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A Study of the Cognitive Profiles of Medicated and Nonmedicated Children Diagnosed with Attention Deficit Hyperactivity Disorder

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A STUDY OF THE COGNITIVE PROFILES OF MEDICATED AND NONMEDICATED CHILDREN DIAGNOSED WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER

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Dissertation Approval

This is to certify that the thesis presented to us by Amy McLaughlin

on the 20th day of May, 2009, in partial fulfillment of the requirements for the degree of Doctor of Psychology, has been examined and is acceptable in both scholarship and literary quality.

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Abstract

Attention Deficit Hyperactivity Disorder is among the most commonly diagnosed childhood disorders, with symptoms that can cause significant difficulties in the educational setting. Deficits related to working memory and processing speed are thought to be a core feature of ADHD. As such, research using traditional measures of cognitive functioning has shown that children diagnosed with ADHD tend to perform poorly on measures of processing speed and working memory, relative to non-ADHD individuals and relative to measures of other cognitive abilities. Psychostimulant medication is a common treatment for ADHD and research overwhelmingly supports its positive impact on behavior and concentration; however, research related to its impact on cognitive functioning is sparse and findings have been equivocal.

The major purpose of the current study was to determine whether or not there are significant differences in the cognitive profiles of individuals with ADHD relative to non-ADHD controls. Of particular interest was the functioning of children with ADHD on measures of processing speed and working memory relative to non-ADHD children and relative to measures of other cognitive abilities. Furthermore, this study was designed to investigate the effects of medication on the performance of ADHD subjects on measures of cognitive functioning.

The results of this study found that students with ADHD did perform significantly lower on measures of processing speed and working memory on the WISC-IV relative to non-ADHD subjects and relative to measures of verbal and nonverbal reasoning skills. This lends support to previous research and hypotheses, indicating that working memory and processing speed deficits are a core feature of ADHD. This study did not find

significant differences between students who were medicated relative to those who were not on overall IQ, Index, or Subtest scores of the WISC-IV. On the other hand, nonmedicated ADHD subjects were more likely than medicated ADHD subjects to display GAI scores greater than WMI, which provides some support for the positive effects of medication on working memory, although much more research is needed to make this claim. No support for positive medication effects on processing speed was found in this study.

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CHAPTER 1

Introduction

Statement of the Problem

Among the most common (Shaywitz, Fletcher, & Shaywitz, 1995) and most highly studied neurobehavioral disorders of childhood (Barkley, 1991; Robins, 1992), Attention Deficit/ Hyperactivity Disorder (ADHD) affects approximately 3% to 8% of the school-aged population in the United States (American Psychiatric Association, 2000; Barkley, 1998). Children with ADHD present with attention, impulse control, and motor activity impairments that cause academic and behavioral difficulties in the educational setting (Landau & Burcham, 1995). In 1998, the National Institutes of Health (NIH) assembled a team of experts representative of various professional disciplines from across the United States for the purpose of reviewing the literature on ADHD and generating a position paper outlining important diagnostic and treatment issues. The consensus paper indicated that ADHD represents a major public health concern (National Institutes of Health, 1998).

Although ADHD is a commonly diagnosed childhood disorder, there are concerns about the best way to diagnose and treat children with ADHD (Connors, 2000). Problems with current diagnostic practices are due to the fact that ADHD is primarily conceptualized as a behavioral disorder; it is often diagnosed on the basis of a parent interview using behavioral criteria, and parent and/ or teacher rating scales. Behavioral methods are imperfect measures because of limited inter-rater agreement, given the fact that expectations and acceptances of child behaviors can vary across settings and have limited discriminant validity (Hale, How, DeWitt, & & Coury, 2001). In addition, other

learning and behavioral disorders can co-occur with ADHD. For example, some research suggests that children with ADHD, Hyperactive-Impulsive Type (ADHD-HIT) are likely to have comorbid externalizing behavior disorders (Jensen, Martin, & Cantwell, 1997), but the inattentive type more frequently co-occurs with learning disabilities and internalizing disorders (Biederman, Faraone, & Lapey, 1992; Jensen et al., 1997; Jensen et al., 1997; Jensen et al., 1997). Comorbidity with other disorders complicates diagnosis (Biederman et al., 1992). Given comorbidity issues and the subjective nature of information gathered from informants, current diagnostic practice for identifying ADHD is problematic.

In addition to behavioral indications, an emerging body of research points to potential cognitive indicators of ADHD. Based on his research, Barkley (1998) conceptualized ADHD as a deficit in behavioral inhibition linked to neuropsychological abilities. As such, he theorized that ADHD results in cognitive impairments in the areas of working memory and processing speed. Ruckledge and Tannock (2002) concluded from their study that neuropsychological deficits such as verbal working memory and processing speed, rather than behavioral inhibition may be central to a description of ADHD. Some research has shown that ADHD affects overall levels of performance on intelligence tests (Barkley, 2000; Mahone et al., 2003). Comparisons of ADHD children with non-ADHD children in the normative samples of various editions of the Wechsler Intelligence Scale for Children have revealed differences in overall IQ, as well as on index and domain scores. Studies have also shown that children diagnosed with ADHD perform poorly on neuropsychological tests, particularly on measures of processing speed

and working memory (Kail & Salthouse, 1994; Kail, 2000; Kalff et al., 2002; Karatekin & Asarnow, 1998).

Psychostimulant medication is perhaps the most common form of intervention for children in the United States who have ADHD (Purdie, Hattie, & Carroll, 2002), which makes the effects of psychostimulant medication relevant to clinicians and educators alike. Barkley (2006) suggested that medication has the most salient effect upon behavior and concentration, with performance on intelligence tests much less affected by medication. In line with Barkley's hypothesis, there is much research supporting the efficacy of stimulant medication for improvement in behavioral symptomatology such as attention, concentration, and hyperactivity (Barkley, 1998; DuPaul, Barkley, & McMurray, 1994; Hale & Fiorello, 2004; Pelham & Milich, 1991). Research investigating the effects of medication on cognitive functioning is sparse; few utilize full-scale tests of intelligence to measure medication effects. Studies evaluating cognitive effects of medication have yielded inconsistent results.

Much of the research investigating the impact of ADHD on cognitive functioning has pointed to deficits in processing speed and working memory. Some research has suggested that as the demands on processing speed and working memory have increased on the Wechsler scales, the IQ's of ADHD students have been found to decrease (Zimmerman & Woo-Sam, 1997). In fact, Mahone and colleagues (2003) found that the ADHD group performed more poorly on the WISC-III than on the WISC-R. The current version of the Wechsler scales for children (WISC-IV) places even greater demands on processing speed and working memory capacities, which has implications for the cognitive performance of children with ADHD. During the initial validation of the

WISC-IV, 89 children ages 8-13 with a diagnosis of ADHD were tested. Data related to medication status was gathered, but the data for medicated and non-medicated children was not analyzed separately. Compared with matched controls, ADHD group mean differences for Processing Speed Index (PSI) scores reflected a moderate effect size. Small effect sizes were reported between the Verbal Comprehension Index (VCI), Working Memory (WMI), and Full Scale Intelligence Quotient (FSIQ) scores. At the subtest level, the largest effect sizes for group mean differences were found on the Coding and Arithmetic subtests. Other WMI and PSI subtests had modest or small effect sizes. Because medicated and nonmedicated students were grouped together, it is possible that improvement in functioning because of medication use masked some of the processing speed and working memory deficits present in the nonmedicated students (Friedman, 2006). In 2006, Friedman utilized two samples of male students diagnosed with ADHD, one medicated and one not medicated, and matched them with non-ADHD controls from the WISC-IV normative sample in order to expand on the validation studies and to explore the relationship between medication and cognitive functioning. The only significant index/ factor scores differences found between groups occurred on the Working Memory Index, with the nonmedicated ADHD group performing more poorly than the non-ADHD matched controls. At the subtest level, the nonmedicated ADHD group performed significantly lower on DS than the medicated ADHD group and non-ADHD controls. Lower performances on the Working Memory Index relative to non-ADHD controls, however, did not carry over to a significant difference in FSIQ as was predicted. Friedman (2006) further noted that preliminary analyses of the data collected suggests potential differences between ADHD and control groups in reference to the

degree of score differences between Verbal Comprehension and Working Memory Index scores and recommended that future research be conducted in this area. In addition, more research is needed to investigate the impact of psychostimulant medication on cognitive functioning.

Purpose of the Study

The major purpose of the current study is to determine whether or not there are significant differences in WISC-IV FSIQ, Index scores and Subtest scores within individuals with ADHD relative to non-ADHD controls. Furthermore, this study will investigate the performance of ADHD and non-ADHD subjects on measures of working memory and processing speed, relative to measures of other cognitive abilities, such as verbal and nonverbal reasoning skills. Finally, this study will investigate the impact of medication status (medicated versus nonmedicated) on Index and Subtest score differences in individuals with ADHD.

Literature Review

Origins of ADHD. References to individuals with problems related to inattention, hyperactivity, and poor impulse control are noted in literature dating back to Shakespeare, although it did not become a serious clinical interest until 1902 when it was introduced by English physician George Still to the Royal Academy of Physicians (Barkley, 1997a). Interest in children with these characteristics surfaced in North America around 1917-1918, at the same time as the great encephalitis epidemic, because children surviving these brain infections demonstrated characteristics similar to the condition that is known today as ADHD (Barkley, 1997a). Over time, however, researchers began to observe these behavioral problems associated with brain damage or

mental retardation in children without evidence of brain injury or retardation. The diagnostic term "minimal brain damage" and later "minimal brain dysfunction" was applied to these cases.

As researchers began focusing attention on the hyperactivity and poor impulse control exhibited by these children, the condition became known as "hyperkinetic impulse disorder" and later "hyperactive child syndrome" in an effort to provide a more descriptive view of the disorder (Barkley, 1997a; Barkley, 2000). Although many clinicians and researchers held onto the belief that the condition had a neurological basis, psychiatry remained influenced by the psychoanalytic view that children's mental disorders were primarily a reaction to various environmental factors (Barkley, 1997a). As a result, in 1968 this disorder was introduced into the Diagnostic and Statistical Manual of Mental Disorders (DSM-II: American Psychiatric Association [APA], 1968) under the label of "Hyperkinetic Reaction of Childhood." Diagnostic criteria for this condition included observable disruptive behavioral excesses. Coinciding with scientific advancements in the field of ADHD, each subsequent edition of the DSM has substantially revised both the nomenclature and nosology of this disorder (Schwean & Saklofske, 2005). The 1980 revision of the DSM (DSM-III), reflected advances in research, demonstrating that subtle cognitive deficits in response inhibition and attention were more prominent and reliable indicators of ADD than were motor excesses; it also suggested subtypes differentiated by the presence or absence of hyperactivity (Schwean & Saklofske, 2005). This differentiation of subtypes was abandoned, however, in the 1987 edition (DSM-III-R) because of a lack of empirical support, and instead a general attention-deficit hyperactivity disorder (ADHD) characterized by developmentally

inappropriate degrees of inattention, impulsivity, and hyperactivity was included (Schwean & Saklofske, 2005). In line with the findings of factor analytic studies supporting the differentiation of two factors (inattention and hyperactive-impulsive) and with studies documenting the external validity of subgroups differentiated by these factors (Lahey et al., 1994), the current version of the DSM (DSM-IV-TR, 2000) recognizes three subtypes of ADHD: ADHD, predominantly inattentive type (ADHD-IT); and ADHD, predominantly hyperactive-impulsive type (ADHD-HIT); and ADHD, Combined Type (ADHD-CT).

Current diagnostic criteria for ADHD, as established by the DSM-IV-TR (APA, 2000), includes the presence of at least 6 of 9 symptoms either of inattention and/ or hyperactivity/ impulsivity that have been present before age 7; have persisted for at least 6 months; are more frequent and severe than is typical for the age group; manifests in multiple settings, and adversely affects functioning. The 9 potential inattentive symptoms are as follows: (a) fails to give close attention to details; (b) has difficulty sustaining attention in tasks or play; (c) does not seem to listen when directly spoken to; (d) does not follow through on instructions and fails to complete school work, chores, etc. (e) has difficulty organizing tasks and activities; (f) avoids, dislikes, or resists engaging in tasks requiring sustained mental effort; (g) loses things necessary for tasks; (h) is easily distracted by extraneous stimuli; and (i) is forgetful. The 9 hyperactive-impulsive symptoms include the following: (a) fidgets with hands or feet or squirms in seat; (b) leaves seat in situations in which remaining seated is expected; (c) runs about or climbs excessively (in adolescents/ adults may be a sense of restlessness); (d) difficulty engaging in activities quietly; (e) is frequently “on the go” or appears as if “driven by a motor”; (f)

talks excessively; (g) blurts out answers before a question is completed; (h) has difficulty awaiting turn; and (i) interrupts or intrudes on others. The DSM-IV definition of ADHD reflects important advances in ADHD knowledge; however, it fails to account for important changes that can occur during the course of development, such as a reduction of hyperactive symptoms over time (Power & DuPaul, 1996).

ADHD is most commonly diagnosed during elementary school when school adjustment is compromised (APA, 2000). In the majority of cases, the disorder remains relatively stable throughout early adolescence, and symptoms such as motor hyperactivity seem to remit during late adolescence and adulthood (APA, 2000). A minority of adults, however, continue to experience a full range of symptoms into mid-adulthood, yet others will retain only some of the symptoms.

Prevalence of ADHD. According to the DSM-IV-TR, the prevalence of ADHD in school-age children is estimated at 3% to 7% (APA, 2000). Reported rates vary depending on methodology, diagnostic system utilized, and the nature of the population studied. When looking at community samples, Rowland, Lesesne and Abramowitz (2002) reported a prevalence of 2% to 18%. There has been a rapid rise in the prevalence rates of ADHD (Purdie, Hattie, & Carroll, 2002). In fact, evidence suggests that the prevalence of ADHD as defined in the DSM-IV may be somewhat greater than the prevalence based on the DSM-III-R criteria because of the inclusion of the Predominantly Hyperactive-Impulsive and Predominantly Inattentive Types (APA, 2000). Robison and colleagues (1999) found that this increase in diagnosis coincided with a 2.9-fold increase in the number of ADHD individuals who had been prescribed stimulant medication. In 2003, the United States National Survey of Children's Health (NSCH) found that

approximately 4.4 million or 7.8% of children ages 5 to 17 have been diagnosed with ADHD. Of these children, 2.5 million or 56% reported taking medication for the disorder. This survey also found that reported ADHD increased with age, and was significantly higher for children greater than 9 years of age than for children aged 4 to 8 years. Regardless of gender, overall medication by age patterns were curvilinear, with the prevalence of medication highest in children aged 9 to 13, compared with younger and with older children (NSCH, 2003).

ADHD is known to occur in various cultures, although there are variations in the reported prevalence rates among Western countries; this is likely related to different diagnostic practices than to differences in clinical presentation (APA, 2000). International cross-cultural studies (i.e., (Anderson, Williams, McGee, & Silva, 1987; Brewis, Schmidt, & Meyer, 2001) suggest that prevalence rates for ADHD worldwide are similar to US rates.

In terms of gender, ADHD appears to be more common in males than in females, with a male-to-female ratio ranging from 2:1 to 9:1, depending on the subtype (the gender ratio is hypothesized to be less pronounced in ADHD-IT) and setting (APA, 2000). Male to female ratios in community samples have been found to be 3:1, but the ratios have ranged from 6:1 to 9:1 in clinic-referred samples (Gaub & Carlson, 1997). Rates of treatment for ADHD followed the same pattern as noted for diagnosis, with males of all ages more likely to take medication for their ADHD diagnosis.

Studies comparing the symptomatology of ADHD in girls versus boys found that girls displayed lower levels of hyperactivity, lower rates of comorbid conduct disorders, lower rates of other externalizing behavior, but greater intellectual impairment

(Biederman et al., 1999; Gaub & Carlson, 1997). On the other hand, a more recent study conducted by (Hartung et al., 2002) found that boys and girls ages 3 to 7 years with a diagnosis of ADHD did not differ on many factors, including internalizing symptoms, academic achievement, subtype prevalence, and cognitive abilities.

The NSCH (2003) found that within the United States the prevalence of ADHD diagnosis is higher among non-Hispanic, primarily English- speaking, and insured children. Prevalence rates were also much higher in families in which the most highly educated adult completed 12 years of education/ high school graduate, compared with children in families in which the most highly educated adult had either a higher or lower level of education (NSCH, 2003). Prevalence rates of ADHD among nonwhite, American ethnic minority groups have not been established and little research has been conducted to describe ADHD in ethnic and racial groups (Kendall & Hatton, 2002). Therefore, ADHD has been characterized as a primarily white, middle class disorder, because the majority of the research has been conducted with this population.

