7
12	11	7.4	4	4.8	7	10.9
13	16	10.8	6	7.1	10	15.6
14	10	6.8	8	9.5	2	3.1
15	13	8.8	6	7.1	7	10.9
16	9	6.1	6	7.1	3	4.7
Grade						
Kindergarten	3	2.0	2	2.4	1	1.6
1st	14	9.5	10	11.9	4	6.3
2nd	19	12.8	9	10.7	10	15.6
3rd	13	8.8	8	9.5	5	7.8
4th	20	13.5	12	14.3	8	12.5
5th	12	8.1	9	10.7	3	4.7
6th	13	8.8	4	4.8	9	14.1
7th	9	6.1	5	5.9	4	6.3
8th	19	12.8	9	10.7	10	15.6
9th	10	6.8	8	9.5	2	3.1
10th	8	5.4	3	3.6	5	7.8
11th	5	3.4	3	3.6	2	3.1
Home schooled	3	2.0	2	2.4	1	1.6
Race/Ethnicity						
Caucasian/White	124	83.8	77	91.7	47	73.4
African-American/Black	9	6.1	2	2.4	7	10.9
Hispanic/Latino(a)	3	2.0	1	1.2	2	3.1
Asian/Asian American	2	1.4	0	0.0	2	3.1
American Indian	1	0.7	1	1.2	0	0.0
Unknown	9	6.1	3	3.6	6	9.4

resulting in a loss of consciousness, or were diagnosed with a chronic severe medical illness. Control subjects consisted of subjects who met the general criteria for inclusion in this study but who did not previously meet criteria for a diagnosis of ADHD and were not diagnosed with ADHD at the clinic.

Reviewing available assessment records, 340 subjects had completed the measures of interest and were evaluated for this study. After excluding subjects who did not meet overall inclusion criteria, a sample of 148 subjects was divided between those who met inclusion criteria for the ADHD group and those without ADHD (i.e., did not receive diagnoses of ADHD or show elevations on the Conners' rating scales). This process separated these 148 subjects into an ADHD group of 64 subjects and a non-ADHD group of 84 subjects. Within the ADHD group, 36 subjects were diagnosed with ADHD-C and 28 subjects were identified with ADHD-PI following assessment. No subjects were diagnosed as ADHD-PHI.

Subjects in this study (n = 148; see Table 1) had a mean age of 11.11, median age of 10 and modal age of 9 years. Participants ranged from 6 years to 16 years of age. In this total sample, 89 subjects were male and 59 were female. The modal educational level was 4^{th} grade with a range from K to 12^{th} grade, including three students who were being home schooled with no discrete educational level. Seven subjects had been retained in school prior to participation in the study assessment. The sample included 124 Caucasian children, nine African American children, three Latino children, two Asian children, one Native American child, and nine children of unidentified ethnic background. Within the subject sample, 125 children were right-handed, 18 left-handed children, and five subjects with mixed hand dominance.

Table 2

Comorbid Diagnoses of Included Subjects (n = 148)

Variable	All subjects		non-ADHD		ADHD	
	n	%	*n*	%	*n*	%
None	93	62.8	40	47.6	53	82.8
Learning Disability	11	7.4	7	8.3	4	6.3
Written	4	2.7	2	2.4	2	3.1
Math	1	0.7	0	0.0	1	1.6
Reading	1	0.7	1	1.2	0	0.0
NOS/Multiple	5	3.4	4	4.8	1	1.6
Reading LD/Depression	1	0.7	1	1.2	0	0.0
Written LD/Depression	1	0.7	1	1.2	0	0.0
LD NOS/Anxiety	1	0.7	1	1.2	0	0.0
Anxiety	6	4.0	6	7.1	0	0.0
Disruptive Behavior	1	0.7	1	1.2	0	0.0
Mood Disorder NOS	1	0.7	1	1.2	0	0.0
Depressive Disorder	5	3.4	5	5.9	0	0.0
PDD	3	2.0	3	3.4	0	0.0
TBI/Seizures	5	3.4	5	5.9	0	0.0
Medical (non-brain)	5	3.4	1	1.2	4	0.0
Communication Disorder	7	4.7	7	8.3	0	0.0
Feeding Disorder	1	0.7	1	1.2	0	0.0
Complex/Other	5	3.4	5	5.9	0	0.0
Mood/Anxiety Disorder	2	1.3	2	2.4	0	0.0

Note. NOS = Not Otherwise Specified, LD = Learning Disability, PDD = Pervasive Developmental Disorder, TBI = Traumatic Brain Injury, Complex = Complex Or Unclear Symptom Presentation.

The subject pool included 61 children previously diagnosed with ADHD-C or unspecified, five previously diagnosed with ADHD-PI, and none who carried a previous diagnosis of ADHD-PHI. With respect to comorbid diagnoses, 93 subjects carried no previous psychiatric or medical diagnoses (see Table 2). Eight subjects were identified with previously diagnosed learning disabilities, two writing, one math, two reading, and six unspecified or with multiple LDs. Eight subjects had been diagnosed with an anxiety disorder. One subject carried a diagnosis of disruptive behavior disorder. Two subjects were identified with mood disorder NOS. Seven subjects carried depressive disorder diagnoses. Three subjects met criteria for pervasive developmental disorders. Five subjects had a history of TBI or seizures. Five subjects had other medically-related disorders. Seven subjects were diagnosed with communication disorders and one with a feeding disorder. Five subjects had multiple psychiatric diagnoses.

Within the subject pool, 31 children were receiving stimulant medication (e.g., Ritalin, Concerta, Adderall), Vyvanse at the time of evaluation, one subject was receiving clonidine, and three subjects were receiving stimulant and clonidine. Additionally, nine subjects were medicated with allergy medication, 13 subjects were receiving other psychiatric medication not related to ADHD (e.g., fluoxetine, Topomax, Zoloft, lithium bicarbonate) and two subjects were taking medications unrelated to psychiatric disorder or allergies.

Procedures

After obtaining approval from the Institutional Review Board of the Eastern Virginia Medical School (EVMS), certain limited demographic patient data was collected from clinic records for each subject: age, gender, grade level, handedness, ethnicity,

information about medication at time of testing, WISC-IV subtest and composite scores, IVA+Plus scores, Conners' ADHD Index scores, and psychiatric diagnoses before and after assessment at the EVMS Neuropsychology Clinic (see Appendix). All patient information was de-identified, and each participant was randomly assigned an identification number. All assessment data were analyzed using only subject identification numbers with no names or other identifying information. American Psychological Association (2002) ethics guidelines were followed in the collection and use of this collected data.