Etiology of ADHD. The exact causes of ADHD are unknown at this time; however, there are several factors that appear to be implicated, because they have been shown to be related to increased risk for ADHD in children. The causal factors that have received the greatest attention and support in the literature are genetic factors and biological factors (i.e., those that have a direct affect on brain development or functioning) (Barkley, 1997a). Research has provided little support for psychosocial factors as contributing to the development of ADHD (Barkley, 1997a). In fact, it has been posited that links found between poor child management by a parent and ADHD may in fact be more attributable to the parent's own ADHD (a genetic factor) than to the

environment (Frick & Jackson, 1993). Although Barkley (1997), a major theorist in the field of ADHD, does not give much attention to psychosocial factors, given the lack of support in the research; however, he does suggest that environment can play a role in shaping and molding the nature and severity of the biologically-based vulnerability to poor inhibition. In addition, he notes that the risk for the development of comorbid disorders with ADHD (i.e., ODD, CD, anxiety, and depression) is highly correlated with family environmental factors. Thus he concludes that environment does not play a primary role in causation; however, it does in determining outcome.

Neurological Factors. Theories of the neurological factors related to ADHD have been developed, based on similarities noted between the symptoms of ADHD and those produced by lesions or injuries to frontal lobes, and more specifically the prefrontal cortex (Benton, 1991; Heilman, Voeller, & Nadeau, 1991; Mattes, 1980). Deficits in sustained attention, inhibition, regulation of emotion and motivation, and the organization of behavior over time were observed in individuals with injuries to the prefrontal region of the brain (Gratton & Eslinger, 1991). There is also other evidence to support a neurological basis of ADHD. For example, ADHD symptomatology is persistent with an early onset; it has been associated with other developmental disorders that are believed to arise from neurological factors; it has a significant relationship to adversities during the pre- and postnatal periods, and symptoms tend to improve dramatically with the use of stimulant medication (Barkley, 1997a). In addition, ADHD subjects have been repeatedly found to perform poorly on neuropsychological tests associated with prefrontal lobe functions including inhibition, persistence, planning, working memory,

motor control and fluency, and verbal fluency (Barkley, DuPaul, & McMurray, 1990; Barkley, 1997b)(Barkley, Grodzinsky, & DuPaul, 1992; Goodyear & Hynd, 1992).

More recent research suggests that the right prefrontal cortex, caudate nucleus and globus pallidus are typically smaller in children with ADHD, indicating problems with connectivity between the brain regions that modulate attention, stimulus processing, and impulsivity (Dophide, 2001). Hale and Fiorello (2004) describe ADHD as a frontal-subcortical disorder. Frontal-subcortical abnormalities, such as asymmetric/ dysmorphic conditions (Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990), abnormal electrical activity (Novak, Solanto, & Abikoff, 1995), and decreased cerebral blood flow (Ernst et al., 1994; Lou, Henriksen, & Bruhn, 1984; Lou, Henriksen, Bruhn, Borner, & Nielsen, 1989; Zametkin et al., 1993) have been found in children with ADHD. Although the research has overwhelmingly supported a link between ADHD and frontal lobe impairment, more research is needed to determine differences across subtypes of ADHD. The few studies that have attempted to investigate frontal lobe impairment across ADHD subtypes have not yielded consistent results. For example, O'Driscoll and colleagues (2005) found impairments in the executive functions of motor planning and response inhibition with ADHD-CT, but not ADHD- IT boys, and Geurts, Verte, Oosterlann, Roeyers, and Sergeant (2005) found similar performance between ADHD-IT and ADHD-CT subtypes on measures of executive function.

Theoretical Conceptualizations of ADHD

Over the years, ADD has been subject to many reconceptualizations, redefinitions, and renamings (Lahey et al., 1988) because of the considerable heterogeneity in etiology, cognitive, academic, psychological, and family correlates;

clinical courses; and outcomes, and intervention responses among children with this disorder (Schwean & Saklofske, 2005). Because of this, efforts have been made to delineate more homogeneous subgroups. There has been particular controversy about whether or not the IT and HIT/ CT are actually subtypes of a single disorder or actually two distinct separate disorders (Cantwell & Baker, 1992).

ADHD was initially conceptualized as excessive motor activity related to minimal brain dysfunction; however, recent advances in medical technology have revised the causal explanatory hypotheses of ADHD (Schwean & Saklofske, 2005). Specifically, this research has implicated the prefrontal-striatal network and its interconnections with other brain regions (Barkley, 1998). Two major theories, Barkley's (Barkley, 1997b; Barkley, 1997a) disinhibition model, and Rapport and colleagues' (Rapport, Chung, Shore, Denney, & Isaacs, 2000; Rapport, Chung, Shore, & Isaacs, 2001) working memory model, have followed from this research.

Disinhibition Model. Barkley (1997) asserts that ADHD is not primarily a disorder of attention, but rather a developmental disorder of behavioral inhibition that hinders the development of effective self-regulation. He defines behavioral inhibition as three interrelated processes which include: inhibiting the initial response; stopping an ongoing response, and interference control (not becoming distracted by competing events and responses). He further postulates four neuropsychological abilities (nonverbal working memory; internalization of speech; self-regulation of affect; motivation and arousal; and reconstitution), which are considered executive functions that rely partially on behavioral inhibition for effective execution. Therefore, the primary impairment in behavioral inhibition leads to secondary impairments in executive functions. Together,

these primary and secondary impairments lead to impairment in a fifth area, the motor control system, which manifests as "decreased effectiveness in motor/ behavioral control or in guidance by internally represented information and self-directed action" (Barkley, 1997a). Self-control, which is dependent upon inhibition, is defined as any response or chain of responses that occur within an individual in order to alter the probability of subsequently engaging in an action in order to maximize both short and long term outcomes (Barkley, 1997a). These self-directed responses that occur during a delay in response are thought to become progressively more covert over the course of development. The self-directed actions that an individual utilizes to self-regulate are known as executive functions. Behavioral inhibition and self-regulation and its associated executive functions are thought to be mediated by the prefrontal cortex of the brain and its interconnections with the striatum (Barkley, 1997a). It is important to note that Barkley (Barkley, 1997a; Barkley, 2000) clarifies the fact that his model does not apply to those with ADHD-IT, which he suggests is qualitatively different from ADHD-HIT and ADHD-CT.

Working Memory Model. Rapport and colleagues worked from the following conceptual model of ADHD:

> Biological influences (e.g., genetics) give rise to individual differences in the functional properties of neurobiological systems (e.g., dopaminergic-noradrenergic neurotransmission) that are etiologically responsible for the core psychological (cognitive and behavioral) features of ADHD. Peripheral (secondary) features are conceptualized as causal by-products of core features (Rapport et al., 2000)

The peripheral or secondary features referred to in this model include difficulties that are thought to be caused by the more primary features of the disorder such as academic underachievement, inadequate social skills, low frustration tolerance and strained family relationships. Rapport and colleagues, therefore, argue that the most effective interventions will be those aimed at core deficits, because treatment-related improvement in core domains should correspond with gains in peripheral areas. Considering this model, however, Rapport and colleagues (2000; 2001) found that gains made in these three core areas (attention, self-control, and hyperactivity) accounted for only 20% of improvement noted in academic achievement (the peripheral variable). Based on this finding and on other research which had been conducted, they proposed a model of ADHD, positing the idea that working memory plays a crucial role in the organization of behavior. As such, organized responding is dependent upon the capacity of working memory to perform three functions. These functions include: (1) generating and holding representations of input stimuli, (2) searching memory for matches, and (3) accessing and holding on to appropriate behavioral responses to input stimuli. Disruption to any of these working memory processes should result in tangential or random responses to environmental stimuli. This model is purported to account for the disorganized behavior that is characteristic of children with ADHD.

A second component to this model posits the idea that failure of working memory not only causes disorganized behavior but also compels children to seek stimulation by redirecting their attention to other environmental stimuli. This inability to maintain working memory representations is speculated, therefore, to lead to behavior serving to increase the rate at which input is delivered to working memory in order to compensate

for the rapid fading of representations. Another potential hypothesis suggested that this redirection of attention is a form of escape from monotonous or extremely demanding tasks. This stimulation- seeking behavior is observed by others as hyperactive and impulsive. Based on this proposed model, impulsive acts are considered disorganized patterns of behavior that manifest from an inability to maintain working memory representations either of the stimulus context, of relevant memory traces, or of both. Therefore, considering the consequences of behavior is considered by these researchers to be highly dependent upon working memory. In contrast to Barkley's model, Rapport and colleagues view impulsivity and hyperactivity as causal by-products and working memory as a core causal cognitive process.

Although there is disagreement and debate over the core deficit of ADHD, there is considerable agreement that these individuals display disorganized behavior, problems with self-control, and weaknesses in cognitive areas such as working memory and processing speed. Much research has been devoted to exploring the cognitive deficits present in individuals with ADHD.

Cognitive Functions and ADHD

Although diagnostic criteria for ADHD are primarily behavioral in nature, research suggests that cognitive deficits, such as impairments in attention, response inhibition, and perceptual-motor speed are also core features of the disorder (Barkley et al., 1990; Sykes, Douglas, & Morgenstern, 1973). In 1992, Barkley, Grodzinsky, and DuPaul reviewed 22 neuropsychological studies of the frontal lobe functions of children with ADHD, both with and without hyperactivity. Based on this review, they concluded that children with ADHD present with cognitive deficits in sustained attention and

inhibitory control, which they related to subtle frontal lobe impairments in the brain as measured on neuropsychological testing. Lahey et al. (1998) found neuropsychological impairment with ADHD manifested as deficits in perceptual-motor processing speed. Based on the literature, many researchers have characterized ADHD as a condition involving executive control difficulties that impact working memory and processing speed (DeFockert, Rees, Frither, & Lavie, 2001; Weiler, Bernstein, Bellinger, & Waber, 2000; Weiler, Bernstein, Bellinger, & Waber, 2002).

Working Memory. Working memory is the ability to maintain information actively in conscious awareness while performing some operation or manipulation with it and producing a result (Wechsler, 2003). Studies conducted by Fry and Hale (1996) and Perlow, Jettuso & Moore (1997), have shown that working memory is an essential component of fluid reasoning and higher order cognitive processes, and is related to achievement and learning. DeFockert and colleagues (2001) suggested that the greater the demands on working memory, the more likely an individual is to become distracted by irrelevant information. They hypothesized that either of these impairments in working memory gives rise to distractibility or vice versa.

The theoretical and functional structure of working memory is still under debate (Alloway, Gathercole, & Pickering, 2006; Engle, Tuholski, Laughlin, & Conway, 1999; Miyake, Friedman, Rettinger, Shah, & Hegarty, 2001). One model suggests that there are two separate pools of modality-specific resources for storing and manipulating auditory-verbal and visual-spatial stimuli (Shah & Miyake, 1996). Another model argues that working memory is composed both of visual (visuospatial sketchpad) and of verbal (phonological loop) storage systems that are regulated and controlled by a central

executive component (Baddeley, 1986). Finally, another model proposes four separable components including the visual-spatial storage of information; the visual-spatial manipulation of information; the auditory-verbal storage of information, and the auditory-verbal manipulation of information (Friedman & Miyake, 2000; Miyake et al., 2001). Baddeley's (1986) tripartite model has been the most influential (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005) and continues to be supported by recent research (Alloway et al., 2006; Bedard, Jain, Hogg-Johnson, & Tannock, 2007).

Working memory impairments have been linked with ADHD theoretically (Barkley, 1997b; Castellanos & Tannock, 2002; Rapport et al., 2001), as well as empirically (Martinussen et al., 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Working memory functions are thought to be highly dependent on frontostriatal brain regions (Bunge, Ochsner, Desmond, Glover, & Gabrieli, 2001; Kondo, Morishita, & Osaka, 2004; Lewis, Dove, Robbins, Barker, & Owen, 2004; Smith & Jonides, 1999) and the cerebellum (Gottwald, Mihaljlovic, Wilde, & Mehdorn, 2003; Lalonde, 2003). Furthermore, depending on the modality of the central executive tasks, different neural structures are activated, with verbal tasks more lateralized to the left and spatial to the right (Fletcher & Henson, 2001). Research has also demonstrated that dopaminergic and noradrenergic systems modulate working memory processes (Arnsten, 2001; Goldman-Rakic, Castener, Svensson, Siever, & Williams, 2004).

In 2005, Martinussen and colleagues conducted a meta-analysis to determine the empirical evidence for working memory deficits in children and adolescents with ADHD. Based on their analysis of 26 studies published between 1997 and 2003, they found that children with ADHD did exhibit deficits in multiple components of working memory that

were independent of comorbidity with language learning disorders and weaknesses in general intellectual ability. The overall effect sizes obtained were greater for spatial storage and spatial central executive working memory than those obtained for verbal storage and verbal central executive control; however, the authors cautioned that findings should be considered exploratory in nature, given the small number of studies included, particularly in the spatial domains. Nonetheless, the finding that working memory deficits are present in ADHD provides support for frontostriatal and dopamine-system dysfunction in ADHD. It was further speculated that the academic difficulties experienced by children with ADHD are related to working memory deficits, rather than to inattention alone.

Karatekin and Asarnow conducted a study in 1997 to investigate verbal and spatial working memory functioning in children with ADHD and children with childhood-onset schizophrenia and matched normal controls. Their results showed that both the ADHD children and the schizophrenic children displayed deficits in verbal and spatial working memory.

Cornaldi and colleagues (2001) set out to investigate whether or not children with ADHD would manifest deficits in working memory related to intrusion errors. They utilized auditory working memory measures in this study. Their results showed that children with ADHD have working memory problems only when a high degree of control is required. No significant problems were noted in ADHD subjects on working memory tasks that required the individual to recall all of the material presented. These results supported findings that a working memory difficulty in children with ADHD can be related to inhibition problems. ADHD children are not capable of suppressing

information that initially has to be encoded and processed, but then must subsequently be excluded from memory. In a second study, Cornaldi and colleagues (2001) looked specifically at visuospatial working memory problems in children with ADHD. The results supported the theory proposed by Baddeley (1986) that ADHD children have deficits in active control processes, also known as the Central Executive component of the Working Memory system. These deficits are subsumed under the category of Executive Dysfunction, which have been linked with pre-frontal lobe functioning (Cornaldi et al., 2001). Thus, this study is consistent with the assumption that the executive dysfunction is a core component of ADHD associated with prefrontal lobe dysfunction. This study also supported Cornaldi and Vecchi's (2000) view that the active working memory deficit implicated in children with a general control problem, such as ADHD, is cross-modal, but can be modality-specific for individuals with other types of learning disabilities.

Processing Speed. In addition to working memory, many studies have also found that ADHD students tend to perform less well on measures of processing speed than nonclinical controls (Prifitera & Dersh, 1993; Rucklidge & Tannock, 2001; Tiholov, Zawallich, & Jansen, 1996; Weiler et al., 2000; Weiler et al., 2002). Clinical research in developmental cognitive psychology has suggested a dynamic relationship between working memory, processing, and reasoning. Given the fact that working memory and processing speed are thought to be interrelated, it is not surprising that in addition to low scores on measures of working memory, ADHD individuals tend to score lower on processing speed measures.

Kail and Salthouse (1994) relate processing speed to mental capacity, and therefore argue that mental capacity can be conceptualized in terms of the speed with which an individual processes many types of information. Research has consistently related speed of information processing, not only to mental capacity, but also to reasoning by the conservation of cognitive resources and the efficient use of working memory for higher order reasoning tasks (Kail & Salthouse, 1994; Kail, 2000). Kail and Salthouse (1994) assert that cognitive change over the lifespan is mediated by change in processing speed and plays a key role in one's ability to think, reason, and remember. For example, they have found that on a wide range of motor, perceptual, and cognitive tasks in which participants must respond rapidly, the following pattern emerges: 8- to 10-year-olds responded at a speed that is 5 to 6 standard deviations below the average speed for young adults; and that 12- and 13-year olds respond at a rate more than one standard deviation below the average for young adults (Kail, 1991). It is theorized that a global mechanism limits the speed with which children and adolescents process information. This global mechanism is not specific to particular tasks or domains, but rather is a fundamental characteristic of the developing information-processing system (Kail, 2000). In addition to age differences, processing speed has been found to be sensitive to neurological conditions such as traumatic brain injury (Donders, 1997). Given the relationship between processing speed and neurological development, as well as the research supporting the relationship between working memory, processing speed, and reasoning, Kail and Salthouse (1994) advocate for the importance of the assessment of processing speed in children. Rapid processing of information is hypothesized to reduce

the demands on working memory, which facilitates reasoning. As such, processing speed is a construct often included on standardized measures of intelligence.

IQ Tests and Effect of Content on ADHD Subjects

Most intelligence tests utilized today yield a general Intelligence Quotient (IQ) score that is derived from a series of tasks that measure various aspects of cognition. For example, the Wechsler Intelligence Scale for Children- Third Edition (WISC-III) yields a Full-Scale IQ (FSIQ) and four index scores which include the Verbal Comprehension Index (VCI), Perceptual Organization Index (POI), Processing Speed Index (PSI), and Freedom from Distractibility Index (FFD). These scores are derived from a series of subtests that measure verbal ability, nonverbal abilities, processing speed, and working memory. The effects of ADHD on IQ can be influenced by the nature and content of the tasks utilized to assess intelligence.

ADHD and the Wechsler Scales

Given the evidence for cognitive deficits related to ADHD, there is support for the use of the Wechsler scales for diagnostic purposes, because they are purported to measure cognitive skills such as processing speed, memory, attention, and visual organization (Sattler, 1992). Since their original publication in 1949, the Wechsler scales have been widely used (Kampaus, 1993). The primary use of these scales has been diagnostic in nature (Wechsler, 1991), although the appropriateness of this utilization has been the topic of considerable debate, particularly related to the identification of children with ADHD (Schwean & Saklofske, 1998). The diagnostic utility of the WISC for the identification of children with ADHD has been empirically tested through a variety of approaches (Assesmany, McIntosh, Phelps, & Rizza, 2001).

One such approach has been to compare the mean performance of children with ADHD to control groups (Schwean & Saklofske, 1998). In general, research of this type has found that students with ADHD demonstrate lower overall levels of intellectual functioning than their non-ADHD counterparts (Barkley, 1990; H. S. Goldstein, 1987). As part of a larger study, Barkley et al. (1990) compared intellectual functioning of children with ADHD to non-ADHD controls, using the WISC-R. Their findings indicated that ADHD students both with and without the hyperactivity component had significantly lower mean FSIQ scores than non-ADHD controls. Goldstein (1987) found that students rated by teachers as inattentive had significantly lower WISC scores than non-ADHD controls. Additionally, he found a significant difference between children with hyperactivity and aggression versus children with inattention; hyperactive and aggressive children had higher IQ's than inattentive children.

Research conducted with the WISC-III continued to support the findings that children with an ADHD diagnosis obtain lower overall scores when compared with non-ADHD controls (Anastopoulos, Spisto, & Maher, 1994; Barkley, 1990; Faraone et al., 1993; Tripp, Ryan, & Peace, 2002; Zhuang, Liu, & Zhang, 2001). According to Barkley (1998), the behavioral inhibition impairments and the related executive dysfunction associated with ADHD could be expected to have a small but significant and negative impact on IQ, particularly Verbal IQ.

During the standardization studies, the WISC-III (Wechsler, 1991) was administered to a sample of 68 children ages 7 to 16, with documented ADHD. This sample demonstrated mean IQ scores near the normative average, with low mean scores on the Processing Speed and Freedom from Distractibility scales. Tiholov, Zawallich,

and Janzen (1996) used a sample of 311 students to determine whether or not the WISC-III Processing Speed Factor could be used to distinguish between groups of children with different types of problems. Their finding, which was related to subjects with ADHD, found that those who were also diagnosed with visual motor integration difficulties demonstrated perceptual discrimination difficulties manifested through significantly lower scores on the Symbol Search subtest, but not lower on the Coding subtest than participants without ADHD and visual motor integration problems.

Weiler et al. (2000) set out to study the neuropsychological profile of students with ADHD, primarily inattentive type. Their subjects included 82 children referred for school-related problems. Processing speed was assessed with a number of measures including the WISC-III Coding and Symbol Search subtests. Findings suggested that children with learning problems, in general, exhibited problems on measures of processing speed, and that those diagnosed with ADHD were particularly vulnerable. Weiler and colleagues replicated this study in 2002, this time using computer-based measures to evaluate information processing. They utilized a visual search task and an auditory processing measure. As they predicted, based on their earlier research, the ADHD-IT students performed more poorly than non-ADHD subjects on the visual search task, but not the auditory processing task. Children with reading disabilities had the reverse pattern. From these results, they concluded that children with ADHD-IT do not have global information processing deficits; rather they process visual information more slowly, especially when the cognitive load is increased and they are required to integrate multiple component operations. ADHD-IT subjects did not perform more slowly on simple reaction time measures.

Researchers have also utilized the WISC-III to evaluate auditory working memory processes in children with ADHD. Results of some studies suggest that FFD for ADHD children, although for the vast majority not a significant weakness relative to peers, was significantly lower relative to other WISC-III factor scores (Anastopoulos, Spisto, & Maher, 1994; Reinecke, Beebe, & Stein, 1999) or FSIQ (Mayes, Calhoun, & Crowell, 1999). Specifically, Anastopoulos and colleagues found that the FFD factor index was significantly lower than the VC and PO factor scores within a small clinical sample (n=40) with ADHD. Analysis at the individual level, however, failed to yield these same differences for many of the children. A study conducted by Reinecke and colleagues (1999) utilized a larger sample (n= 200) and also found differences between FFD and other factor scores; however, correlational analyses failed to support the validity of the FFD as a measure of attention. An even larger study (n= 301) by Krane and Tannock (2001) yielded similar findings, with results again suggesting a limited ability to predict ADHD because of high false-negative rates within the ADHD group and high false-positive rates within the clinical and nonclinical comparison groups.

Although the aforementioned studies have provided useful information regarding the cognitive functioning of children with ADHD, they have limited diagnostic utility, given the fact that the cognitive patterns of children with ADHD are similar to the patterns exhibited by children with other educational disabilities (Newby, Recht, Caldwell, & Schaefer, 1993; Teeter & Smith, 1993). Furthermore, the analyses were conducted using group mean differences, which limits the ability to predict patterns of individual performance (Assesmany et al., 2001).

Some studies have examined the subtest level for patterns among ADHD students. For example, Saklofske, Schwean, Yackulic and Quinn (1994) found that in their sample of 45 children diagnosed with ADHD, the Processing Speed subtests of Symbol Search and Coding were among the lowest subtest scores. Reliable research conducted with the earlier versions of the WISC in the ADHD population found low mean scores on the Arithmetic, Coding, Information, and Digit Span subtests, a pattern that came to be referred to as the ACID profile (Dykman, Ackerman, & Oglesby, 1980; Prifitera & Dersh, 1993).

Standardization studies of the WISC-III (Wechsler, 1991) demonstrated that the lowest mean subtest scores for students with ADHD occurred on the Coding and Digit Span subtests. In addition, the WISC-III standardization studies included further analysis, considering the ACID profile. An ACID composite score was calculated, based on Arithmetic, Coding, Information, and Digit Span subtest scores. Findings indicated that the full ACID profile was exhibited in the scores of 11.8% of the ADHD sample, compared with 1.1% of the standardization sample. The partial ACID profile (based on any three of the ACID subtests) was demonstrated by 27.9% of the ADHD sample, compared with 5.6% of the standardization sample. Based on these results, it was recommended that when the ACID profile is present, an attention-deficit disorder should be considered; however, it was also cautioned that if not present, ADHD should not be ruled out, given the fact that a majority of children with ADHD in this sample did not exhibit the ACID profile.

With the third revision of the WISC, a new performance profile pattern for ADHD was proposed to reflect the addition of the Symbol Search subtest (Kaufman,

1994; Mayes, Calhoun, & Crowell, 1998). This new profile, SCAD, reflected a pattern of lower scores on the Symbol Search, Coding, Arithmetic, and Digit Span subtests. In 1999, Mayes, Calhoun and Crowell analyzed the WISC-III data in clinical samples of ADHD children and normally developing children. They found that for the ADHD group, the mean FSIQ was greater than the FFD at all ages. Subtest analysis lent support for the SCAD profile. Twenty-three percent of the ADHD subjects and none of the non-ADHD group had Digit Span and Arithmetic as two of their three lowest subtest scores.

On the other hand, Kaufman (1994) argued that the SCAD profile added little improvement to the ACID profile in terms of differential diagnosis; he suggested, however, that comparing the SCAD profile with the subtests contributing to the Verbal Comprehension and/ or Perceptual Reasoning Index could be useful in distinguishing ADHD or LD students from non-ADHD children.

Although the performance profiles have provided useful information about the cognitive performance of students with ADHD, they, too, have limited differential diagnostic utility (Prifitera & Dersh, 1993; Schwean & Saklofske, 1998). Furthermore, the profile analysis studies that have been discussed continue to be based on mean group differences, which allows them to answer the question of whether or not group membership is associated with reliable mean differences, but does not address questions of classification or prediction (Assesmany et al., 2001). As a result, another approach has been used by some researchers to address the question of whether or not a reliable, differential diagnosis between groups of children with ADHD and children without ADHD can be made from a child's Wechsler scores (Ownby & Matthews, 1995; Stewart & Moely, 1983; Wielkiewicz, 1990). This approach, known as discriminant function

analysis, was designed to provide classification into groups, as well as an interpretation of the dimensions underlying the differences in predictors (Assesmany et al., 2001). Studies employing the WISC-R found that the Freedom from Distractibility factor (FFD) did not discriminate children with ADHD from those without (Ownby & Matthews, 1995; Stewart & Moely, 1983; Wielkiewicz, 1990). Neither did studies utilizing the WISC-III lend support for the use of the Freedom from Distractibility factor in diagnosing ADHD (Prifitera & Dersh, 1993; Riccio, Cohen, Hall, & Ross, 1997). In addition, analysis by Riccio and colleagues (1997) found that FFD does not correlate significantly with other measures of attention or concentration and may perhaps be a better measure of memory.

On the other hand, Assesmany and colleagues (2001) utilized discriminant function analysis and found that the WISC-III has considerable discriminant validity for ADHD diagnosis. Based on the results of their study, which included 80 children (40 with ADHD and 40 controls), they concluded that four WISC-III subtests contributed significantly to the prediction of group membership. When these four subtests, which included Digit Span, Information, Vocabulary, and Picture Completion, were used as diagnostic predictors, 90% of children classified as ADHD and 87.5% of the non-ADHD children were correctly identified. Of these subtests, the two best predictors were Digit Span and Information. They suggested that Digit Span and Information subtest performance could be useful in determining whether or not additional assessment should be conducted to substantiate a diagnosis of ADHD.

The latest revision of the Wechsler Scales, the WISC-IV (Wechsler, 2000), puts greater emphasis on working memory and processing speed, the subdomains that appear

to be most highly affected by ADHD. Because such recent research has suggested that the WISC-IV might adversely impact IQ scores of ADHD children (Barkley, 2000; Mahone et al., 2003), Mahone and colleagues (2003) reported that reviews of the Wechsler Scales for children indicate that individuals score, on average, 5 to 6 points lower on the WISC-III (Wechsler, 1991) than on the WISC-R (Wechsler, 1974). These differences were distributed disproportionately over subtests with larger discrepancies found within the Performance Scale. Horn-Alsberge (1999) also found decreases in performance between the WISC-R and WISC-III. Specifically, results found that children with learning disabilities, ADHD, and affective disorders earned FSIQ's approximately 6 points lower on the WISC-III than the WISC-R. VIQ and PIQ were also approximately 5 points lower on the WISC-III than the WISC-R. Based on these findings, Mahone et al. (2003) cautioned that changes on reviewed subtests of the WISC-III Performance Scale may place children with ADHD at a disadvantage when compared with their performances on the analogous WISC-R subtest. They hypothesized that the increased executive demands associated with the WISC-III contributed to the lower FSIQ scores. In addition, Barkley (2000) argued that the FFD subtests included in the WISC-III assess working memory and therefore may place children with ADHD at a disadvantage.

In light of the research documenting weak processing speed and working memory in individuals with ADHD, it is not all that surprising that decreases were found between WISC-III and WISC-R scores. In general, the emphasis of these abilities on intelligence tests has had a great impact on children diagnosed with ADHD. Schwean, Saklofske, Yackulic and Quinn (1993) reported that, based on a discriminant validity study of the

WISC-III and 45 ADHD children in which intercorrelations between subtests, index scores and IQ scores were examined, patterns of correlations for the ADHD group were similar to those reported for the WISC-III standardization sample across parallel age groups. Furthermore, Prifitera, Saklofske, and Wiess (2005) describe the WISC-III as a highly robust measure that retains its characteristics when used with individuals who have ADHD. Given the fact that the WISC-IV maintains many of the same subtests and a similar factor structure, Prifitera et al. (2005) contend that, in a manner similar to the WISC-III, the WISC-IV will prove to be a psychometrically sound instrument applicable for use in assessing ADHD children.

WISC-IV and ADHD

The WISC-IV continues to provide a reliable measure of global intelligence (FSIQ); it has also enhanced the measure of more discrete domains of functioning (i.e., processing speed and working memory) and changed the dual IQ and index score structure utilized in the WISC-III (J. Friedman, 2006). In addition to the FSIQ, the WISC-IV yields four index scores (Verbal Comprehension Index (VCI), Perceptual Reasoning Index (PRI), Processing Speed Index (PSI), and the Working Memory Index (WMI)). The VCI is composed of the Similarities (SI), Comprehension (CO), and Vocabulary (VO) subtests, which measure crystallized knowledge, verbal reasoning, comprehension, and conceptualization. The Information (IN) and Word Reasoning (WR) subtests are supplemental VC measures. The PRI is composed of the Block Design (BD), Matrix Reasoning (MR), and Picture Concepts (PCN) subtests, which measure perceptual reasoning and organization. A supplemental measure of perceptual reasoning, the Picture Completion (PCM) subtest, is also available. The WMI is composed of the Digit Span

(DS) and Letter Number Sequencing (LNS) subtests, which are purported to measure attention, concentration, and working memory. The Arithmetic (AR) subtest is a supplemental measure of working memory. Finally, the PSI is composed of the Coding (CD) and Symbol Search (SS) subtests, which measure the speed of mental and graphomotor processing. The Cancellation (CA) subtest, a supplemental measure of PS, is also available on the WISC-IV.

The WISC-IV reflects the current status of intelligence theory, recognizing that both global functioning and specific elements or abilities compose intelligence (J. Friedman, 2006) . In addition, there is increased emphasis on working memory, which reflects the perceived importance of this construct on learning and on overall cognitive functioning. With the revisions of the WISC-IV, the working memory and processing speed subtests now account for 4 of the 10 subtests included in the FSIQ calculations versus 2 of the 10 subtests on the WISC-III. Because of the increased weight of working memory and processing speed on the WISC-IV, the IQ scores of ADHD students could be negatively impacted.

As part of its initial standardization, the WISC-IV was administered to 89 children ages 8 to 13 identified as having ADHD according to DSM-IV-TR diagnostic criteria. The ADHD group included various subtypes (i.e., ADHD-IT, ADHD, HIT, and ADHD-CT). Of the ADHD subjects, approximately 64% were treated pharmacologically at the time of testing. Findings of the ADHD study included a moderate effect size for group mean difference for the PSI and small effect sizes for the VCI, WMI, and FSIQ. At the subtest level, the largest effect sizes for group mean differences occurred on the Coding and Arithmetic subtests. There were only modest differences on other working

memory and processing speed subtests. Small effect sizes for group mean score differences occurred on Digit Span, Letter-Number Sequencing, Symbol Search, and Cancellation.

Further analysis of the WISC-IV standardization study reveals, that relative to their matched controls, ADHD children had slightly lower mean FSIQ scores (97.6 versus 102.7). Although these differences are statistically significant (p = .01), the effect size (.38) is not large. These results support the previous studies that have found that ADHD children typically achieve scores near the normative range of intellectual functioning, but their performances may be worse on measures of processing speed and working memory than on measures of verbal comprehension and perceptual reasoning (Barkley, Murphy, & Bush, 2001; Doyle, Biederman, Seidman, Weber, & Faraone, 2000). Similar to findings with the WISC-III, children with ADHD who were administered the WISC-IV obtained their lowest index score on the PSI and lowest subtest score on Coding. ADHD children also performed lower than matched controls on the WMI, with their lowest WMI subtest score on Arithmetic. The WISC-IV Technical and Interpretive Manual (Wechsler, 2003) suggests that further research is needed to examine separate samples of ADHD based on clinical subtype, as well as studies comparing the performance of medicated versus nonmedicated ADHD children. Although data exists regarding the medication status of the ADHD sample utilized, it was not analyzed as part of the standardization study.