Instruments

Wechsler Intelligence Scale for Children—Fourth Edition. The WISC-IV (Wechsler, 2003a) is a measure of intellectual ability based on ten core subtests. Five supplemental subtests are also available but were not used as part of the test variables of interest in this research. Index scores of Verbal Comprehension, Perceptual Reasoning, Processing Speed, and Working Memory were derived as based on subtest scores. The WISC-IV was standardized using a normative group of 2,200 children ages 6 to 16 (except the Arithmetic subtest, which was standardized with 1,100 children; Sattler & Dumont, 2004). Age, gender, ethnicity, geographic region, and parental education were considered in developing a representative normative sample based on the March 2000 U.S. Census. Each age group (11 total) consisted of 100 boys and 100 girls (50 boys and 50 girls for the Arithmetic subtest; Sattler & Dumont, 2004). The WISC-IV has demonstrated strong internal consistency reliability with mean composite reliability between .81 and .95 for all 11 age groups. Internal consistency reliability for the 15 subtests ranges from .79 to .90. Standard error of measure of each index ranges from 3.78

and 5.21 (Sattler & Dumont, 2004). Index scores also demonstrated good test-retest reliability, ranging from .79 to .89. PSI demonstrated the lowest test-retest and internal consistency reliability and the greatest standard error of measure (Sattler & Dumont, 2004). WISC-IV validity was evaluated for criterion validity by comparing it to other Wechsler intellectual assessment instruments. Overall, adequate criterion validity was demonstrated. Construct validity was considered and the WISC-IV provided a good measure of overall intellectual ability and related factors (Wechsler, 2003c). Subtest correlations by composite are adequate, ranging from .40 to .64. The Cancellation subtest, a supplemental subtest not used for the purposes of this study, had the weakest correlation with the other 14 subtests (Sattler & Dumont, 2004).

Integrated Visual and Auditory Continuous Performance Test. The IVA+Plus is an intentionally rote computer test in which individuals are asked to respond selectively to visual and auditory stimuli. The test was designed to assist in the diagnosis of ADHD and differentiate between the clinical subtypes of ADHD as identified in the DSM-IV (Sandford & Turner, 2004b). Full scale quotient scores are calculated based on responses to both auditory and visual stimuli. The Full Scale Response Control Quotient evaluates response inhibition, consistent, and compares the subject's reaction speed in the beginning and end of the test. The Full Scale Attention Quotient evaluates response omissions, response speed variability, and average response time. These two full scale quotients are key scales used in evaluating the presence of an attention-related disorder. Quotient scores are reported as standard scores with a mean of 100 and standard deviation of 15 (Sandford & Turner, 2004b).

The IVA+Plus was normed using 1,700 subjects between the ages 6 and 96. Subject groups were divided by gender and age groups with approximately an equal number of 30 males and 30 females in each of the age groups (Sandford & Turner, 2004b). The normative group excluded subjects who were "without identified neurological, current psychological, learning, attentional or self-control problems" (Sandford & Turner, 2004b, p. 92). Standard error was between 3 and 4 standard score units. In the standardization sample, auditory and visual performance were significantly correlated with generally greater impulsivity to auditory stimuli and greater inattention to visual stimuli. Compared to males, females had slower reaction times but fewer impulsive errors (responding to non-target stimuli). The IVA+Plus demonstrated significant test-retest reliability with correlations between .37 and .75 and only small quotient differences (Sandford & Turner, 2004b). Preliminary validation research on the IVA+Plus suggested strong validity with over 90% of subjects correctly classified by the test; less than 10% of cases resulted in false negatives (Sandford & Turner, 2004b).

Conners' Rating Scales—Revised. The Conners' Rating Scales—Revised (CRS-R) were developed to assist in clinical assessment of ADHD in children and adolescents. Observer and self-report rating scales are available. For the purposes of this study, parent/caregiver rating scales of the CRS-R will be considered when assigning subjects to group. The CPRS-R:S consist of 27-items from which the following 4 subscales are derived: Oppositional, Cognitive Problems, Hyperactivity, and ADHD Index (Conners, 1997). The normative sample for the CPRS-R:S included 2426 children and adolescents with 1220 males and 1206 females (Conners, 1997). Within this normative group, 84% of parents identified themselves as Caucasian, 4.3% as African American or Black, 3.8% as

Hispanic, 2.1% as Asian American, and 1.0% as Native American. An additional 4.7% of parents self-identified as Other or declined to provide this information. The CPRS-R:S demonstrates good internal reliability ranging from 0.857 to 0.938 (Conners, 1997). Test-retest reliability ranged from moderate (.47) to high (.88) for the CPRS-R forms (Conners, 1997). Convergent and divergent validity are also demonstrated with high correlations between the long and short forms of the CPRS-R and significant variability between subscores when comparing parent and teacher questionnaires. These parent-teacher differences are supported by the literature, which often finds variable agreement between the two (Conners, 1997).

CHAPTER V

RESULTS

Data Analyses

Addressing Assumptions. All the assumptions of the independent measures two-tailed t-test were met or addressed prior to performing analyses. All variables had equal variances. Frequencies and descriptive statistics were produced to evaluate the presence of any outliers and examine the significance of any missing data. No significant outliers were found for included subjects. Twelve subjects in the non-ADHD group were missing Conners' ADHD Index scores with no other data missing for included subjects. Descriptive statistics showed all analyzed variables to be normally distributed. Descriptive information can be found in Table 3. Variables were evaluated for normality by examining skewness and kurtosis. No variables were found to be skewed or kurtotic.

Addressing Hypotheses. The data were analyzed using independent measures two-tailed t-tests to evaluate the ability of discrepancy scores to discriminate patients in the ADHD and non-ADHD groups.

Hypotheses 1 through 4 were evaluated using independent t-tests to compare subject performances for subjects from ADHD and non-ADHD subject groups on Delta scores between GAI scores and WISC-IV WMI, WISC-IV PSI, IVA Response Control Quotient, and IVA Attention Quotient scores, respectively (see Table 4). Assessing hypothesis 1, contrary to prediction, the ADHD group did not significantly differ in the GAI minus WMI discrepancy ($M = 6.44$, $SD = 10.40$) compared to non-ADHD subjects

Table 3

Descriptive Statistics for All Subjects' Pattern and Level of Performance

	Values						
	n	*M*	*SD*	Skewness	Kurtosis	Min	Max
GAI-WMI	148	5.91	11.295	-.130	.199	-26	35
GAI-PSI	148	10.72	14.97	.077	-.145	-24	55
GAI-RC	148	12.62	20.23	.119	-.055	-45	64
GAI-Attention	148	14.32	24.98	.466	.527	-.47	103
WISC-IV							
FSIQ	148	101.17	12.25	.271	-.506	80	133
GAI	148	104.82	12.67	.309	-.583	78	138
VCI	148	103.24	12.70	.290	-.503	75	132
PRI	148	104.39	12.96	.184	-.228	75	139
WMI	148	98.91	10.60	.518	.006	80	129
PSI	148	94.09	13.38	.154	.025	59	133
IVA							
Full RC	148	92.20	17.19	-.325	-.061	45	136
Full Attention	148	90.50	23.32	-1.021	1.518	0	134
CPRS-R:S	136	66.96	11.01	-.340	-.446	41	90

Note. GAI = General Ability Index, WMI = WISC-IV Working Memory Index, PSI = WISC-IV Processing Speed Index, RC = IVA+Plus Response Control, Attention = IVA+Plus Attention, FSIQ = Full Scale IQ, CPRS-R:S = Conners' Parent Rating Scale – Revised: Short Form.

Table 4

Group Differences on Discrepancy Variables

	ADHD		Non-ADHD				
	M	*SD*	*M*	*SD*	*df*	*t*	*d*
GAI-WMI	6.44	10.40	5.51	11.98	146	-.49	.08
GAI-PSI	11.00	15.81	10.51	14.38	146	-.20	.03
GAI-RC	14.41	20.04	11.26	20.38	146	-.94	.15
GAI-Attention	16.55	25.80	12.62	24.36	146	-.95	.15

Note. GAI = General Ability Index, WMI = WISC-IV Working Memory Index, PSI = WISC-IV Processing Speed Index, RC = IVA+Plus Response Control, Attention = IVA+Plus Attention.