In 2006, Friedman conducted a study of the cognitive profiles of 109 students with ADHD, utilizing WISC-IV data. These subjects were matched and compared with non-ADHD controls. In keeping with the standardization data, Friedman did not find

significant differences among FSIQ scores in ADHD samples versus non-ADHD controls, but did find that the nonmedicated ADHD group performed significantly lower than their non-ADHD controls on the WMI. Lowest WMI scores for the ADHD group occurred on the Digit Span subtest. Unlike the WISC-IV standardization sample, no significant differences were found on measures of processing speed. Based on her study, Friedman indicated that further analysis is needed to look at VCI and WMI differences in ADHD children, because her data suggested that statistical differences may be present.

Stimulant Medication

The impaired frontal lobe functioning and abnormalities in the dopamine neurotransmitter system associated with ADHD (Preston, O'Neal, & Talaga, 2005) are the reasons why dopamine agonists (i.e., stimulant medications) have been identified as an efficacious intervention for reducing the symptoms of ADHD. In fact, medication is perhaps the most common form of intervention for children with ADHD in the United States. Of the 11 million prescriptions written for methylphenidate (Ritalin) each year, approximately 80% are for children (Purdie et al., 2002). Given the number of children with ADHD who receive psychopharmacological treatment, the effects of stimulant medication on various aspects of functioning are relevant to many clinicians and educators.

Additional support for the use of stimulant medications for ADHD comes from numerous research studies, the majority of which have found that stimulants are highly effective in treating both the executive and the behavior deficits of ADHD (Hale & Fiorello, 2004). In 1992, Thomson examined the responses of children with ADHD to stimulant medication. Overall, she found that measures of inattention and overactivity

are the best predictors of responses to a stimulant medication as determined by parent and teacher rating scales. Campbell (1991) examined the differences in cognitive and affective characteristics relative to responses to stimulant medication. Two groups (one with ADHD and one with undifferentiated ADD) were compared in a pre-test-post-test design. Baseline measures included IQ, impulsivity, problem behavior, self-reported depression, and self-esteem. After three months, medication response was measured. No significant differences were found between groups for IQ, impulsivity, depression, self-esteem, anxiety, peer relationships, and social withdrawal. The stimulant medication did decrease the hyperactive behaviors for the ADHD group. An improvement in the peer relationships and aggressive behaviors in the ADD group was seen as a result of the medication; however, these same benefits were not observed in the ADHD group. Additional studies by Pelham and Milich (1991) and DuPaul, Barkley, and McMurray (1994) have also found positive effects of stimulant medication on hyperactivity, attention, concentration, and classroom behavior.

Also important to note is the Multimodal Treatment Study of Children with ADHD (The MTA Cooperative Group, 1999). This study measured medication effects on ADHD symptomatology, representing the largest study of this kind to date. This study was a 14-month randomized clinical trial in which 579 children, aged 7 to 9.9 years who had ADHD-CT, were assigned to various treatment modalities including medication management, behavioral treatment, a combination of medication and behavioral management or community-based treatment. Similar to the aforementioned studies, this study found that students in the medication management or combined treatment group demonstrated greater improvements in hyperactive-impulsive symptoms than those in the

behavioral treatment alone or in community-based treatment groups. For other areas of functioning, such as academic achievement, few differences among treatment groups were found, except in the area of reading achievement assessed by the Wechsler Individual Achievement Test. This study supported the robust, short-term efficacy of medication management that has been found in other studies, and also extended these findings by demonstrating that the benefits persisted for up to 14 months.

Stimulant Medication and Cognitive Performance. The effect of stimulant medication on cognition and learning is less-well documented than the effect on behavior and varies according to the measure and methodology utilized. Hale and colleagues (1998) noted that a particular problem in the research literature is that many studies include heterogeneous participants with different types of attention deficits, in addition to ADHD group for analysis of MPH effects; this problem can obscure any robust results for individual children.

Barkley (1998) noted that the impact of medication upon behavior and concentration was most significant, but that performance on intelligence tests was less affected by medication. Brown and Borden (1989) reported that stimulant medication improves performance on rote or simple tasks, but that measures requiring the processing of higher-order information may be less influenced. Furthermore, a study by Livingston and colleagues (1996), in which the WISC-R and WISC-III scores for medicated and nonmedicated children and adolescents were compared, did not find significant differences in cognitive functioning between medicated and nonmedicated samples. Both the medicated and nonmedicated groups performed poorly on the Freedom from Distractibility Index, which included the Arithmetic, Digit Span and Coding subtests.

Based on their findings, Livingston et al. (1996) concluded that although studies have found that stimulant medication yields positive responses on overt ADHD symptomatology and laboratory tests of cognitive performance, there is little evidence of parallel long-term improvement on more traditional intellectual, neuropsychological, or achievement measures. To explain this discrepancy between positive short-term effects of psychostimulants on behavioral and cognitive functioning and lack of long-term improvements, several hypotheses were made. First, they cite methodological limitations such as lack of random assignment and intergroup differences as a possible confounding variable. Psychostimulants have been found to enhance the functioning of subcortical attention centers, but have limited impact on the information processing of cortical areas (S. Goldstein & Goldstein, 1990). Because the measures used in this and many other studies (Kagan, 2000; Saklofske, Schwean, & O'Donnell, 1996; Tannock, Martinussen, & Frijters, 2000) primarily assess cortical functioning, they would not detect enhanced subcortical functioning. One final hypothesis put forth by Livingston and colleagues is related to the homeostatic down-regulation of receptors at different rates across brain sites. Down-regulation is related to an inherent tendency to return to baseline or homeostasis, which occurs through neuronal adaptations. Chronic exposure to high levels of stress or certain medications causes a bombardment of certain excitatory receptors, which often leads to a reduction in the number and density of excitatory receptors, also known as down- regulation (Preston et al., 2005). Thus, after prolonged use of psychostimulants, the neural systems responsible for short-term cognitive gains may down- regulate; however, systems responsible for behavioral improvements may have limited down- regulation (Livingston, Mears, Marshall, Gray, & Haak, 1996).

Livingston and colleagues conclude that failure to document long-term improvement in neurocognitive abilities is not taken as evidence against the efficacy of psychostimulant medication for ADHD, but rather as evidence that ancillary interventions are also warranted.

There is a need for further study of ADHD individuals, both pre- and post-pharmacological intervention, in order to determine the impact of medication on test sensitivity (Doyle et al., 2000). Some studies that have examined the short-term effects of methylphenidate on the WISC-III have not revealed significant treatments effects for subtest, factor, and index scores (Saklofske & Schwean, 1993; Schwean, Saklofske, Yackulic, & Quinn, 1993). On the other hand, in 2003 Faraone reviewed the methods for comparing medications across studies and provided examples, explaining how to apply them to medicines utilized to treat ADHD. Using Cohen's (1988) Standard Mean Difference (SMD) to report efficacy in terms of continuous measurements, he calculated effect sizes for stimulants and nonstimulants in the treatment of ADHD. In general, he found greater effect sizes for stimulants than nonstimulants (.9 versus .6), with long acting stimulants having slightly larger effect sizes on IQ (as measured by the Wechsler scales). A small effect was defined as an increase in IQ of 3 points with the use of medication; an increase in 7.5 IQ points was considered a medium effect, and an increase of 12 points was considered a large effect size. Faraone found that the use of nonstimulants increased IQ by 9 points; stimulants, both immediate release and long-acting, increased IQ by 14 points.

Despite the large effect sizes for stimulant medication on intelligence tests found in the Faraone study, the literature on long-term results of stimulant medication on

cognitive functioning is relatively sparse. A long-term placebo-controlled study by Gillberg and colleagues (1997) found positive effects of amphetamine treatment of ADHD that remained 15 months after the start of treatment. This study included a comparison between WISC-R scores of students who had been taking the placebo for 6 months or more, with those who had been taking the amphetamine for 9 months or more. They found the mean change in IQ from 0 to 15 months for the group treated with amphetamines for 9 or more months to be + 4.5 points (SD, 4.7), compared with + 0.7 (SD, 7.2) in the placebo group. As they had predicted, the amphetamine group demonstrated a positive change in 28 of the 34 individuals, whereas in the placebo group only 4 of 8 showed improvement.

Some studies have attempted to measure the effects of methylphenidate (MPH) on specific areas of cognitive functioning known to be impacted by ADHD. These studies have focused on the construct of working memory. Research in this area is conflicting; some studies show that MPH improves both visual-spatial and auditory-verbal working memory (i.e., Bedard et al., 2004; Mehta, Goodyer, & Shahakian, 2004; Tannock, Ickowicz, & Schachar, 1995; Zeiner at al., 1999), but others have not (i.e., Rhodes, Coghill, & Matthews, 2004; 2006). Most of these studies, however, measured only one aspect of the four identified dimensions of working memory, utilizing only a single measure. In order to gain a more comprehensive examination of the effects of MPH on working memory, Bedard and colleagues (2007) focused on all four dimensions of working memory. Specifically, they investigated both the modality (auditory-verbal or visual-spatial) and processing (storage and manipulation) components of working memory in school-aged children with ADHD. Major findings were that MPH had

selective beneficial effects on working memory. Although it improved the ability to store visual-spatial information, it did not impact auditory-verbal storage. MPH also improved the ability to manipulate both visual-spatial and auditory-verbal information; however, improvements were dependent upon the measure utilized to index working memory manipulation. For example, MPH had a positive effect on visual-spatial manipulation on the Finger Windows Backward subtest, but not on the Spatial Span Backward subtest. MPH had a positive impact on the Letter Span Backward subtest, which was considered a more effortful task, but not on the Digit Span Backward subtest.

Bedard and colleagues noted in their finding, that MPH has differential effects on the storage of visual-spatial and auditory-verbal information is consistent with evidence of lateralization of visual-spatial storage, attentional dysfunction in ADHD, and of MPH effects. They linked their findings with those of Fletcher and Henson (2001), who hypothesized that auditory-verbal storage tasks are left lateralized but that visual-spatial are right lateralized in the brain. They further cited the evidence (i.e., Bellgrove et al., 2005; Hermens et al., 2005; Sangal & Sangal, 2004) for left-sided inattention in ADHD associated with right hemisphere dysfunction that responds well to MPH, stipulating that MPH has selective effects on right neural networks supporting visual-spatial storage, but not auditory-verbal storage. They note that additional research is needed using neuroimaging and in examining different types of auditory-verbal and visual-spatial non-span tasks which may recruit very different neural networks.

The WISC-IV standardization study did not analyze differences in ADHD samples by treatment, although Prifitera and colleagues (2005) predicted that it would be unlikely that WISC-IV performance would be affected by medication, considering the

lack of treatment effects found with medication use in ADHD samples on WISC-III performance. Schwean and Saklofske (2005), however, suggested that the WISC-IV has considerable clinical value for monitoring cognitive changes of paramount importance in determining the efficacy of medical, psychological, and educational programs.

In 2006, Friedman examined differences in FSIQ, Index scores, and Subtest scores among medicated and nonmedicated ADHD samples and matched controls selected from the WISC-IV normative sample. Friedman found no differences in overall IQ between groups, but did find that the nonmedicated ADHD group had significantly lower Working Memory Index scores than their matched controls. At the subtest level, Digit Span scores were found to be significantly lower in the nonmedicated ADHD sample than both the medicated ADHD group and non-ADHD controls.

Research Questions

The present study will replicate and expand on Friedman's (2006) study which examined mean differences between children with ADHD and non-ADHD controls on a measure of cognitive functioning, as well as compared mean differences between medicated ADHD students and non-medicated ADHD students. Friedman's (2006) study was an expansion of work completed during the standardization of the WISC-IV. In the present study, Friedman's hypotheses will be evaluated with the current data set. In addition, Index score splits will be examined to determine whether or not ADHD students display a significantly different cognitive profile from non-ADHD students. Although Friedman's (2006) comparison of Index and Subtest scores between the ADHD group and non-ADHD group yielded significant differences that were limited to the Working Memory Index and Digit Span subtest, it is possible that utilizing group means concealed

some important intra-individual differences that could demonstrate diagnostic utility. In addition, the current study will examine the Index score splits of individuals with ADHD who are medicated, versus individuals with ADHD who are not medicated in an effort to explore further the impact of medication on cognitive performance. Results of research conducted to explore the effects of medication on cognitive performance are equivocal, and thus further investigation in this area is needed. This information could have implications both for the medical and for the educational treatment of ADHD.

Research Hypotheses

The first set of research hypotheses are designed to replicate Friedman's (2006) study. These research questions and hypotheses are as follows:

1. What is the impact of ADHD diagnosis on FSIQ scores of the WISC-IV?
    a. It is predicted that results of this study will be consistent with results from Friedman's (2006) study, which did not find significant differences between the mean FSIQ scores of the ADHD subjects and their non-ADHD counterparts.
2. What is the impact of ADHD diagnosis of factor scores (VCI, PRI, WMI, and PSI) of the WISC-IV?
    a. In line with Friedman's (2006) findings, it is predicted that there will be no statistically significant differences between the ADHD groups and their non-ADHD counterparts on the VCI, PRI, or PSI. It is predicted that there will be a significant difference between the ADHD groups and non-ADHD groups on the WMI. This difference is predicted to be more

significant for the nonmedicated group of ADHD subjects than for the non-ADHD groups and the medicated ADHD group.

3. How do ADHD individuals perform relative to non-ADHD individuals on each subtest of the WISC-IV?
    a. Consistent with Friedman's (2006) finding, it is predicted that the mean scores of ADHD students will be comparable with the mean scores of their non-ADHD counterparts on the Verbal Reasoning subtests (VO, CO, SI), Perceptual Reasoning subtests (BD, MR, and PCN), and Processing Speed subtests (CD and SS). Some differences are expected between the ADHD groups and their non-ADHD controls on the working memory subtests (DS and LNS).
4. How does medication status impact WISC-IV FSIQ scores of students with ADHD?
    a. Consistent with Friedman's (2006) findings, no differences are predicted between the mean scores of the ADHD medicated group and the ADHD nonmedicated group.
5. How does medication status impact ADHD students' performance on the WISC-IV Indices?
    a. Friedman (2006) did not find any significant differences between the medicated ADHD students and nonmedicated ADHD students for any of the WISC-IV Indices. It is predicted that the current study will also demonstrate comparable mean performances on the WISC-IV Indices between the ADHD medicated and nonmedicated groups.

6. How does medication status impact the performance of ADHD students on the core subtests of the WISC-IV?
    a. It is predicted that this study will replicate Friedman's results. Specifically, no significant differences are expected between the mean subtests scores of the ADHD medicated and nonmedicated groups for any of the 10 core subtests of the WISC-IV.

A second set of research questions specific to the current study will also be investigated. The questions and hypotheses listed below are based on the reviewed literature that overwhelmingly suggests that executive control deficits related to working memory and processing speed are a core feature of ADHD and related to neuropsychological factors.

7. How do individuals with ADHD perform on cognitive measures of working memory relative to their performance on measures of other cognitive abilities? This question will be explored by comparing the Working Memory Index and Verbal Comprehension Index score splits between the ADHD groups and their non-ADHD matched controls and by comparing the Working Memory Index and General Ability Index score splits for the ADHD groups versus their non-ADHD controls. The GAI is a sum of VCI and PRI scores and provides an overall measure of reasoning abilities.
    a. It is predicted that the ADHD groups will have a larger number of cases with VCI scores greater than WMI scores relative to ADHD controls.

b. It is also predicted that there will be a larger number of cases in the ADHD groups with GAI scores greater than WMI scores, relative to non-ADHD controls.

8. How do individuals with ADHD perform on cognitive measures of processing speed, relative to their performance on measures of other cognitive abilities? This question will be explored by comparing splits between Processing Speed Index and Perceptual Reasoning Index scores in ADHD groups versus their non-ADHD controls. The General Ability Index – Processing Speed Index score splits will also be compared among groups.

   a. It is predicted that there will be a greater number of cases in the ADHD groups with PRI scores greater than PSI scores, relative to non-ADHD controls.

   b. It is also predicted that there will also be a larger number of cases in the ADHD groups with PRI scores greater than PSI scores, relative to non-ADHD controls.

In addition, this study seeks to find if there are differences in the cognitive profiles of children with ADHD who are medicated versus those who are not. To evaluate this, the following research questions and hypotheses will be studied.

9. To what extent does stimulant medication impact the working memory capacity of ADHD children? This question will be explored by comparing the Working Memory and Perceptual Reasoning Index score splits between the medicated ADHD individuals and the non-medicated ADHD individuals. General Ability Index and Working Memory score splits will also be compared among groups.

a. It is predicted that the nonmedicated ADHD group will display more cases of VCI scores greater than WMI scores, relative to the medicated ADHD group.
b. It is predicted that the nonmedicated ADHD group will also display more cases of GAI scores greater than WMI scores, relative to the medicated ADHD group.

10. To what extent does stimulant medication impact processing speed in ADHD children? This question will be explored by examining the Processing Speed Index score versus measures of other cognitive abilities for medicated ADHD individuals versus non-medicated ADHD individuals.
    a. It is predicted that there will be a higher number of cases in the nonmedicated ADHD group with PRI scores greater than PSI scores, relative to the medicated ADHD group.
    b. It is predicted that there will be a larger number of cases in the nonmedicated ADHD group with GAI scores greater than PSI scores, relative to the medicated ADHD group.

Chapter 2

Method

Participants

The data utilized in the current study was archival in nature. Specifically, test data archived in public school files were accessed to obtain the WISC-IV scores of 111 male students between the ages of 8 and 16, who had been diagnosed with ADHD by a physician or psychologist, and had been tested as part of the school district's educational referral process. Archived public school data was obtained from a northeastern region of the United States that included New Jersey, Pennsylvania, Delaware, Maryland, and Connecticut. Data on students identified as having comorbid disabilities were not systematically excluded from this study. In addition, students with all three subtypes of ADHD (ADHD-IT, ADHD-HIT, and ADHD-CT) were included in the study.

The ADHD data was divided into two groups. The first group consisted of test data from a group of 62 students not being treated pharmacologically for ADHD (i.e., the nonmedicated group). In order to be assigned to this group, students met the following criteria: (1) diagnosis of ADHD, (2) a WISC-IV Verbal Comprehension Index (VCI) Standard Score greater than 80, and (3) indication that medication prescribed for the symptoms of ADHD was not being taken at the time of the WISC-IV testing. The second group consisted of test data from a group of 49 students who were being medicated for ADHD at the time of assessment. Assignment to this group included the following criteria: (1) diagnosis of ADHD, (2) a WISC-IV VCI Standard Score greater than 80, (3) indication that medication prescribed for the symptoms of ADHD was being taken at the

time of WISC-IV testing. Information regarding medication status was based on parent report or information from the student's file. Students taking stimulants, nonstimulants or combinations of medication were included in this study.

In addition to the ADHD group, data was collected for a non-ADHD sample. This non-ADHD sample was obtained from The Psychological Corporation's WISC-IV standardization sample. This non-ADHD group was divided into two groups. One group consisted of 62 non-ADHD subjects matched as closely as possible with the ADHD non-medicated group on the basis of chronological age, gender, ethnicity, parent education level (when available), geographic region, and Verbal Comprehension Index. The second group of 49 subjects was matched with the ADHD medicated group on the same variables.

Confidentiality was assured by removing identifying information such as name and date of birth. Only archived data were utilized. Information was collected using data collection forms, which were secured in a locked file cabinet. Test scores and protocols collected by the examiner were protected from unauthorized release and access. The medicated and non-medicated groups were predetermined by parental choice; therefore, the withholding of treatment was not an issue for this study. Test scores were interpreted with consideration to contextual and cultural variables, as well as to the limitations of current research and practice related to ADHD.

Variables

Independent Variables. The two independent variables included in this study involve: ADHD diagnostic status (ADHD or non-ADHD) and treatment status (ADHD medicated or ADHD nonmedicated).

Dependent Variables. Dependent variables included the WISC-IV Index scores (i.e., VCI, PRI, WMI, PSI, and FSIQ) and the differences or splits between factor scores (i.e., VCI and WMI; GAI and WMI; PRI and PSI; GAI and PSI; VCI and PRI; and PSI and WMI).

Overview of the Research Design

Participants were assigned to groups based on diagnosis and treatment status. Mean scores for each of the WISC-IV FSIQ, Index scores, and subtest scores were computed. General Ability Index (GAI) scores were also calculated for all groups. Mean differences between WISC-IV Index scores (i.e., VCI and WMI; GAI and WMI; PRI and PSI; GAI and PSI; VCI and PRI; and PSI and WMI) were then calculated for all groups (ADHD medicated group; ADHD nonmedicated group; and the two non-ADHD matched control groups) and were compared.

Measure and Procedure. School psychologists of selected schools were sent a letter requesting participation in the study. Those who opted to participate received permission from their school districts and signed letters of agreement. The school psychologists were then asked to record WISC-IV test scores and demographic information from ADHD students' records on a data collection form. The information requested on the data collection form included the following: raw and standard scores of the 10 core WISC-IV subtests, as well as index scores; chronological age of the child; gender; ethnicity; parent education level; diagnosis; treatment status; brand name of medication; dosage and time of medication treatment; ADHD subtype; and additional diagnoses. Raw scores were requested so that the accuracy of the reported standard scores could be checked.

The WISC-IV subtest and composite score data are considered interval data and allow for the comparison of the four groups (medicated, nonmedicated, and two control groups) among several variables (mean Index scores, mean Subtest scaled scores and index score differences).

The WISC-IV, with over 60 years of research to support its practical and clinical utility (Sattler, 2001), is considered a valid and reliable instrument with sufficient test sensitivity to assess the constructs of working memory and processing speed. The theoretical basis of the Wechsler Scales is further supported by its high correlation with other measure of cognitive abilities, as well as by the appearance of similar subtests on other measures of intelligence (Wechsler, 2003).

Statistical Analysis

Control groups were matched to the ADHD groups as closely as possible on demographic variables of age, parent education level, ethnicity, and the Verbal Comprehension Index so that there would be no significant differences on these variables between the controls and their ADHD counterparts. It is noteworthy that parent education level was not available for 47 of the ADHD cases.

The ADHD sample was divided into two groups. The first group consisted of the students who were being treated pharmacologically for their ADHD symptoms (medicated group) at the time of testing, and the second consisted of children who were not being treated pharmacologically for ADHD (nonmedicated group). Two non-ADHD control groups were then created by selecting samples from the archived WISC-IV standardization data set obtained from the Psychological Corporation. The first non-ADHD sample (Control 1) was matched with the ADHD medicated group, and the

second non-ADHD sample (Control 2) was matched with the ADHD nonmedicated group. Only subjects residing in the northeast or north central regions of the country were utilized for the control groups.

Hypotheses Tests

To test the first hypotheses regarding mean FSIQ differences between ADHD groups and non-ADHD groups, Univariate Analysis of Variance (ANOVA) was utilized. ANOVA allows for the comparison of multiple groups on one dependent variable. To test hypotheses relating to mean differences between WISC-IV Index scores and Subtest scores, Multivariate Analysis of Variance (MANOVA) was utilized. MANOVA allows for multiple groups to be compared among multiple dependent variables simultaneously to minimize the Type I error that could result from conducting multiple tests of significance among the dependent variables.

To test the second set of hypotheses involving the ADHD samples and their matched controls, new variables representing Index score splits were calculated. Cumulative percentages were then obtained for splits of 10, 15, 20, 25, 30 and 35 points for the following: VCI > WMI; WMI > VCI; GAI > WMI; WMI > GAI; PRI > PSI; PSI > PRI; GAI > PSI; PSI > GAI; VCI > PRI: PRI > VCI; PSI > WMI; and WMI > PSI. The cumulative percentages were then utilized to calculate n values at each level in order to use Fisher's Exact Test to test the hypotheses. Fisher's Exact Test allows for a comparison of the proportions between groups. Fisher's Exact Test is similar to Chi-Square. It calculates an exact probability value for the relationship between two dichotomous variables. This statistic calculates the difference between the data expected and the data observed, relative to the given marginal and assumptions of the model of

independence. This statistic has advantages over the Chi-Square statistic, which gives only an estimate of the true probability value, which may not be very accurate when the marginal is uneven or should there be a small value in one of the cells (Uitenbroek, 1997). Specifically, Fisher's Exact Test was utilized to compare the proportions of subjects at each level of difference (10, 15, 20, 25, 30 and 35 points) for each of the Index score splits between the ADHD groups and the non-ADHD groups. Fisher's Exact Test was calculated using Wang's online significance test for comparing two proportions (Wang, 1996).

Chapter 3

Results

The results of the statistical tests described in Chapter 2 are presented in this chapter, including the final composition of the sample, the statistical analysis utilized to the test the hypotheses, and the results of the data analyses. Data was initially entered into a Microsoft Excel spreadsheet and the exported to an SPSS file. Data were analyzed using the Statistical Package for the Social Sciences (SPSS- 16.0). The significance level for testing hypotheses was set at .05, although SPSS-16.0 reports significance at all levels. As mentioned earlier, Wang's (1996) online significance test for comparing two proportions was utilized for the Fisher's Exact Test calculations.

Demographic Information

The ADHD sample for this study consisted of 111 male students between the ages of 8 and 16 who had been diagnosed with ADHD and tested with the WISC-IV as part of a school district's educational referral process. The first group consisted of 62 ADHD male students who were not being medicated for their ADHD. The second group consisted of 49 ADHD male students who were being medicated for their ADHD symptoms. The brand name of the medication was available for 46 (94%) of the cases. The largest proportion of cases took stimulant medications, with Concerta and Adderall being the most frequently prescribed. The brand names of the ADHD medications were as follows: 19 students were taking Concerta (29%); 9 students were taking Adderall (14.5%); 4 students were taking Ritalin (6.5%); 4 students were taking Straterra (6.5%); 4 students were taking Focalin (6.5%) and 2 students took Daytrana (3.2%). Other less common medications included Depakote, Abilify, Tegretol, Seroquel, and Risperdol.

Four of the subjects were prescribed more than one medication (i.e., Focalin and Abilify; Seroquel and Tegretol; Concerta and Depakote; and Straterra and Focalin). Only 14 of the data forms included information regarding whether or not the medication was immediate release or long-acting. Of those 14 cases, 5 were taking immediate release medication, 8 were taking long-acting medications, and 1 was taking a combination of both. The time of medication treatment was reported for 15 cases. Of these 15 cases, 12 took medication in the morning, 1 took medication in the afternoon, and 3 took medication in both the morning and afternoon.

Two non-ADHD samples were then selected to serve as matched controls. These samples were taken from the archived WISC-IV standardization data set that was obtained from the Psychological Corporation. The non-ADHD sample consisted of 111 male students between the ages of 8 and 16, residing in the northeast regions of the country. The non-ADHD sample was divided into two groups. The first group consisted of 62 males and was matched to the nonmedicated ADHD group (Control 1). A second group of 49 males was matched to the ADHD medicated group (Control 2). As mentioned previously, the control groups were matched as closely as possible on the basis of chronological age, gender, ethnicity, parent education level, and Verbal Comprehension Index Standard Score.

The largest proportion both of the nonmedicated and of the medicated ADHD groups were age 8. Over one- half of the children in both groups were age 10 or younger. The controls were matched as closely as possible with their non-ADHD counterparts on chronological age. Exact matches were not possible for a few subjects; this varied by one to two years, at most. Table 1 provides the frequency distributions for chronological age

in each of the four groups (ADHD Nonmedicated, Matched Control 1, ADHD Medicated, and Matched Control 2).

Table 1

Frequency Distribution for Age

		Age								
Group		8	9	10	11	12	13	14	15	16
ADHD Nonmedicated										
	n	14.00	11.00	12.00	5.00	4.00	7.00	5.00	2.00	2.00
	%	22.60	17.70	19.40	8.10	6.50	11.30	8.10	3.20	3.20
Control 1[a]										
	n	14.00	11.00	11.00	5.00	4.00	8.00	4.00	3.00	2.00
	%	22.60	17.70	17.70	8.10	6.50	12.90	6.50	4.80	3.20
ADHD Medicated										
	n	13.00	5.00	10.00	3.00	3.00	4.00	6.00	3.00	2.00
	%	26.50	10.20	20.40	6.10	6.10	8.20	12.20	6.10	4.10
Control 2[b]										
	n	6.00	12.00	7.00	6.00	3.00	5.00	7.00	1.00	2.00
	%	12.20	24.50	14.30	12.20	6.10	10.20	14.30	2.00	4.10

Note. Age range from 8 to 16 years.

[a]Control 1 = non-ADHD group matched to ADHD nonmedicated group

[b] Control 2 = non-ADHD group matched to ADHD medicated group

Parent Education Level ranged from 0-8 years up to a college or graduate degree. Parent Education Levels were not reported for 47 of the ADHD cases; therefore, exact matches could not be made and were estimated as best as they could possibly be. The frequency distributions for Parent Education Level by group are provided in Table 2.

Table 2

Frequency Distribution for Parent Education Level

		Parent Years of Education				
Group		0-8	9-11	12	13-15	16+
ADHD Nonmedicated						
	n	0.00	2.00	16.00	8.00	9.00
	%	0.00	3.20	25.80	12.90	14.50
Control 1						
	n	2.00	6.00	24.00	15.00	15.00
	%	3.20	9.70	38.70	24.20	24.20
ADHD Medicated						
	n	0.00	2.00	9.00	5.00	13.00
	%	0.00	4.10	18.40	10.20	26.50
Control 2						
	n	1.00	4.00	12.00	14.00	18.00
	%	2.00	8.20	24.50	28.60	36.70

Note. 0-8 = eighth grade education or less; 9-11 = some high school; 12 years = high school or equivalent; 13-15 years = some college or associate degree; 16 or more years = college or graduate degree. Matched Control 1 = ADHD nonmedicated control; matched Control 2 = ADHD medicated control.

Parents of the nonmedicated ADHD children most frequently reported as having a high school diploma (25.8%), but the medicated group most frequently reported having a college or graduate degree (26.5%). Parent education levels were slightly higher among the medicated group, with 36.7% with parent education levels falling in the some college to college/ graduate degree range, and 26.4% of the nonmedicated group had parent education levels in that range. The distributions reported for these groups appear similar to those reported in the literature (NSCH, 2003).

The ADHD groups were also matched with controls on the basis of ethnicity. Table 3 is a summary of the ethnicity for each of the four groups.

Table 3

Frequency Distribution for Ethnicity

	Ethnicity			
Group	White	Black	Hispanic	Other
ADHD Nonmedicated				
n	46.00	9.00	5.00	2.00
%	74.20	14.50	8.10	3.20
Control 1				
n	43.00	9.00	9.00	1.00
%	69.40	14.50	14.50	1.60
ADHD Medicated				
n	39.00	5.00	5.00	0.00
%	79.60	10.20	10.20	0.00
Control 2				
n	35.00	7.00	6.00	1.00
%	71.40	14.40	12.20	2.00

Finally, the ADHD groups were matched with controls by Verbal Comprehension Index scores. VCI scores of the ADHD groups ranged from a low of 80 to a high of 155. The ADHD children with VCI scores below 80 were not included in the study. VCI means and standard deviations of the four groups are included in Table 4.

Table 4

Verbal Comprehension Index Scores by Group

Group	*M*	*SD*
ADHD Nonmedicated	96.37	11.72
Control 1	96.58	11.58
ADHD Medicated	101.71	14.92
Control 2	101.24	13.77

Statistical Analysis

The control groups were matched with the ADHD groups on the demographic factors of age, parent education levels, ethnicity, and the Verbal Comprehension Index, to the degree that no significant differences were found among these variables between the controls and their ADHD counterparts. Prior to statistical analysis, the Levene Statistic was utilized to determine the extent to which the data met the assumptions required for appropriate use of parametric inferential statistical tests of significance. The data did meet the assumption of homogeneity of group variances for all study variables. No significant differences were found among the variable variances of the ADHD groups and matched controls, which allowed for the use of parametric inferential statistical procedures.

Hypotheses Tests

To test the hypotheses comparing the ADHD with non-ADHD controls and their mean performances on different indices and subtests, three separate analyses were utilized:

1. A one-way ANOVA investigated the differences among the mean FSIQ scores of the medicated and nonmedicated ADHD groups, and the ADHD groups and their matched controls.
2. A MANOVA was conducted to test the research question of whether or not there were significant differences between the mean VCI, PRI, WMI, and PSI scores of the medicated and nonmedicated ADHD groups and between the ADHD groups and their matched controls.
3. A MANOVA was also conducted to test the research questions related to whether or not there were significant differences between the medicated and nonmedicated ADHD groups and the ADHD groups and their matched controls for each of the 10 WISC-IV core subtests (SI, CO, VC, BD, MR, PCn, DS, LNS, CD, and SS).