(M = 5.51, SD = 11.980), $t(146)$ = -.49, *ns*, CI = -4.64 to 2.79. Similarly, analysis of the GAI minus PSI discrepancy to evaluate hypothesis 2 indicated no significant difference between the ADHD group (M = 11, SD = 15.81) and the non-ADHD group (M = 10.51, SD = 14.38), $t(146)$ = -.20, *ns*, CI = -5.41 to 4.44. Also for hypothesis 3, no significant difference was found between ADHD (M = 14.41, SD = 20.04) and non-ADHD (M = 11.26, SD = 20.38) subjects for the GAI minus IVA Response Control discrepancy variable, t(146) = -.94, *ns*, CI = -9.78 to 3.49. Analysis of the GAI minus IVA Attention to evaluate hypothesis 4 also indicated no significant differences between subjects in the ADHD group (M = 16.55, SD = 25.80) and non-ADHD group (M = 12.62, SD = 24.36), $t(146)$ = -.95, *ns*, CI = -12.12 to 4.27.

Analysis of level of performance using independent measure two-tailed t-tests also failed to indicate significant between group differences (see Table 5). WISC-IV

scores on the FSIQ, GAI, VCI, PRI, WMI, and PSI were adequately homogeneous. Similarly IVA scores on the Full Response Control and Attention Indices were adequately homogeneous. Nonetheless, no significant differences were indicated between ADHD and non-ADHD groups on measures of performance. Comparing FSIQ level of performance, ADHD subjects (M = 99.73, SD = 11.27) did not differ significantly from those in the non-ADHD group (M = 102.26, SD = 12.90), $t(146) = 1.25$, *ns*, CI = -1.48 to 6.54. Similarly for GAI level of performance, ADHD subjects (M = 103.83, SD = 12.33) did not significantly differ from non-ADHD subjects (M = 105.57, SD = 12.94), $t(146) = .83$, *ns*, CI = -2.42 to 5.90.

Level of performance analyses on WISC-IV component scales also indicated no significant differences between ADHD and non-ADHD subjects. Subjects with ADHD (M = 102.20, SD = 12.57) and those in the non-ADHD group (M = 104.04, SD = 12.81) did not differ significantly when comparing VCI results, $t(146) = .87$, *ns*, CI = -2.34 to 6.00. Similarly, ADHD subjects (M = 103.67, SD = 12.41) and non-ADHD subjects (M = 104.93, SD = 13.41) did not significantly differ on PRI level of performance, $t(146) = .58$, *ns*, CI = -3.00 to 5.52. ADHD subjects (M = 97.39, SD = 9.20) and non-ADHD subjects (M = 100.06, SD = 11.48) did not differ significantly on WMI scores, $t(146) = 1.52$, *ns*, CI = -.79 to 6.13. No significant differences were noted comparing PSI scores for subjects with ADHD (M = 92.83, SD = 12.29) and without (M = 95.06, SD = 14.15), $t(146) = 1.01$, *ns*, CI = -2.16 to 6.62.

Analysis of IVA level of performance scores also did not indicate significant between group differences. ADHD (M = 89.42, SD = 16.32) and non-ADHD (M = 94.31, SD = 17.63) subjects did not significantly differ on IVA Full Scale Response Control,

Table 5

Group Differences on Level of Performance Variables

	ADHD		Non-ADHD				
	M	*SD*	*M*	*SD*	*df*	*t*	*d*
WISC-IV							
FSIQ	99.73	11.27	102.26	12.90	146	1.25	.20
GAI	103.83	12.33	105.57	12.94	146	.83	.13
VCI	102.20	12.57	104.04	12.81	146	.87	.14
PRI	103.67	12.41	104.93	13.41	146	.58	.08
WMI	97.39	9.20	100.06	11.48	146	1.52	.09
PSI	92.83	12.29	95.06	14.15	146	1.01	.16
IVA							
Full RC	89.42	16.32	94.31	17.63	146	1.73	.28
Full Attention	87.28	24.19	92.95	22.46	146	1.47	.25

Note. FSIQ = Full Scale IQ, GAI = General Ability Index, WMI = WISC-IV Working Memory Index, PSI = WISC-IV Processing Speed Index, RC = IVA+Plus Response Control, Attention = IVA+Plus Attention.

$t(146) = 1.73$, *ns*, CI = -.71 to 10.49. Similarly, no significant differences were noted on IVA Full Scale Attention performance for subjects with ADHD ($M = 87.28$, $SD = 24.19$) and those without ($M = 92.95$, $SD = 22.46$), $t(146) = 1.47$, *ns*, CI = -1.94 to 13.29.

Exploratory Analyses

In light of these unexpected non-significant differences between groups on all predicted test measures, additional analyses were explored. The criteria for inclusion in the ADHD group were raised to include only subjects with scores above two standard deviations on the Conners' ADHD Index scale. Using this strict criteria, 47 subjects met criteria for ADHD group inclusion, and there were still no significant differences between groups for GAI minus WMI, $t(129) = -.37$, *ns*, CI = -4.94 to 3.37; GAI minus PSI, $t(129) = -.09$, *ns*, CI = -5.59 to 5.08; GAI minus IVA Response Control, $t(129) = -.91$, *ns*, CI = -10.69 to 3.94; and GAI minus IVA Attention, $t(129) = -.81$, *ns*, CI = -12.31 to 5.17.

Furthermore, even looking at the most reasonably restrictive criteria for inclusion in the ADHD group, evaluating only those with Conners' ADHD Index scores 2 standard deviations above the mean and those diagnosed with ADHD-C or undefined, and in the non-ADHD group, evaluating only those without Conners' ADHD Index scores 1.5 standard deviations above the mean and with no previous diagnoses of ADHD, no significant differences were noted between ADHD and non-ADHD groups. Given these most restrictive criteria, the ADHD group did not significantly differ in the GAI minus WMI discrepancy ($M = 7.50$, $SD = 10.19$) compared to non-ADHD subjects ($M = 4.85$, $SD = 11.80$), $t(68) = -.96$, *ns*, CI = -8.02 to 2.72. Similarly, analysis of the GAI minus PSI discrepancy indicated no significant difference between the ADHD group ($M = 11.17$, SD

= 17.39) and the non-ADHD group (M = 9.60, SD = 14.32), $t(68)$ = -.41, *ns*, CI = -9.14 to 6.00. Also no significant difference was found between ADHD (M = 15.77, SD = 21.57) and non-ADHD (M = 13.93, SD = 17.94) subjects for the GAI minus IVA Response Control discrepancy variable, t(68) = -.39, *ns*, CI = -11.27 to 7.59. Analysis of the GAI minus IVA Attention indicated no significant differences between subjects in the ADHD group (M = 18.13, SD = 28.27) and non-ADHD group (M = 16.52, SD = 22.76), $t(68)$ = -.26, *ns*, CI = -13.78 to 10.56.

CHAPTER VI

CONCLUSIONS

Evaluating Hypotheses

These analyses indicate no significant statistical difference between ADHD and non-ADHD subjects on either the pattern or the level of performance scores of interest in this study. Although non-significant differences may be due to inadequate statistical power, power analyses conducted for this study suggested that there was adequate sample size for these statistical tests given prior reports in the literature of a medium effect size (Cohen, 1992). Hypotheses 1 through 4 were not supported with significant results. Furthermore, given the lack of statistical evidence supporting the use of these discrepancies or level of performance in the identification of ADHD, hypotheses 5 and 6 were not directly evaluated.