To test the second group of hypotheses related to whether or not students with ADHD display weaknesses in working memory and processing speed, relative to their other cognitive abilities, and also to whether or not the use of medication impacts working memory and processing speed splits, the following procedures were utilized:

1. New variables were created to represent Index score splits. These variables included: VCI-WMI difference, GAI-WMI difference, PRI-PSI difference, and GAI-PSI difference.

2. The cumulative percentage of subjects in each group with VCI-WMI, GAI-WMI, PRI-PSI, and GAI-PSI splits of 10, -10, 15, -15, 20, -20, 25, -25, 30, -30, 35, and -35 points was recorded.
3. N values were then calculated at each level for each group.
4. Fisher's Exact Test was then utilized to analyze differences in proportions at each level between the ADHD groups and their matched controls (i.e., nonmedicated ADHD group and Control Group 1, and medicated ADHD group and Control Group 2), and between the nonmedicated ADHD group and medicated ADHD group.

Results of Hypotheses Tests

The FSIQ, Index, and Subtest means and standard deviations of the four groups are listed in Tables 5 and 6.

Table 5

Index Mean Score by Group

	Diagnostic Group							
Index Scores	ADHD Non-Med (N=62)		Control 1 (N=62)		ADHD Med (N=49)		Control 2 (N=49)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
VCI	96.37	11.71	96.58	11.58	101.71	14.92	101.24	13.77
PRI	96.34	14.65	101.68	14.75	101.51	15.56	104.14	13.91
WMI	92.23	12.38	98.11	12.74	96.37	16.05	104.41	16.89
PSI	92.35	11.46	98.65	16.12	92.63	14.46	101.16	13.03
FSIQ	93.27	11.96	98.52	13.24	98.39	15.29	103.63	14.60

Table 6

Subtest Scores by Group

	Diagnostic Group							
	ADHD Non-Med		Control 1		ADHD Med		Control 2	
Index Scores	(N=62)		(N=62)		(N=49)		(N=49)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
SI	10.06	2.70	9.32	2.72	11.06	3.08	10.55	2.64
VO	9.00	2.66	9.79	2.49	10.12	2.76	10.71	2.67
CO	9.13	2.29	9.19	2.42	9.94	2.98	9.61	2.78
BD	9.19	2.99	10.18	2.89	10.04	2.84	10.59	2.79
PCN	9.69	3.39	10.45	3.05	10.18	3.23	10.53	2.73
MR	9.03	3.31	10.16	2.89	10.45	3.04	10.84	3.09
DS	9.10	2.47	9.73	2.70	9.49	3.11	10.82	3.15
DSF	9.17	2.96	10.11	3.03	9.79	3.16	10.86	3.04
DSB	9.74	2.00	9.44	2.63	9.58	3.06	10.51	2.99
LNS	8.40	2.96	9.84	2.75	9.41	3.42	10.94	3.31
CD	8.32	2.76	9.63	3.10	8.08	2.57	9.57	2.52
SS	8.98	2.18	9.87	3.19	9.00	3.44	10.73	2.68

Tests of Hypotheses 1 and 4. An ANOVA was utilized to evaluate whether or not there were any significant differences between the mean FSIQ scores of any of the four groups. The results of this test revealed that there were significant differences in mean FSIQ scores between groups (F (3, 218) = 5.28, p = .002). See Table 7 for the ANOVA results.

Table 7

Analysis of Variance for FSIQ

SS	*df*	*MS*	*F*	*Sig.*	η^2
2957.950	3	985.983	5.280	0.002	0.067

Given the fact that the results of the initial ANOVA revealed statistical differences, post hoc analysis was conducted, utilizing Tukey's Honestly Significant Difference (Tukey HSD), with the significance level set at $p < .05$. Table 8 contains the results of post hoc analysis.

Table 8

FSIQ Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	5.11	2.612	0.207
ADHD nonmed vs. Control 1	-5.24	2.454	0.145
ADHD med vs. Control 2	-5.24	2.761	0.231

Post hoc results did not find any significant differences in mean FSIQ scores between the medicated and nonmedicated ADHD groups; between the nonmedicated ADHD groups and their matched controls, or between the medicated ADHD groups and their matched controls. The only significant difference that did occur was between the nonmedicated ADHD group and Control Group 2 ($p = .002$). Control Group 2 was the control group for the medicated ADHD group and therefore this comparison is not meaningful to the research questions of this study.

Tests of Hypotheses 2 and 5. Research questions 2 and 5, which were related to the WISC-IV Index scores, were analyzed with a Multivariate Analysis of Variance (MANOVA) to test multiple dependent variables (Index or Subtest scores) simultaneously. The MANOVA was utilized to minimize the Type 1 error that could result from conducting multiple tests of significance among the dependent variables. Significant differences found during the Multivariate Analysis of Variance were followed-up, using Tukey's HSD, with the significance level set at $p < .05$.

Table 9 contains the results of the MANOVA conducted to test hypotheses 2 and 5 for group mean differences on the dependent variables, VCI, PRI, WMI, and PSI.

Table 9

Multivariate Analysis for Index Scores

Index Scores	*SS*	*Df*	*MS*	*F*	*Sig.*	η^2
VCI	1377.302	3	459.101	2.753	0.043*	0.037
PRI	1851.481	3	617.164	2.846	0.039*	0.038
WMI	4149.065	3	1383.022	6.655	0.000*	0.084
PSI	3116.486	3	1038.829	5.385	0.001*	0.069

Note. Items with an asterisk are statistically significant.

The F tests of the group mean differences reveal significant differences between groups on all WISC-IV Indices (VCI, PRI, WMI, and PSI). Although statistical differences were found, the effect sizes were small for each factor.

The follow-up multiple comparisons of VCI, PRI, WMI, and PSI mean scores among the four groups are shown in Tables 10, 11, 12, and 13.

Table 10

VCI Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	5.34	2.468	0.136
ADHD nonmed vs. Control 1	-2.1	2.319	1.000
ADHD med vs. Control 2	0.47	2.69	0.998

Although the Multivariate Analysis of Variance was significant for VCI differences, follow-up tests of significance, which were conducted to test pair-wise comparisons among group means on the VCI, revealed no statistically significant differences among the medicated and nonmedicated ADHD groups; among the nonmedicated ADHD group and their matched controls, or among the medicated ADHD group and their controls. Cross comparisons among non-related groups (i.e., nonmedicated ADHD group versus Control Group 2; medicated ADHD group versus Control Group 1; and Control Group 1 and Control Group 2) also did not reveal statistically significant differences.

Table 11

PRI Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	5.17	2.815	0.259
ADHD nonmed vs. Control 1	-5.34	2.645	0.184
ADHD med vs. Control 2	-2.63	2.975	0.813

Multivariate Analysis of Variance was significant for PRI mean differences among groups; therefore, post hoc analyses were conducted to test pair-wise comparisons among group means on the PRI. These analyses, using Tukey's HSD revealed no statistically significant differences among the medicated and nonmedicated ADHD groups; among the nonmedicated ADHD group and their matched controls; or among the

medicated ADHD group and their controls. The only statistically significant difference that occurred was between the nonmedicated ADHD group and the matched controls for the medicated ADHD group, a difference that is not relevant to the research questions proposed in this study.

Table 12

WMI Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	4.14	2.755	0.437
ADHD nonmed vs. Control 1	-5.89	2.589	0.107
ADHD med vs. Control 2	-8.04	2.912	0.032*

Note. Items with an asterisk are statistically significant.

Follow-up multiple comparisons of WMI mean differences, using Tukey's HSD reveals a statistically significant difference between the ADHD medicated group and their matched controls. There were no significant differences between the medicated and nonmedicated ADHD groups and the nonmedicated ADHD group and their controls. Cross comparisons among non-related groups (i.e., nonmedicated ADHD group versus Control Group 2; medicated ADHD group versus Control Group 1, and Control Group 1 and Control Group 2) revealed statistical differences between the nonmedicated ADHD group and Control Group 2.

Table 13

PSI Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	0.28	2.655	1.000
ADHD nonmed vs. Control 1	-6.29	2.495	0.059
ADHD med vs. Control 2	-8.53	2.806	0.014*

Note. Items with an asterisk are statistically significant.

Post hoc tests of significance, using Tukey's HSD revealed statistical differences in mean PSI scores between the nonmedicated ADHD group and their matched controls and between the medicated ADHD group and their controls. There was not a statistical difference between the medicated and nonmedicated ADHD groups. Cross comparisons among non-related groups (i.e., nonmedicated ADHD group versus Control Group 2; medicated ADHD group versus Control Group 1, and Control Group 1 and Control Group 2) revealed a statistical difference between the nonmedicated ADHD group and Control Group 2.

Tests of Hypotheses 3 and 6. To test the hypotheses (3 and 6) involving differences between groups on each of the 10 core WISC-IV subtests, a Multivariate Analysis of Variance (MANOVA) was conducted to test the multiple dependant variables. Significant differences found during the multivariate analysis of variance were followed-up, using Tukey's HSD, with the significance level set at $p < .05$. Results of the MANOVA are included in Table 14.

Table 14

Multivariate Analysis for Subtest Scores

Subtest Scores	*SS*	*Df*	*MS*	*F*	*Sig.*	η^2
SI	91.199	3	30.400	3.939	0.009*	0.051
VO	85.253	3	28.416	4.088	0.008*	0.053
CO	23.392	3	7.797	1.154	0.328	0.016
BD	59.231	3	19.744	2.375	0.071	0.032
PCN	25.196	3	8.399	0.862	0.462	0.012
MR	103.100	3	34.367	3.603	0.014*	0.047
DS	85.497	3	28.499	3.531	0.016*	0.046
LNS	181.914	3	60.638	6.349	0.000*	0.080
CD	108.509	3	36.170	4.717	0.003*	0.061
SS	108.714	3	36.238	4.332	0.005*	0.056

Note. Items with an asterisk are statistically significant.

The MANOVA revealed statistically significant differences between mean group Subtest scaled scores on the SI, VO, MR, DS, LNS, CD and SS subtests. Although statistical differences are found, effect sizes are again relatively small. Follow-up post hoc analysis was conducted to evaluate further the nature of the statistically significant differences. The results of these analyses are reported in Tables 15, 16, 17, 18, 19, 20, and 21.

Table 15

SI Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	1.00	0.531	0.241
ADHD nonmed vs. Control 1	0.74	0.499	0.447
ADHD med vs. Control 2	0.51	0.561	0.800

Although the MANOVA was significant for group differences in mean SI subtest score, post hoc analysis did not reveal any significant differences between the medicated ADHD and nonmedicated ADHD groups; the nonmedicated ADHD groups and their matched controls, or between the medicated ADHD group and their matched controls. The only significant difference in mean SI subtest score occurred between the medicated ADHD group and Control Group 1, the control group matched to the nonmedicated ADHD group ($p = 0.007$).

Table 16

VO Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	1.12	0.504	0.119
ADHD nonmed vs. Control 1	-0.79	0.474	0.343
ADHD med vs. Control 2	-0.59	0.533	0.683

The MANOVA also revealed statistically significant differences in the mean VO subtest score among groups. Therefore, post hoc analyses were conducted to explore further the nature of these differences. Results of post hoc analysis, using Tukey's HSD did not reveal significant differences in mean VO subtest score between the medicated and nonmedicated ADHD groups; between the nonmedicated ADHD group and their matched controls, or between the ADHD medicated group and their matched controls. The only significant difference occurred between the nonmedicated ADHD group and Control Group 2, the matched controls for the medicated ADHD group, (p = 0.004).

Table 17

MR Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	1.42	0.590	0.080
ADHD nonmed vs. Control 1	-1.13	0.555	0.178
ADHD med vs. Control 2	-0.39	0.624	0.925

The MANOVA revealed statistical differences in the mean MR subtest score among groups. Post hoc analyses were conducted to explore further the nature of these differences. Results of Tukey's HSD analysis did not reveal significant differences in mean MR subtest score between the medicated and nonmedicated ADHD groups; between the nonmedicated ADHD group and their matched controls, or between the ADHD medicated group and their matched controls. The only significant difference

occurred between the nonmedicated ADHD group and Control Group 2, the matched controls for the medicated ADHD group ($p = 0.013$).

Table 18

DS Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	0.39	0.543	0.888
ADHD nonmed vs. Control 1	-0.63	0.510	0.607
ADHD med vs. Control 2	-1.33	0.574	0.099

The statistical differences in the mean DS subtest score among groups found in the initial MANOVA were followed up with post hoc analyses using Tukey's HSD. Results of the post hoc analysis did not reveal statistical differences in mean DS subtest score between the medicated and nonmedicated ADHD groups; between the nonmedicated ADHD group and their matched controls, or between the ADHD medicated group and their matched controls. The only significant difference occurred between the nonmedicated ADHD group and Control Group 2 , which were the matched controls for the medicated ADHD groups ($p = 0.009$).

Table 19

LNS Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	1.00	0.591	0.326
ADHD nonmed vs. Control 1	-1.44	0.555	0.050*
ADHD med vs. Control 2	-1.53	0.624	0.071

Note. Items with an asterisk are statistically significant.

Statistically significant differences between groups related to mean LNS subtest score were followed up with post hoc analysis, using Tukey's HSD. Results of post hoc analysis revealed statistical differences in mean LNS subtest scores between the nonmedicated ADHD group and their matched controls. No statistically significant differences occurred between the nonmedicated and medicated ADHD groups or between the medicated ADHD group and their matched controls. One other statistical difference occurred between the nonmedicated ADHD group and Control Group 2, the matched controls for the medicated ADHD group ($p < 0.01$).

Table 20

CD Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	-0.24	0.529	0.969
ADHD nonmed vs. Control 1	-1.31	0.497	0.045*
ADHD med vs. Control 2	-1.49	0.559	0.041*

Note. Items with an asterisk are statistically significant.

Statistically significant differences between groups for mean CD subtest score found in the initial MANOVA were followed up with post hoc analysis to determine the nature of the differences. Results of the post hoc analysis, using Tukey's HSD revealed statistically significant differences in mean CD subtest score between the nonmedicated ADHD group and their matched controls and between the medicated ADHD group and their matched controls. There was not a statistically significant difference in mean CD subtest score between the medicated and nonmedicated ADHD groups. Other statistically significant differences were found in a cross comparison of the medicated ADHD group and Control Group 1, the matched controls for the nonmedicated ADHD group ($p = 0.02$).

Table 21

SS Mean Difference Comparisons

Group Comparison	*M Dif.*	*SE*	*Sig.*
ADHD med vs. ADHD nonmed	0.02	0.553	1.000
ADHD nonmed vs. Control 1	-0.89	0.519	0.322
ADHD med vs. Control 2	-1.73	0.582	0.017*

Note. Items with an asterisk are statistically significant.

Statistical differences in mean SS subtest score identified from the initial MANOVA were followed up with post hoc analysis to explore the nature of these differences. Results of the post hoc analysis utilizing Tukey's HSD revealed statistically significant differences in mean SS subtest score between the medicated ADHD group and their matched controls. There were no statistically significant differences between the medicated and nonmedicated ADHD groups or between the nonmedicated ADHD group and their matched controls. Cross comparisons also revealed statistically significant differences in mean SS subtest scores between the nonmedicated ADHD group and Control Group 2, the matched controls for the medicated ADHD group ($p = 0.009$).

A separate MANOVA was conducted to explore mean differences in DSF and DSB subtest scaled score between groups. This analysis was conducted separately because these scores were not available for each subject, as the other subtest scores were. DSF and DSB scores were available for 42 of the 62 nonmedicated ADHD subjects and for 33 of the 49 medicated ADHD subjects. DSF and DSB scores were available for all

62 subjects in Control Group 1 and for all 49 subjects in Control Group 2. The results of the MANOVA are presented in Table 22.

Table 22

Multivariate Analysis for DSF and DSB Scores

Subtest Scores	*SS*	*Df*	*MS*	*F*	*Sig.*	η^2
DSF	67.178	3	22.393	2.427	0.067	0.038
DSB	34.747	3	11.582	1.600	0.191	0.026

There were no statistically significant differences in mean scaled scores on the DSF and DSB subtests between groups in this study.