These findings are contrary to findings in previous studies evaluating the use of discrepancy analyses to discriminate between ADHD and non-ADHD subjects (e.g., Brinkman, 2005; Mayes and Calhoun, 2002; Mayes and Calhoun, 2006). Mayes and Calhoun (2002) evaluated the use of discrepancy scores related to the WISC-III and Gordon Diagnostic System (GDS) to discriminate between children with and without ADHD. That study found the discrepancy between IQ and the GDS Composite could accurately predict identification of ADHD in 87.8% of cases. Similarly, Mayes and Calhoun (2002) found the discrepancy between IQ and the WISC-III FDI to significantly predict ADHD identification. In a related study, Mayes and Calhoun (2006) found similar results and noted even greater discrepancy scores between ability and efficiency

measures when using WISC-IV indices compared to WISC-III indices for children diagnosed with ADHD. On average, children with ADHD demonstrated a difference of 21 standard score points between VCI and WMI and a mean difference of 24 standard score points between PRI and PSI. In this study, discrepancy scores were much smaller between VCI and WMI ($M = 4.81$, $SD = 11.35$) and between PRI and PSI ($M = 10.84$, $SD = 14.56$) among subjects with ADHD. These two studies contrast notably from the present study that identified no significant differences between groups using WISC-IV and IVA+Plus scores.

Brinkman (2005) identified both level of performance and some pattern of performance (discrepancy scores) differences between children with and without ADHD. That study noted that children with ADHD tended to obtain lower scores on the FDI and GDS scores. However, Brinkman (2005) was not able to replicate the significant difference in discrepancy scores seen between children with and without ADHD for IQ minus FDI. Only by modifying the FDI to include the Coding subtest (currently included in the WISC-IV PSI) was a significant difference noted between ADHD and non-ADHD subjects for discrepancy scores. Brinkman (2005) concluded that the WISC-III Coding subtest might have significantly contributed to the identification of ADHD.

In contrast to these previous studies, the present study identified somewhat elevated discrepancy scores for both ADHD and non-ADHD subjects. Unlike Mayes and Calhoun (2002), who found mean values of two or less for each IQ minus index score discrepancy, the present study identified differences greater than five for subjects without ADHD and greater than six for subjects with ADHD. Delta scores were notably high for both ADHD and non-ADHD subjects in the present study. Some of this difference may

be attributable to the different index used to estimate global intellectual functioning. Previous studies, including Mayes and Calhoun (2002) and Brinkman (2005) relied upon FSIQ, an index that includes all the composite indices (including the index used to calculate the discrepancy such as FDI). The present study attempted to separate the impact of those indices theorized to contribute to ADHD identification by using the GAI, an index not composed of the subtests involved in the WMI and PSI. Within the present study, those identified in the non-ADHD group appeared to have fairly discrepant and lower WMI and PSI scores compared to GAI scores. Theoretically, the GAI minus WMI and GAI minus PSI discrepancy may represent a significantly different construct than noted when FSIQ is utilized. For example, the use of GAI in generating discrepancy scores may have identified greater reasoning and efficiency deficits in all included subjects, even clinically-referred children without identified ADHD. Alternatively, as each subject was referred for testing, the overall sample may simply have demonstrated deficits noted on the WMI and PSI.

Notable differences between subject samples in this study versus others samples reported in prior investigations may also have contributed significantly to these findings (See Table 6). The Mayes and Calhoun (2002) sample of ADHD subjects included children with comorbid behavior, mood, and learning disorders. The present study attempted to contrast ADHD and non-ADHD subjects similarly to other studies, but those subjects classified as ADHD in this study were excluded for the presence of comorbid psychiatric diagnoses, neurological insults, or histories of chronic severe medical illness. Similarly, Brinkman (2005), using only subjects with or without ADHD and not identified comorbid/other psychiatric or neurological diagnoses, did not find group

Table 6

Comparing the Present Study with Mayes and Calhoun (2002)

Factors	Mayes and Calhoun (2006)	Present Study
Intelligence Scale	WISC-III	WISC-IV
Continuous Performance Test	Gordon Diagnostic System	IVA+Plus
Sampling	Clinically-referred children at a neuropsychology clinic	Clinically-referred children at a neuropsychology clinic
ADHD Determined By	Agreement between two raters; based on DSM-IV criteria and parent questionnaire	Agreement between one rater and one parent questionnaire for ADHD group
Inclusion Criteria		
Full Scale IQ	≥80	≥80
Medication	Subjects not medicated with ADHD medication	Subjects often medicated for ADHD symptoms
ADHD types	ADHD-C only	All types of ADHD included
Comorbid Disorders	No psychosis, PDD, bipolar disorder, neurological disorders in either ADHD or comparison groups	No psychiatric or neurological disorder of any kind in ADHD group; no severe psychiatric or neurological disorders in comparison group

differences between subjects with and without ADHD when comparing the same Delta variable (IQ minus FDI) as in the Mayes and Calhoun (2002) research. Thus, it is possible that the types of comorbid problems included in the Mayes and Calhoun (2002) sample compared to the present sample may have significantly impacted the ability to distinguish between ADHD and non-ADHD groups.

Additionally, it was notable that the present study included subjects in the ADHD group receiving medication at the time of testing. Mayes and Calhoun (2002), on the other hand, included only ADHD subjects not on medication at the time of testing. The inclusion of ADHD subjects using ADHD medication may have tampered down any large discrepancies, causing these subjects to generate Delta scores similar to this study's non-ADHD subjects.

Furthermore, studies have varied in their degree of inclusion of multiple ADHD types in subject samples. For example, Mayes and Calhoun (2002) included only children identified with ADHD-C in the ADHD group. The present study, however, included a large number of children with ADHD-PI in the ADHD group. The ADHD-PI subtype has been described as highly heterogeneous, and the presence of these children in this ADHD sample may have influenced the current results in a way that is not yet understood. Children with this ADHD subtype might not show some of the same symptom characteristics as other children with ADHD. In addition to possible differences in how the ADHD groups were operationally defined in these different studies, there may have also been differences in how the non-ADHD groups were comprised. In this study, the non-ADHD subgroup may not have been as clearly devoid of ADHD symptoms as may have been the case in prior studies. A number of subjects in the non-ADHD group within

this research sample carried earlier diagnoses of ADHD that were not supported by the evaluation from which this study data was drawn. It is possible that some residual symptoms of ADHD, even if not clinically definitive, helped blur distinctions between ADHD and non-ADHD subjects in the present sample to a larger degree than in prior research. The strict requirement that ADHD subjects had to meet both criteria of a formal diagnosis and clinically significant elevations on ADHD scales of parent-completed behavior rating scales may have permitted some children with one but not both of these criteria to have been placed in a non-ADHD group. Nonetheless, even when subjects in the ADHD group consisted of only those with ADHD-C or unspecified type were compared with non-ADHD subjects with no known prior diagnosis of ADHD and no Conners' ADHD Index scores above 1.5 standard deviations, no statistical differences were noted between groups.

Nevertheless, in evaluating the principal approach to this present study, which allowed for sufficient power to identify moderate between-group differences, key factors must be considered between the present and past studies. While the Mayes and Calhoun (2006) research may have placed more children with various clinical problems (both ADHD diagnoses and other comorbid problems) into their ADHD group, the present study may have held the ADHD group to stricter criteria for the presence of ADHD while inadvertently permitting the control group to be comprised of more children with sub-clinical ADHD symptoms. Thus, the Mayes and Calhoun (2002) finding may illustrate a difference between an experimental group of (1) both children with ADHD and some other clinical problems, where ADHD is untreated pharmacologically and a control group of (2) children without either ADHD or significant other clinical problems. In contrast,

the present study may illustrate a lack of difference on variables of interest between an experimental group of (1) children with ADHD and no other clinical problems who might be taking medication for those symptoms and a control group of (2) children without ADHD (but with possible tendencies toward ADHD) and with other clinical problems. It is noteworthy that the Mayes and Calhoun (2002) groups are apt to be the most distinct with respect to attention symptoms, while the groups in the present study are the least distinct. While the Mayes and Calhoun (2002) groups are important in early research investigations, the groups comprised for this present study may represent a more realistic and more difficult diagnostic problem in modern clinical settings – that of differentiating children with ADHD from children with other major clinical concerns. Interestingly, in research on continuous performance tests, there have been parallel findings to those reported here. Namely, continuous performance tests have often successfully differentiated children with ADHD from normally developing controls, but they have not reliably distinguished children with ADHD from children with other clinical problems. In a succinct summary of this problem, Riccio et al. (2002), in their review of the research on CPTs, asserted that CPT performance is specific in terms of symptom (inattention), but not in terms of disorder (ADHD). The results of the Mayes and Calhoun (2002) study, when considered along side the results of the present study suggest that something similar could be said about the use of discrepancy scores to differentiate children with ADHD from others. In the final consideration, various sampling considerations in comprising these groups are sufficiently complicated to suggest a need for future research that will parcel out various nuances between subject

characteristics (i.e., degree of comorbidity and presence of medication) in ADHD and non-ADHD groups in this area of research.

In addition to the differences in sample selection and/or subject group assignments discussed above, previous research with ADHD and pattern of performance analysis involving continuous performance testing have utilized the GDS. The present study, on the other hand, utilized a different CPT, the IVA+Plus. The IVA+Plus was developed as a unique CPT with both auditory and visual stimuli. Additionally, the IVA+Plus is a relatively new CPT and has not benefited from the long history of research seen with older systems such as the GDS. As a result, the aspects of executive functioning and ADHD the IVA+Plus posits to measure may be significantly different from that measured by the GDS. However, since the present study failed to find diagnostic significance for discrepancies between global intelligence and mental efficiency measures within the WISC-IV as well as between global intelligence and measures within the IVA+Plus, it is unlikely that differences between the IVA+Plus and the GDS are the primary explanation for different findings between the present study and past research findings (e.g., Brinkman, 2005; Mayes and Calhoun, 2002).

Limitations

The limitations of the present study were largely related to convenience sampling inherent in obtaining subjects all referred to and tested at one neuropsychological testing clinic. The study sample was regionally constricted and demographically limited. Furthermore, ADHD and non-ADHD group assignment relied upon one measure (the CPRS-R:S) and the clinical judgment of one clinical psychologist. Scores on the Conners' ADHD Index, examined as part of the evaluation being received, likely

contributed direction to the clinical determination of the psychologist. As noted by Mayes and Calhoun (2002), the inclusion of a second evaluator, specific utilization of the DSM-IV criteria, and the requirement for complete agreement between the two raters for inclusion in the ADHD group may have generated a more clearly defined ADHD and non-ADHD differentiation in subjects. Therefore, it is unclear how results may have been impacted by a broader and more diverse sample with different inclusion criteria.

In the present study, the best predictor of ADHD and non-ADHD group identification was ultimately the results of the Conners' ADHD Index, rather than the clinical diagnosis. The ADHD group inclusion criteria included a score on the ADHD Index of the CPRS-R:S that was 1.5 standard deviations above the mean. Potentially, over reliance on one measure of ADHD as a major means of differentiating groups may have resulted in a distinctly unique group assignment when compared with previous discrepancy analysis research. This factor is particularly important to consider given the, at times, biased nature of parent-completed behavioral report measures.

The criteria for identification of the ADHD and non-ADHD groups are likely the most important factors in determining whether level of performance and discrepancy scores significantly predict group membership. The difficulty in defining criteria for inclusion in the ADHD group for the purposes of research mirrors the difficulties seen in the clinical diagnosis of the disorder. In their study, noting the utility of WISC-III discrepancies in the identification of subjects with ADHD, Mayes and Calhoun (2002) relied on multiple clinicians' diagnoses, evaluation directly based on DSM-IV criteria, and allowed for children with a number of psychiatric disorders in determining inclusion in their ADHD group. Given the differences between their study outcomes, the variable

findings of the Brinkman (2005) study, and the present study results, the importance of carefully structuring the criteria for ADHD and non-ADHD group criteria is highly evident.

Future Directions for Research

Future research will be required to continue evaluating the utility of discrepancy analyses in the identification of children with and without ADHD. It will be important for future studies to carefully define ADHD and non-ADHD groups. Group inclusion criteria must be thoughtfully considered, as group differences directly impact what is being evaluated through discrepancy analysis. While the goal is to measure directly for the effects of ADHD, other between group differences may have a greater impact on any significant discrepancies seen in past research. Additionally, the impact of comorbid diagnoses versus solely identified ADHD may significantly impact the utility of discrepancy analyses. The addition of a control group with no clinical diagnoses may also be helpful in identifying potential differences between ADHD and non-ADHD subjects related to the discrepancy variables. Furthermore, it will be equally important to continue evaluating the use of discrepancy analyses within more tightly controlled, research-specific, clinical settings versus more general, clinical settings. Evaluator differences and the impact of specific criteria used to identify ADHD and non-ADHD groupings will also be important to consider and control for as needed.

REFERENCES

Achenbach, T. M., & Rescorla, L. A. (2001). *Manual for the ASEBA school-age forms and profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families.

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

American Psychological Association. (2002). Ethical principles of psychologists and code of conduct. *American Psychologist, 57*, 1060-1073.

Barkley, R. A. (1991). The ecological validity of laboratory and analogue assessment methods of ADHD symptoms. *Journal of Abnormal Child Psychology, 19*, 149-178.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*, 65-94.

Barkley, R. A. (2003). Attention-deficit/hyperactivity disorder. In. E. J. Mash & R. A. Barkley (Eds.), *Child psychopathology* (2nd ed., pp. 75-143). New York: Guilford Press.

Barkley, R. A. (2006a). A theory of ADHD. In R. A. Barkley (Ed.), *Attention-deficit/hyperactivity disorder: A handbook for diagnosis and treatment* (3rd ed., pp. 184-218). New York: Guilford Press.

Barkley, R. A. (2006b). Comorbid disorders, social and family adjustment, and subtyping. In R. A. Barkley (Ed.), *Attention-deficit/hyperactivity disorder: A*

handbook for diagnosis and treatment (3rd ed., pp. 184-218). New York: Guilford Press.

Barkley, R. A. (2006c). Primary symptoms, diagnosis criteria, prevalence, and gender differences. In R. A. Barkley (Ed.), *Attention-deficit/hyperactivity disorder: A handbook for diagnosis and treatment* (3rd ed., pp. 76-121). New York: Guilford Press.