Tests of Hypotheses 7 and 8. To test the research questions and hypotheses regarding ADHD diagnosis and Index score splits, the following variables were calculated for each subject: VCI-WMI differences; GAI-WMI differences; PRI-PSI differences, and GAI-PSI differences. Cumulative percentages were then obtained for differences at the following magnitudes: 10 points, 15 points, 20 points, 25 points, 30 points, 35 points, -10 points, -15 points, -20 points, -25 points, -30 points, and -35 points. Cumulative percentages were then converted to n values. The n values were then utilized to compare the significance between proportions, using Fisher's Exact Test. The z values and significance levels of these analyses are reported in Tables 23 though 30.

Table 23.1

Frequency of VCI > WMI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	23	13	22	7
15 points	12	7	12	5
20 points	7	4	6	3
25 points	2	1	2	2
30 points	2	1	1	0
35 points	1	1	0	0

Table 23.2

Fisher's Exact Test for VCI > WMI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	1.978	0.024*	3.320	0.001*
15	1.247	0.106	1.867	0.031*
20	1.215	0.112	1.049	0.147
25	1.017	0.155	0.000	0.500
30	1.017	0.155	1.005	0.157
35	0.585	0.280	--	--

Note. Items with an asterisk are statistically significant.

The frequency of subjects evidencing greater VCI than WMI scores tended to be greater for the ADHD groups than for their matched controls at all levels. To test the significance of these frequency differences, Fisher's Exact Test was utilized to compare the proportion of subjects in each group who evidenced VCI scores greater than WMI scores at each level of difference (i.e., 10, 15, 20, 25, 30 and 35). The results of these calculations reveal significantly more 10 point splits in the nonmedicated ADHD group than in Control Group 1. Specifically 37.1% (n = 23) of the nonmedicated ADHD group evidenced 10 point VCI-WMI splits, but 21% (n = 13) of Control Group 1 evidenced 10 point VCI-WMI splits. Significantly more 10 and 15 point VCI-WMI splits were found in the medicated ADHD group (10 point split = 44.9%; 15 point split = 24.5%) than in Control Group 2 (10 point split = 14.3%; 15 point split = 10.2%).

Table 24.1

Frequency of WMI > VCI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	13	20	8	17
15 points	8	9	4	10
20 points	2	3	3	7
25 points	0	2	0	2
30 points	0	2	0	1
35 points	0	0	0	1

Table 24.2

Fisher's Exact Test for WMI > VCI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	-1.422	0.078	-2.086	0.019*
15	0.000	0.500	-1.732	0.042*
20	0.000	0.500	-1.335	0.091
25	-1.426	0.077	-1.429	0.077
30	-1.426	0.077	-1.005	0.157
35	--	--	-1.005	0.157

Note. Items with an asterisk are statistically significant.

The frequency of subjects with WMI scores greater than VCI scores tended to be greater for the non-ADHD controls than for the ADHD groups at all levels. Fisher's Exact Test was utilized to determine the significance of these differences in frequencies across groups. These calculations indicated that there were significantly more 10 and 15 point splits in Control Group 2 than in their ADHD counterparts (medicated ADHD group). Slightly more than twice as many subjects in Control Group 2 evidenced 10 (34.7%) and 15 (20.4%) point WMI-VCI splits than in the medicated ADHD group (10 point split = 16.3%; 15 point splits = 8.2%). There were no significant differences between the nonmedicated ADHD group and their matched controls.

Table 25.1

Frequency of GAI > WMI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	24	20	23	12
15 points	13	11	9	8
20 points	9	7	5	3
25 points	5	0	2	2
30 points	4	0	0	0
35 points	2	0	0	0

Table 25.2

Fisher's Exact Test GAI > WMI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	0.751	0.226	2.319	0.010*
15	0.455	0.325	0.267	0.395
20	0.536	0.296	0.738	0.230
25	2.283	0.011*	0.000	0.500
30	2.033	0.021*	--	--
35	1.426	0.077	--	--

Note. Items with an asterisk are statistically significant.

The frequency of cases evidencing GAI scores greater than WMI scores was greater in the ADHD groups, compared with their matched controls at almost all levels. To determine the significance of the differences in frequencies among groups, Fisher's Exact Test was utilized to compare the proportion of subjects in each group evidencing GAI scores greater than the WMI scores. Comparisons revealed that there were significantly more subjects in the nonmedicated ADHD group with GAI-WMI splits of 25 (8.1%) and 30 (6.5%) points than in Control Group 1, which had no instances of 25 or 30 point splits. Almost twice as many medicated ADHD subjects had GAI-WMI splits of 10 points (46.9%) than subjects in Control Group 2 (24.5%).

Table 26.1

Frequency of WMI > GAI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	13	16	6	12
15 points	7	5	1	6
20 points	5	3	0	3
25 points	2	1	0	1
30 points	0	0	0	0
35 points	0	0	0	0

Table 26.2

Fisher's Exact Test WMI >GAI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	-0.637	0.262	-1.565	0.059
15	0.608	0.728	-1.961	0.025*
20	0.731	0.768	-1.759	0.039*
25	0.585	0.721	-1.005	0.157
30	--	--	--	--
35	--	--	--	--

Note. Items with an asterisk are statistically significant.

Comparing the frequency of cases between groups with WMI scores greater than GAI was not as consistent. There was no predictable pattern between the nonmedicated ADHD group and their controls. On the other hand, the ADHD medicated group tended to evidence lower frequencies when compared with their matched controls. To determine the significance of the differences in frequency, Fisher's Exact Test was utilized to compare proportions of subjects with WMI scores greater than GAI scores. These calculations reveal that significantly more 10 and 15 point differences occurred in Control Group 2 compared with the medicated ADHD group. Specifically, approximately twice as many controls evidenced 10 (34.7% vs. 16.3%) and 15 (20.4% vs. 8.2%) point splits when compared to their medicated ADHD counterparts. There were

no significant differences between the nonmedicated ADHD group and their matched controls.

Table 27.1

Frequency of PRI >PSI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	20	21	24	16
15 points	14	18	21	10
20 points	11	10	14	6
25 points	7	6	6	4
30 points	4	3	4	1
35 points	3	2	4	1

Table 27.2

Fisher's Exact Test PRI > PSI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	-0.191	0.576	1.644	0.050
15	-0.821	0.794	2.389	0.008*
20	0.239	0.405	2.005	0.023*
25	0.293	0.384	0.667	0.252
30	0.389	0.349	1.377	0.084
35	0.457	0.324	1.377	0.084

Note. Items with an asterisk are statistically significant.

The frequency of cases evidencing greater PRI scores than PSI scores was generally higher for the ADHD groups than for their controls. The results of Fisher's Exact Test revealed that significantly more medicated ADHD subjects evidenced 15 and 20 point PRI-PSI splits than their matched controls. Specifically, over two times as many medicated ADHD subjects evidenced 15 points splits (42.9%) than their matched controls (20.4%). Over twice as many medicated ADHD subjects also evidenced 20 point PRI-PSI splits (28.6%) compared with their non-ADHD counterparts (12.2%). There were no statistically significant differences between the nonmedicated ADHD group and their matched controls.

Table 28.1

Frequency of PSI > PRI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	9	15	6	10
15 points	6	9	6	6
20 points	4	6	3	3
25 points	2	2	3	3
30 points	0	2	2	0
35 points	0	1	0	0

Table 28.2

Fisher's Exact Test PSI > PRI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	-1.364	0.086	-1.093	0.137
15	-0.826	0.204	0.000	0.500
20	-0.660	0.255	0.000	0.500
25	0.000	0.500	0.000	0.500
30	-1.426	0.077	1.429	0.924
35	-1.004	0.158	--	--

The frequency of cases in Control Group 1who evidenced stronger PSI than PRI scores tended to be slightly greater than in their ADHD counterparts (nonmedicated ADHD group); however, the frequency of cases with stronger PSI scores was fairly similar between the medicated ADHD group and their controls. Z values were computed to determine whether or not any statistically significant differences occurred between groups. The results of this computation did not yield any significant differences between the proportions of cases with PSI scores greater than PRI scores in each group.

Table 29.1

Frequency of GAI > PSI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	21	18	25	14
15 points	13	12	16	9
20 points	10	6	15	7
25 points	7	4	12	4
30 points	3	3	4	2
35 points	2	1	3	1

Table 29.2

Fisher's Exact Test GAI > PSI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	0.580	0.281	2.270	0.012*
15	0.224	0.411	1.622	0.052
20	1.072	0.142	1.937	0.026*
25	0.948	0.172	2.186	0.014*
30	0.000	0.500	0.843	0.200
35	0.585	0.280	1.021	0.154

Note. Items with an asterisk are statistically significant.

The frequency of cases with GAI scores greater than PSI scores was higher for the ADHD groups at all levels than for their matched controls. The significance of differences in proportions between groups was tested by calculating z values, using Fisher's Exact Test. These calculations found statistically significant differences between the medicated ADHD group and their matched controls at the 10, 20 and 25 point levels. Just over twice as many medicated ADHD subjects (30.6%) as subjects in Control Group 2 (14.3%) evidenced 20 point GAI-PSI splits. Three times as many medicated ADHD subjects (24.5%) evidenced 25 point splits when compared with Control Group 2 (8.2%). There were no statistically significant differences between the nonmedicated ADHD group and their controls.

Table 30.1

Frequency of PSI > GAI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	10	15	6	9
15 points	7	11	4	6
20 points	4	8	3	4
25 points	3	3	2	3
30 points	0	2	0	3
35 points	0	0	0	1

Table 30.2

Fisher's Exact Test PSI > GAI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	-1.119	0.132	-0.842	0.200
15	-1.020	0.154	-0.667	0.252
20	-1.214	0.112	-0.392	0.347
25	0.000	0.500	-0.459	0.323
30	-1.426	0.077	-1.759	0.039*
35	--	--	-1.005	0.157

Note. Items with an asterisk are statistically significant.

The frequency of cases with PSI scores greater than GAI scores tended to be greater at all levels for the controls than for the ADHD groups. To compare the significance of differences in the proportions among groups, Fisher's Exact Test was utilized. These calculations revealed that significantly more controls (6.1%) evidenced 30 point PSI-GAI differences than their medicated ADHD counterparts (n = 0).

Comparisons of Other Factor Scores. Statistical analysis was also conducted to evaluate splits between other factor scores. Specifically, VCI-PRI splits and WMI-PSI splits were examined. Given the fact that that no differences were predicted, the Fisher's Exact Test evaluated the null hypothesis utilizing a two-sided z-test. Tables 31 and 32 contain the frequencies and results of Fisher's Exact Test.

Table 31.1

Frequency of VCI > PRI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	8	8	10	6
15 points	4	5	8	2
20 points	1	4	3	0
25 points	1	2	2	0
30 points	0	1	2	0
35 points	0	0	0	0

Table 31.2

Fisher's Exact Test VCI >PRI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	0.000	1.000	1.093	0.274
15	0.346	0.729	2.002	0.045*
20	1.370	0.171	1.759	0.079
25	0.584	0.559	1.429	0.153
30	1.004	0.315	1.429	0.153
35	--	--	--	--

Note. Items with an asterisk are statistically significant.

The proportions of nonmedicated ADHD subjects and subjects in the matched control group with VCI scores greater than PRI scores were similar at all levels. The proportion of medicated ADHD subjects with VCI scores greater than PRI scores was significantly greater than their matched controls at the 15 point level. Approximately four times the number of medicated ADHD subjects had VCI-PRI splits of 15 points when compared with their matched controls (16.3% versus 4.1%).

Table 31.3

Frequency of PRI > VCI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	8	23	11	11
15 points	4	18	7	7
20 points	1	7	5	4
25 points	1	3	3	3
30 points	0	0	1	3
35 points	0	0	0	1

Table 31.4

Fisher's Exact Test PRI >VCI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	3.111	0.002*	0.000	1.000
15	3.291	0.001*	0.000	1.000
20	2.193	0.029*	0.350	0.727
25	1.017	0.309	0.000	1.000
30	--	--	1.021	0.307
35	--	--	1.005	0.315

Note. Items with an asterisk are statistically significant.

The proportion of subjects in Control Group 1with PRI scores 10, 15 and 20 points greater than their VCI scores was significantly greater than the proportion of subjects from the nonmedicated ADHD group with these differences. Specifically, 37.1% of Control Group 1 had PRI score greater than VCI scores by 10 points, but only 12.9% of the nonmedicated ADHD group did. Of Control Group 1, 29% also displayed PRI scores greater than VCI scores by 15 points, but 6.5% of the nonmedicated ADHD group displayed this level of difference. Finally, 11.3% of Control Group 1 displayed PRI scores 20 points greater than VCI scores, compared with 1.6% of the nonmedicated ADHD group. There were no statistically significant differences between the medicated ADHD group and their matched controls.

Table 32.1

Frequency of WMI > PSI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	20	17	20	18
15 points	8	10	13	9
20 points	3	5	9	6
25 points	0	3	7	5
30 points	0	2	3	2
35 points	0	1	3	2

Table 32.2

Fisher's Exact Test WMI >PSI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	0.589	0.556	0.415	0.678
15	0.510	0.610	0.968	0.333
20	0.731	0.465	0.842	0.400
25	1.753	0.080	0.616	0.538
30	1.426	0.154	0.459	0.646
35	1.004	0.315	0.459	0.646

Comparisons were also made between the WMI and PSI. The proportion of nonmedicated ADHD subjects and subjects in Control Group 1with WMI scores greater than PSI scores was comparable at all levels. Similarly, the proportion of medicated ADHD subjects and subjects in Control Group 2 with WMI scores greater than PSI scores was comparable at all levels.

Table 32.3

Frequency of PSI > WMI by Group

	Diagnostic Group			
Index Score Differences	ADHD Non-Med (N=62)	Control 1 (N=62)	ADHD Med (N=49)	Control 2 (N=49)
10 points	17	16	12	14
15 points	8	13	9	10
20 points	6	9	5	2
25 points	4	5	4	1
30 points	2	1	2	0
35 points	1	1	1	0

Table 32.4

Fisher's Exact Test PSI >WMI

	Nonmedicated ADHD vs. Control 1 (n = 62)		Medicated ADHD vs. Control 2 (n = 49)	
Index Score Differences	*z value*	*p value*	*z value*	*p value*
10	0.203	0.839	0.458	0.647
15	1.197	0.231	0.256	0.798
20	0.826	0.409	1.177	0.239
25	0.346	0.729	1.377	0.168
30	0.585	0.559	1.429	0.153
35	0.000	1.000	1.005	0.315

The proportion of nonmedicated ADHD subjects and subjects in Control Group 1with PSI scores greater than WMI scores was comparable at all levels. Similarly, the proportion of medicated ADHD subjects and subjects in Control Group 2 with PSI scores greater than WMI scores was comparable at all levels.

Tests of Hypotheses 9 and 10. To test the research questions and hypotheses regarding the impact of medication status on working memory and processing speed, comparisons were made among VCI-WMI, GAI-WMI, PRI-PSI, and GAI-PSI Index scores splits between the nonmedicated and medicated ADHD groups. Similar to the tests for hypotheses 7 and 8, cumulative percentages and n values were obtained for differences at the following magnitudes: 10 points, 15 points, 20 points, 25 points, 30 points, 35 points, -10 points, -15 points, -20 points, -25 points, -30 points, and -35 points. The n values were then utilized to compute Fisher's Exact Test to compare the significance between proportions. Tables 33 through 40 contain the z values and significance levels for these comparisons.

Table 33

Fisher's Exact Test VCI > WMI

Nonmedicated vs. Medicated (n = 62; n = 49)		
Index Score Differences	*z value*	*p value*
10 points	-0.831	0.797
15 points	-0.653	0.743
20 points	0.104	0.458
25 points	0.191	0.424
30 points	0.785	0.216
35 points	0.382	0.351

Note. Items with an asterisk are statistically significant.

Table 34

Fisher's Exact Test WMI > VCI

Nonmedicated vs. Medicated (n = 62; n = 49)		
Index Score Differences	*z value*	*p value*
10 points	0.620	0.732
15 points	1.034	0.849
20 points	-0.297	0.383
25 points	--	--
30 points	--	--
35 points	--	--

The frequency of subjects evidencing VCI and WMI splits in either direction was similar across ADHD groups.

Table 35

Fisher's Exact Test GAI > WMI

	Nonmedicated vs. Medicated (n = 62; n = 49)	
Index Score Differences	*z value*	*p value*
10 points	-0.871	0.808
15 points	0.341	0.366
20 points	0.680	0.248
25 points	0.857	0.195
30 points	1.811	0.035*
35 points	1.269	0.102

Table 36

Fisher's Exact Test WMI > GAI

	Nonmedicated vs. Medicated (n = 62; n = 49)	
Index Score Differences	*z value*	*p value*
10 points	1.212	0.887
15 points	1.871	0.969
20 points	2.034	0.979
25 points	1.269	0.897
30 points	--	--
35 points	--	--

The frequency of cases evidencing GAI scores greater than WMI scores was slightly greater in the nonmedicated ADHD group than in the medicated ADHD group. To determine the significance of the differences in frequencies among groups, Fisher's Exact Test was utilized to compare the proportion of subjects. Comparisons revealed that there were significantly more subjects in the nonmedicated ADHD group with GAI-WMI splits of 30 points (6.5%, n = 4) than in the medicated ADHD group (n = 0).