Barkley, R. A., & Edwards, G. (2006). Diagnostic interview, behavior rating scales, and the medical examination. In R. A. Barkley (Ed.), *Attention-deficit/hyperactivity disorder: A handbook for diagnosis and treatment* (3rd ed., pp. 337-368). New York: Guilford Press.

Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2004). Young adult follow-up of hyperactive children: Antisocial activities and drug use. *Journal of the American Academy of Child and Adolescent Psychiatry, 45*, 195-211.

Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2006). Young adult outcome of hyperactive children: Adaptive functioning in major life activities. *Journal of the American Academy of Child and Adolescent Psychiatry, 45*, 192-202.

Biederman, J. (2005). Attention-deficit/hyperactivity disorder: A selective overview. *Biological Psychiatry, 57*, 1215-1220.

Biederman, J., Faraone, S., Keenan, K., Benjamin, J., Krifcher, B., Moore, C., et al. (1992). Further evidence for family-genetic risk factors in attention deficit hyperactivity disorder: Patterns of comorbidity in probands and relatives of psychiatrically and pediatrically referred samples. *Archives of General Psychiatry, 49*, 728-738.

Biederman, J., Faraone, S. V., Mick, E., Spencer, T., Wilens, T., Kiely, K., et al. (1995). High risk for attention deficit hyperactivity disorder among children of parents with childhood onset of the disorder: A pilot study. *American Journal of Psychiatry, 152*, 431-435.

Biederman, J., Faraone, S. V., Monuteaux, M. C., Bober, M., & Cadogen, E. (2004). Gender effects on attention-deficit/hyperactivity disorder in adults, revisited. *Biological Psychiatry, 55*, 692-700.

Biederman, J., Lapey, K. A., Milberger, S., Faraone, S. V., Reed, E. D., & Seidman, L. J. (1994). Motor preference, major depression and psychosocial dysfunction among children with attention deficit hyperactivity disorder. *Journal of Psychiatric Research, 28*, 171-184.

Biederman, J., Mick, E., Faraone, S. V., Braaten, E., Doyle, A., Spencer, T., et al. (2002). Influence of gender on attention deficit hyperactivity disorder in children referred to a psychiatric clinic. *American Journal of Psychiatry, 159*, 36-42.

Biederman, J., Newcorn, J., & Sprich, S. (1991). Comorbidity of attention deficit hyperactivity disorder with conduct, depressive, anxiety, and other disorders. *American Journal of Psychiatry, 148*, 564-577.

Biederman, J., Petty, C. R., Wilen, T. E., Fraire, M. G., Purcell, C. A., Mick, E., et al. (2008). Familial risk analyses of attention deficit hyperactivity disorder and substance use disorders. *American Journal of Psychiatry, 165*, 107-115.

Blumenfeld, H. (2002). *Neuroanatomy through clinical cases*. Sunderland, MA: Sinauer Associates, Inc.

Breton, J., Bergeron, L., Valla, J., Berthiaume, C., Gaudet, N., Lambert, J., et al. (1999). Quebec child mental health survey: Prevalence of DSM-III-R mental health disorders. *Journal of Child Psychology and Psychiatry, 40*, 375-384.

Brinkman, M. L. (2005). *A comparison of two methods of inference in the use of neuropsychological testing to diagnose childhood ADHD.* Unpublished doctoral dissertation, Virginia Consortium Program in Clinical Psychology, Virginia Beach, VA.

Caplan, L. R., Schmahmann, J. D., Kase, C. S., Feldmann, E., Baquis, G., Greenberg, J. P., et al. (1990). Caudate infarcts. *Archives of Neurology, 47*, 133-143.

Chi, T. C., & Hinshaw, S. P. (2002). Mother–child relationships of children with ADHD: The role of maternal depressive symptoms and depression-related distortions. *Journal of Abnormal Child Psychology, 30*, 387-400.

Cohen, J. (1992). A power primer. *Quantitative Methods in Psychology*, 112, 155-159.

Conners, C. K. (1997). *Conners' Rating Scales — Revised: User's manual.* North Tonawanda, NY: Multi-Health Systems, Inc.

Corkum, P. V., & Siegel, L. S. (1993). Is the continuous performance task a valuable research tool for use with children with attention-deficit-hyperactivity disorder? *Journal of Child Psychology and Psychiatry, 34*, 1217-1239.

DuPaul, G. J., Power, T. J., Anastopoulous, A. D., & Reid, R. (1998). *ADHD rating scale – IV: Checklist, norms, and clinical interpretations.* New York: Guilford Press.

Durston, S. (2003). A review of the biological bases of ADHD: What have we learned from imaging studies? *Mental Retardation and Developmental Disabilities Research Review, 9*, 184-195.

Durston, S., Tottenham, N. T., Thomas, K. M., Davidson, M. C., Eigsti, I., Yang, Y., et al. (2003). Differential patterns of striatal activation in young children with and without ADHD. *Biological Psychiatry, 53*, 871-878.

Erford, B. T. (1993). *Disruptive Behavior Rating Scale*. East Aurora, NY: Slosson.

Faraone, S. V., Biederman, J., Mick, E., Williamson, S., Wilens, T., Spencer, T., et al. (2000). Family study of girls with attention deficit hyperactivity disorder. *American Journal of Psychiatry, 157*, 1077-1083.

Faraone, S., Biederman, J., & Monuteaux, M. C. (2002). Further evidence for the diagnostic continuity between child and adolescent ADHD. *Journal of Attention Disorders, 6*, 5-13.

Faraone, S. V., Perlis, R. H., Doyle, A. E., Smoller, J. W., Goralnick, J. J., Holmgren, M. A., et al. (2005). Molecular genetics of attention-deficit/hyperactivity disorder. *Biological Psychiatry, 57*, 1313-1323.

Faraone, S. V., Sergeant, J., Gillberg, C., & Biederman, J. (2003). The worldwide prevalence of ADHD: Is it an American condition? *World Psychiatry, 2*, 104-113.

Filipek, P. A., Semrud-Clikeman, M., Steingard, R. J., Renshaw, P. F., Kennedy, D. N., & Biederman, J. (1997). Volumetric MRI analysis comparing subjects having attention-deficit hyperactivity disorder with normal controls. *Neurology, 48*, 589-601.

Frazier, T. W., Demaree, H. A., & Youngstrom, E. A. (2004). Meta-analysis of intellectual and neuropsychological test performance in attention-deficit/hyperactivity disorder. *Neuropsychology, 18*, 543-555.

Gaub, M., & Carlson, C. L. (1997). Gender differences in ADHD: A meta-analysis and critical review. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 1036-1045.

Gershon, J. (2002). A meta-analytic review of gender differences in ADHD. *Journal of Attention Disorders, 5*, 143-154.

Goodman, R., & Stevenson, J. (1989). A twin study of hyperactivity—II. The aetiological role of genes, family relationships and perinatal adversity. *Journal of Child Psychology and Psychiatry, 30*, 691-709.

Gordon, M., Barkley, R. A., & Lovett, B. J. (2006). Tests and observational measures. In R. A. Barkley (Ed.), *Attention-deficit/hyperactivity disorder: A handbook for diagnosis and treatment* (3rd ed., pp. 369-388). New York: Guilford Press.

Heilman, K. M., Watson, R. T., & Valenstein, E. (2003). Neglect and related disorders. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (4th ed., pp. 296-346). New York: Oxford University Press, Inc.

Hendren, R. L., De Backer, I., & Pandina, G. J. (2000). Review of neuroimaging studies of child and adolescent psychiatric disorders from the past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 815-828.

Ivnik, R. J., Smith, G. E., Petersen, R. C., Boeve, B. F., Kokmen, E., & Tangalos, E. G. (2000). Diagnostic accuracy of four approaches to interpreting neuropsychological test data. *Neuropsychology, 14*, 163-177.

Johnstone, S. J., Barry, R. J., & Anderson, J. W. (2001). Topographic distribution and developmental timecourse of auditory event-related potentials in two subtypes of

attention-deficit hyperactivity disorder. *International Journal of Psychophysiology, 42*, 73-94.

Kessler, R. C., Adler, L., Barkley, R., Biederman, J., Conners, C. K., Demler, O., et al. (2006). The prevalence and correlates of adult ADHD in the United States: Results from the National Comorbidity Survey Replication. *American Journal of Psychiatry, 163*, 716-723.

Lachar, D., & Gruber, C. P. (2001). *Personality Inventory for Children, Second Edition.* Los Angeles: Western Psychological Services.

LeFever, G. B., Butterfoss, F. D., & Vislocky, N. F. (1999). High prevalence of attention deficit hyperactivity disorder: Catalyst for development of a school health coalition. *Family Community Health, 22*, 38-49.

LeFever, G. B., Dawson, K. V., & Morrow, A. L. (1999). The extent of drug therapy for attention deficit-hyperactivity disorder among children in public schools. *American Journal of Public Health, 89*, 1359-1364.

Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychological Review,* 295-327.

Loo, S. K., & Barkley, R. A. (2005). Clinical utility of EEG in attention deficit hyperactivity disorder. *Applied neuropsychology, 12*, 64-76.

Luria, A. R. (1973). *The working brain: An introduction to neuropsychology.* (B. Haigh, Trans.). New York: Basic Books.

Mannuza, S., Klein, R. G., Bessler, A., Malloy, P., & LaPadula, M (1998). Adult psychiatric status of hyperactive boys grown up. *American Journal of Psychiatry, 155*, 493-498.

Mayes, S. D., & Calhoun, S. L. (2002). The Gordon Diagnostic System and WISC-III Freedom from Distractibility Index: Validity in identifying clinic-referred children with and without ADHD. *Psychological Reports, 91*, 575-587.

Mayes, S. D., & Calhoun, S. L. (2006). WISC-IV and WISC-III profiles in children with ADHD. *Journal of Attention Disorders, 9*, 486-493.

Mayes, S. D., Calhoun, S. L., & Crowell, E. W. (1998). WISC-III freedom from distractibility as a measure of attention in children with and without attention deficit hyperactivity disorder. *Journal of Attention Disorders, 2*, 217-227.

Mayes, S. D., Calhoun, S. L., & Crowell, E. W. (2000). Learning disabilities and ADHD: Overlapping spectrum disorders. *Journal of Learning Disabilities, 33*, 417-424.

Mayes, S. D., Calhoun, S. L., & Crowell, E. W. (2001). Clinical validity and interpretation of the Gordon Diagnostic System in ADHD assessment. *Child Neuropsychology, 7*, 32-41.

Mendez, M. F., Adam, N. L., & Lewandowski, K. S. (1989). Neurobehavioral changes associated with caudate lesions. *Neurology, 39*, 349-354.

Monastra, V. J., Lubar, J. F., & Linden, M. (2001). The development of a quantitative electroencephalographic scanning process for attention deficit-hyperactivity disorder: reliability and validity studies. *Neuropsychology,15*, 136-144.

Monastra, V. J., Lubar, J. F., Linden, M., VanDeusen, P., Green, G., Wing, W., et al. (1999). Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: an initial validation study. *Neuropsychology, 13*, 424-433.

Monuteaux, M. C., Faraone, S. V., Gross, L. M., & Biederman, J. (2007). Predictors, clinical characteristics, and outcome of conduct disorder in girls with attention-deficit/hyperactivity disorder: A longitudinal study. *Psychological Medicine, 37*, 1731-1741.

Morrison, J. R., & Stewart, M. A. (1973). The psychiatric status of the legal families of adopted hyperactive children. *Archives of General Psychiatry, 28*, 888–891.

Muglia, P., Jain, U., & Kennedy, J. L. (2002). A transmission disequilibrium test of the Ser9/Gly dopamine D3 receptor gene polymorphism in adult attention-deficit hyperactivity disorder. *Behaviour Brain Research, 130*, 91-95.

Nevin, S. T. (2003). Diagnostic efficiency of a battery of cognitive and activity measures for classification of childhood ADHD. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 64*, 427.

Nigg, J. T. (2001). Is ADHD a disinhibitory disorder? *Psychological Bulletin, 127*, 571-598.

Pelham, W. E., Fabiano, G. A., & Massetti, G. M. (2005). Evidenced-based assessment of attention deficit hyperactivity disorder in children and adolescents. *Journal of Clinical Child and Adolescent Psychology, 34*, 449-176.

Quay, H. C. (1997). Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 25*, 7-13.

Quinn, C. A. (2003). Detection of malingering in assessment of adult ADHD. *Archives of Clinical Neuropsychology, 18*, 379-395.

Raiford, S. E., Weiss, L. G., Rolfhus, E., & Coalson, D. (2005). *General Ability Index.* Retrieved April 7, 2005, from http://harcourtassessment.com/hai/Images/pdf/wisciv/WISCIVTechReport4.pdf

Reynolds, C. R., & Kamphaus, R. W. (2004). *Behavior Assessment System for Children, Second Edition.* Circle Pines, MN: American Guidance Service.

Reich, W. (2000). Diagnostic Interview for Children and Adolescents (DICA). *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 59-66.

Riccio, C. A., Reynolds, C. R., Lowe, P., & Moore, J. J. (2002). The continuous performance test: A window on the neural substrates for attention?. *Archives of Clinical Neuropsychology, 17*, 235-272.

Samuel, V. J., George, P., Thornell, A., Curtis, S., Taylor, A., Brome, D., et al. (1999). A pilot controlled family study of DSM-III-R and DSM-IV ADHD in African-American children. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*, 34-39.

Sandford, J. A., & Turner, A. (1995). *Intermediate Visual and Auditory Continuous Performance Test.* Richmond, VA: Braintrain, Inc.

Sandford, J. A., & Turner, A. (2004a). *IVA+Plus: Integrated Visual and Auditory Continuous Performance Test: Administration manual.* Richmond, VA: Braintrain, Inc.

Sandford, J. A., & Turner, A. (2004b). *IVA+Plus: Integrated Visual and Auditory Continuous Performance Test: Interpretation manual.* Richmond, VA: Braintrain, Inc.

Sattler, J. M. (2001). *Assessment of children: Cognitive applications* (4th ed.). San Diego, CA: Jerome M. Sattler, Publisher, Inc.

Sattler, J. M., & Dumont, R. (2004). *Assessment of children: WISC-IV and WPPSI-III supplement.* San Diego, CA: Jerome M. Sattler, Publisher, Inc.

Schachar, R., & Logan, G. D. (1990). Impulsivity and inhibitory control in normal development and childhood psychopathology. *Developmental Psychology, 26,* 710-720.

Shaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwab-Stone, M. E. (2000). NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 28-38.

Seidman, L. J., Biederman, J., Valera, E. M., Monuteaux, M. C., Doyle, A .E., & Faraone, S. V. (2006). Neuropsychological functioning in girls with attention-deficit/hyperactivity disorder with and without learning disabilities. *Neuropsychology, 20,* 166-177.

Seidman, L. J., Valera, E. M., Makris, N., Monuteaux, M .C., Boriel, D. L., Kelkar, K., et al. (2006). Dorsolateral prefrontal and anterior cingulate cortex volumetric abnormalities in adults with attention-deficit/hyperactivity disorder identified by magnetic resonance imaging. *Biological Psychiatry, 60,* 1071-1080.

Sergeant, J. (2000). The cognitive-energetic model: An empirical approach to attention-deficit hyperactivity disorder. *Neuroscience and Biobehavioral Reviews, 24,* 7-12.

Sergeant, J. A., Geurts, H., Huijbregts, S., Scheres, A., & Oosterlaan, J. (2003). The top and bottom of ADHD: A neuropsychological perspective. *Neuroscience and Biobehavioral Reviews, 27*, 583-592.

Shanahan, M. A., Pennington, B. F., Yerys, B. E., Scott, A., Boada, R., Willcutt, E. G., et al. (2006). Processing speed deficits in attention deficit/hyperactivity disorder and reading disability. *Journal of Abnormal Child Psychology, 34*, 585-602.

Sherman, D. K., Iacono, W. G., & McGue, M. K. (1997). Attention-deficit hyperactivity disorder dimensions: A twin study of inattention and impulsivity-hyperactivity. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 745-753.

Singh, M. K., DelBello, M. P., Kowatch, R. A., & Strakowski, S. M. (2006). Co-occurrence of bipolar and attention-deficit hyperactivity disorders in children. *Bipolar Disorders, 8*, 710-720.

Smith, B. H., Barkley, R. A., & Shapiro, C. J. (2007). Attention-deficit/hyperactivity disorder. In E. J. Mash & R. A. Barkley (Eds.), *Assessment of childhood disorders* (4th ed., pp. 53-131). New York: Guilford Press.

Sonuga-Burke, E. J. S. (2002). Interval length and time-use by children with AD/HD: A comparison of four models. *Journal of Abnormal Child Psychology, 30*, 257-264.

Sonuga-Burke, E. J. S., Houlberg, K., & Hall, M. (1994). When is "impulsiveness" not impulsive? The case of hyperactive children's cognitive style. *Journal of Child Psychology and Psychiatry, 35*, 1247-1253.

Sonuga-Barke, E. J. S., Sergeant, J. A., Nigg, J., & Willcutt, E. (2008). Executive dysfunction and delay aversion in attention deficit hyperactivity disorder:

Nosologic and diagnostic implications. *Child and Adolescent Psychiatric Clinics of North America, 17*, 367-384.

Sprich, S., Biederman, J., Crawford, M. H., Mundy, E., & Faraone, S. V. (2000). Adoptive and biological families of children and adolescents with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 1432-1437.

Stevenson, J., Asherson, P., Hay, D., Levy, F., Swanson, J., Thapar, A., et al. (2005). Characterizing the ADHD phenotype for genetic studies. *Developmental Science, 8* (2), 115-121.

Stuss, D. T., Binns, M. A., Murphy, K. J., & Alexander, M. P. (2002). Dissociations within the anterior attentional system: Effects of task complexity and irrelevant information on reaction time speed and accuracy. *Neuropsychology, 16*, 500-513.

Swanson, J. M., Kinsbourne, M., Nigg, J., Lanphear, B., Stefanatos, G. A., Volkow, N., et al. (2007). Etiologic subtypes of attention-deficit/hyperactivity disorder: Brain imaging, molecular genetic and environmental factors and the dopamine hypothesis. *Neuropsychology Review, 17*, 39-59.

Szatmari, P., Offord, D. R., & Boyle, M. H. (1989). Ontario Child Health Study: Prevalence of attention deficit disorder with hyperactivity. *Journal of Child Psychology and Psychiatry, 30*, 219-230.

Szobot, C. M., & Bukstein, O. (2008). Attention deficit hyperactivity disorder and substance use disorders. *Child and Adolescent Psychiatric Clinics of North America,* 17, 309-323.

Tannock, R. (1998). Attention deficit hyperactivity disorder: Advances in cognitive, neurobiological, and genetic research. *Journal of Child Psychology and Psychiatry, 39*, 65-99.

Van den Oord, E. J. C. G., Boomsma, D. I., & Verhulst, F. C. (1994). A study of problem behaviors in 10- to 15-year old biologically related and unrelated international adoptees. *Behavior Genetics, 24*, 193-205.

Waldman, I. D., & Gizer, I. R. (2006). The genetics of attention deficit hyperactivity disorder. *Clinical Psychology Review, 26*, 396-432.

Waldman, I. D., & Rhee, S. H. (2002). Behavioural and molecular genetic studies. In S. Sandberg (Ed.), *Hyperactivity and attention disorders of childhood* (2nd ed., pp. 290–335). New York: Cambridge University Press.

Wechsler, D. (1991). *Wechsler Intelligence Scale for Children—Third Edition.* San Antonio, TX: The Psychological Corporation.

Wechsler, D. (2003a). *Wechsler Intelligence Scale for Children—Fourth Edition.* San Antonio, TX: The Psychological Corporation.

Wechsler, D. (2003b). *WISC-IV administration and scoring manual.* San Antonio, TX: The Psychological Corporation.

Williams, P. E., Weiss, L. G., & Rolfhus, E. (2003). *WISC-IV technical report #1: Theoretical model and test blueprint.* Retrieved February 23, 2008 from http://harcourtassessment.com/hai/Images/pdf/wisciv/WISCIVTechReport1.pdf

Woods, S. P., Lovejoy, D. W., Stutts, M. L., Ball, J. D., & Fals-Stewart, W. (2002). Comparative efficiency of a discrepancy analysis for the classification of

attention-deficit/hyperactivity disorder in adults. *Archives of Clinical Neuropsychology, 17*, 351-369.

Woods, S. P., Lovejoy, D. & Ball, J. D. (2002). Neuropsychological evaluation of adults with ADHD: A comprehensive review of initial studies. *The Clinical Neuropsychologist, 16*, 12-34.

Zametkin, A. J., Nordahl, T. E., Gross, M., King, A. C., Semple, W. E., Rumsey, J., et al. (1990). Cerebral glucose metabolism in adults with hyperactivity of childhood onset. *The New England Journal of Medicine, 323*, 1361-1366.

APPENDIX

Sample Data Collection Worksheet

Subject ID#	001	002	003
DOB			
DOE			
Gender			
Highest Grade			
Ethnicity			
Handedness			
WISC-IV FSIQ			
WISC-IV GAI			
WISC-IV VCI			
WISC-IV PRI			
WISC-IV WMI			
WISC-IV PSI			
WISC-IV Similarities			
WISC-IV Vocabulary			
WISC-IV Comprehension			
WISC-IV Block Design			
WISC-IV Picture Concepts			
WISC-IV Matrix Reasoning			
WISC-IV Digits Span			
WISC-IV L-N S			
WISC-IV Coding			
WISC-IV Symbol Search			
IVA+Plus Full Scale RC			
IVA+Plus Full Scale A			
Diagnosis pre-assessment			
Diagnosis post-assessment			
Connors ADHD Index			

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