The frequency of cases with WMI scores greater than GAI scores was slightly higher in the nonmedicated ADHD group than in the medicated ADHD. Fisher's Exact Test revealed no statistically significant differences.

Table 37

Fisher's Exact Test PRI > PSI

	Nonmedicated vs. Medicated (n = 62; n = 49)	
Index Score Differences	*z value*	*p value*
10 points	-1.788	0.963
15 points	-2.283	0.988
20 points	-1.356	0.912
25 points	-0.155	0.561
30 points	-0.346	0.635
35 points	-0.716	0.762

Table 38

Fisher's Exact Test PSI >PRI

Nonmedicated vs. Medicated (n = 62; n = 49)		
Index Score Differences	*z value*	*p value*
10 points	0.348	0.635
15 points	-0.433	0.332
20 points	0.071	0.528
25 points	-0.731	0.528
30 points	-1.601	0.054
35 points	--	--

The frequency of cases evidencing greater PRI scores than PSI scores was slightly higher for the medicated ADHD group than for the nonmedicated ADHD group, opposite of what was predicted. Therefore, Fisher's Exact Test calculations did not reveal any statistically significant findings, utilizing the assumptions of the research hypothesis. If the Fisher's Exact Test is run with the opposite hypothesis, the medicated ADHD group actually displays significantly larger proportions of students with PRI scores at least 10 points ($p = 0.037$) or 15 points ($p = 0.011$) larger than PSI, relative to the nonmedicated ADHD group.

The frequency of cases with PSI scores greater than PRI scores was fairly similar across the nonmedicated and medicated ADHD groups. No statistically significant differences were found.

Table 39

Fisher's Exact Test GAI > PSI

Nonmedicated vs. Medicated (n = 62; n = 49)		
Index Score Differences	*z value*	*p value*
10 points	-1.821	0.965
15 points	-1.392	0.918
20 points	-1.814	0.965
25 points	-1.833	0.966
30 points	-0.716	0.762
35 points	-0.731	0.767

Table 40

Fisher's Exact Test PSI > GAI

Nonmedicated vs. Medicated (n = 62; n = 49)		
Index Score Differences	*z value*	*p value*
10 points	0.579	0.718
15 points	0.548	0.708
20 points	0.071	0.528
25 points	0.191	0.575
30 points	--	--
35 points	--	--

The frequency of cases with GAI scores greater than PSI scores was slightly higher for the medicated ADHD group than for the nonmedicated ADHD group, which was opposite of the finding predicted. Therefore, no statistical significant differences were found in support of the hypothesis. The frequency of cases with PSI scores greater than GAI scores was fairly similar across groups and statistical comparisons did not reveal any significant differences.

Table 41 contains a summary of the significant Index score splits found across groups.

Table 41

Summary of Significant Index Score Splits

VCI > WMI		
Nonmedicated ADHD vs. Control 1*	Medicated ADHD vs. Control 2*	Nonmedicated vs. Medicated
10 points	10, 15 points	None
WMI > VCI		
Nonmedicated ADHD vs. Control 1	Medicated ADHD vs. Control 2**	Nonmedicated vs. Medicated
None	10, 15 points	None
GAI > WMI		
Nonmedicated ADHD vs. Control 1*	Medicated ADHD vs. Control 2*	Nonmedicated vs. Medicated ***
25 & 30 points	10 points	30 points

WMI > GAI		
Nonmedicated ADHD vs. Control 1	Medicated ADHD vs. Control 2**	Nonmedicated vs. Medicated
None	15, 20 points	None
PRI > PSI		
Nonmedicated ADHD vs. Control 1	Medicated ADHD vs. Control 2*	Nonmedicated vs. Medicated ****
None	15, 20 points	10, 15 points
PSI > PRI		
Nonmedicated ADHD vs. Control 1	Medicated ADHD vs. Control 2	Nonmedicated vs. Medicated
None	None	None
GAI > PSI		
Nonmedicated ADHD vs. Control 1	Medicated ADHD vs. Control 2*	Nonmedicated vs. Medicated
None	10, 20, 25 points	None
PSI > GAI		
Nonmedicated ADHD vs. Control 1	Medicated ADHD vs. Control 2**	Nonmedicated vs. Medicated
None	30 points	None
VCI > PRI		
Nonmedicated ADHD vs. Control 1	Medicated ADHD vs. Control 2*	Nonmedicated vs. Medicated
None	15 points	--

PRI > VCI		
Nonmedicated ADHD vs. Control 1**	Medicated ADHD vs. Control 2	Nonmedicated vs. Medicated
10, 15, 20 points	None	--
WMI > PSI		
Nonmedicated ADHD vs. Control 1	Medicated ADHD vs. Control 2	Nonmedicated vs. Medicated
None	None	--
PSI > WMI		
Nonmedicated ADHD vs. Control 1	Medicated ADHD vs. Control 2	Nonmedicated vs. Medicated
None	None	--

Note. Dashes indicate that comparison was not computed.

Proportion of ADHD groups greater than non-ADHD groups. **Proportion of non-ADHD group greater than ADHD groups. * Proportion of nonmedicated ADHD group greater than medicated ADHD group. ****Proportion of medicated ADHD group greater than nonmedicated ADHD group.*

Chapter 4

Discussion

Summary of Results

The current study intended to examine the effects of ADHD and medication use on the cognitive processing of children. This chapter includes a discussion of the results in relation to the research questions of the study and hypotheses proposed. The contributions to the field of psychology, limitations of the study, and recommendations for future research are also addressed in this chapter.

The first set of research questions were designed to replicate Friedman's 2006 study. Specifically, the first and second research questions involved an investigation of the impact of ADHD diagnosis on FSIQ scores and factor scores (VCI, PRI, WMI, and PSI) of the WISC-IV. Similar to Friedman's (2006) study and in line with the hypothesis, the current study did not find any significant differences between the mean FSIQ scores of the two ADHD groups and their matched controls. Additionally, it was predicted that there would be no significant differences between the ADHD groups and their non-ADHD counterparts on the VCI, PRI, or PSI, but that there would be significant differences between the ADHD groups and non-ADHD groups on the WMI. As predicted, the results of the statistical analysis found no significant differences between the mean VCI and PRI scores of the ADHD groups compared with their matched controls. Statistical comparisons did reveal that the medicated ADHD group had significantly lower mean WMI and PSI scores than their matched controls.

The third research question involved an examination of how ADHD individuals perform, relative to non-ADHD individuals on each subtest of the WISC-IV. Consistent with Friedman's (2006) finding, it was predicted that the mean scores of ADHD students would be comparable with the mean scores of their non-ADHD counterparts on the verbal reasoning subtests (VO, CO, SI), perceptual reasoning subtests (BD, MR, and PCn), and processing speed subtests (CD and SS). Some differences were expected between the ADHD groups and their non-ADHD controls on the working memory subtests (DS and LNS). As hypothesized, no significant differences were found between groups on the verbal reasoning or perceptual reasoning subtests. Statistical analysis did find significant differences on the Letter Number Sequencing, Coding and Symbol Search subtests. Specifically, the nonmedicated ADHD group evidenced significantly lower mean scores on the LNS and CD subtests than their matched controls. The medicated ADHD group evidenced significantly lower mean scores on the CD and SS subtests than their matched controls.

The fourth and fifth research questions set out to investigate how medication status impacts WISC-IV FSIQ and factor scores of students with ADHD. It was hypothesized that the findings in this area would replicate Friedman's findings so that no significant differences would be found between the mean FSIQ scores or factor scores of the medicated ADHD group and the nonmedicated ADHD group. The results of this study supported these hypotheses because the ADHD medicated and nonmedicated groups performed comparably on the WISC-IV FSIQ, VCI, PRI, WMI, and PSI.

The sixth research question sought to investigate the impact of medication on the performance of ADHD students on the WISC-IV core subtests. Again, it was predicted

that this study would replicate Friedman's results. Specifically, no significant differences were hypothesized between the mean subtests scores of the ADHD medicated and nonmedicated groups for any of the 10 core WISC-IV subtests. The results of this study supported the hypothesis because no significant differences between ADHD groups were found across subtests.

A second set of research questions specific to the current study were also investigated. These questions and hypotheses were based on the reviewed literature, that overwhelmingly suggests that executive control deficits related to working memory and processing speed are a core feature of ADHD and are related to neuropsychological factors. Although the current study did find significant differences between the medicated ADHD subjects and their matched controls on the WMI and PSI factors, the effect sizes were small and significant differences were not found between the nonmedicated ADHD group and their matched controls. There continued to be a question, however, about how well the ADHD subjects performed on measures of working memory and processing speed, relative to measures of other cognitive abilities. Perhaps more significant working memory and processing speed impairments would be found in ADHD students when these impairments are compared with verbal and nonverbal reasoning skills. Thus, the second investigation was proposed to evaluate this question. This research question was explored by comparing the Working Memory Index and Verbal Comprehension Index score splits, the Working Memory Index and General Ability Index score splits, the Processing Speed Index and Verbal Comprehension Index score splits, and the Processing Speed Index and General Ability Index score splits between the ADHD groups and their non-ADHD controls.

Relative to the working memory splits, it was hypothesized that the proportion of ADHD subjects with greater VCI and GAI scores relative to WMI scores would be larger than the proportion of subjects in the matched control group with this pattern. The results of the current study partially supported the hypothesis by finding that compared with their matched controls, the nonmedicated ADHD group had significantly more cases of VCI scores at least 10 points greater than WMI scores. The medicated ADHD group also demonstrated significantly more cases of VCI scores at least 10 and 15 points greater than WMI score when compared with their matched controls. It is important to note that at other levels, even though the results were nonsignificant , the trend of the data supported the stated hypothesis. Cases in which the WMI was greater than the VCI happened more frequently in the non-ADHD groups. Specifically, there were significantly more cases of WMI scores at least 10 or 15 points greater than VCI scores in Control Group 2 than in their nonmedicated ADHD counterparts. The results of this study also found more cases of GAI scores greater than WMI scores in the ADHD groups than in the non-ADHD controls. Specifically, the nonmedicated ADHD group had significantly more cases of GAI scores that were at least 25 and 30 points greater than WMI scores, relative to their matched controls. The medicated ADHD group had larger proportions of GAI scores, at least 10 points greater than WMI scores relative to their non-ADHD counterparts. At other levels, even though not statistically significant, the trend of the data supported the stated hypothesis. In contrast, there were more instances of WMI scores greater than GAI scores by 15 or 20 points in the non-ADHD Control Group 2 than in their nonmedicated ADHD counterparts.

Relative to the Processing Speed Index splits, it was predicted that there would be more cases of PRI scores greater than PSI scores in the ADHD groups than in the non-ADHD control groups. Results of statistical analysis partially support the hypothesis. There were significantly more cases of PRI scores that were at least 15 and 20 points greater than PSI scores in the medicated ADHD group, relative to their non-ADHD counterparts. At other levels, even though the findings were not statistically significant, the trend of the data supported the stated hypothesis. Similarly, it was predicted that there would be more occurrences of GAI scores greater than PSI scores in the ADHD groups than in their non-ADHD counterparts. This hypothesis was also partially supported in the current study, because there were significantly more subjects in the medicated ADHD group with GAI scores who were at least 10, 20, and 25 points greater than PSI scores relative to their non-ADHD counterparts. Also of some note, is the fact that at other levels, the trend of the data supported the stated hypothesis, even though not at a statistically significant level. In contrast, there were significantly more occurrences of PSI scores at least 30 points greater than GAI scores in the non-ADHD control group than in the medicated ADHD group.

Additional analyses were conducted to explore whether or not any pattern of differences occurred between the VCI and PRI and between the WMI and PSI among groups. The groups were predicted to have similar proportions of subjects with VCI scores greater than and less than PRI scores and with WMI scores greater than or less than PRI scores. Consistent with the hypothesis, the proportions of students with WMI scores greater than or less than PSI scores were found to be statistically similar among groups. Inconsistent with the hypotheses, the medicated ADHD group had a significantly

greater proportion of subjects with VCI scores at least 15 points higher than PRI scores relative to their non-ADHD control group. The non-ADHD Control Group 1 had more cases of PRI scores at least 10, 15 or 20 points greater than VCI scores. Although this finding was not predicted, a possible explanation of these differences could be that the symptoms of ADHD are more likely to impact performance on the perceptual reasoning tasks than on the verbal comprehension tasks. For example, the impulsivity and quick response style symptomatic of ADHD may be more likely to hinder the greater attention to detail that is required on the perceptual reasoning subtests. Further research is needed to investigate this hypothesis.

In addition to comparing ADHD groups with non-ADHD groups, this study also sought to evaluate whether or not differences occur in the cognitive profiles of children with ADHD who are medicated versus those who are not. The first research question related to this question stated, "To what extent does stimulant medication impact the working memory capacity of ADHD children?" This question was explored by comparing the VCI and WMI splits between the medicated ADHD group and the non-medicated ADHD group. GAI and WMI score splits were also compared among groups. Given the fact that medication treats the symptoms of ADHD, it was predicted that the number of cases with VCI scores greater than WMI scores would be larger for the nonmedicated or untreated ADHD group than for the medicated ADHD group. This hypothesis was not supported. In fact, the proportion of subjects in the nonmedicated ADHD group with VCI scores greater than WMI scores was comparable with the proportions of subjects with this difference in the medicated ADHD group. There were also no significant differences among groups relative to the proportion of cases with

WMI scores greater than VCI scores. Relative to the GAI- WMI splits, it was predicted that the proportion of cases with GAI scores greater than WMI scores would be larger for the nonmedicated ADHD group than for the medicated ADHD group. Results partially supported this hypothesis. There were significantly more nonmedicated ADHD subjects than medicated ADHD subjects with GAI scores at least 30 points greater than WMI scores. The proportion of subjects with WMI scores greater than GAI scores was comparable among groups.

The final research question sought to examine the extent to which stimulant medication impacts processing speed in ADHD children. This question was explored by examining the PSI scores versus measures of other cognitive abilities for medicated ADHD individuals versus nonmedicated ADHD individuals. It was predicted that the number of cases with PRI scores greater than PSI scores would be larger in the nonmedicated group than in the medicated group. This hypothesis was not supported. There were actually significantly more students in the medicated ADHD group with PRI scores greater than PSI scores by at least 10 or 15 points. The proportion of cases with PSI scores greater than PRI scores was similar across ADHD groups. Finally, it was predicted that the number of subjects with GAI scores greater than PSI scores would be larger in the nonmedicated ADHD group than in the medicated ADHD group. This hypothesis was also not supported. The proportion of students with GAI scores greater than PSI scores was similar across groups.

Significance of the Results

The current study replicated Friedman's (2006) finding that the FSIQ scores of ADHD students are comparable with those of non-ADHD students. This finding is

contrary to previous research that found lower FSIQ scores in ADHD samples (i.e., Barkley, 1990; Barkley, DuPaul & McMurray, 1992, Faraone, et al., 1993; Tripp, Ryan & Peace, 2002; Zhuang, Liu & Zhang, 2001; and Zimmerman & Woo-Sam, 1997). Although processing speed and working memory, thought to be most vulnerable to symptoms of ADHD, weigh more heavily on WISC-IV FSIQ calculations than previous versions of the Wechsler Scales, they still contribute less weight than the verbal reasoning and perceptual reasoning subtests. Specifically, working memory and processing speed each make up 20% of FSIQ, and verbal reasoning and perceptual reasoning each make up 30% of FSIQ. This difference in weight makes it less likely that working memory and processing speed deficits will be reflected in significantly lower FSIQs.

Friedman (2006) attributed her lack of significant findings related to FSIQ in ADHD versus non-ADHD subjects, which was contrary to previous research, to methodological differences. Unlike the previous studies, Friedman matched ADHD and non-ADHD subjects on demographics and ability levels through the VCI, whereas matching in other studies occurred only on demographic variables. She suggested that had ability levels been allowed to vary in an uncontrolled manner, results may have possibly been different. Similar to Friedman's study, this study also matched controls on the ability level through VCI and supports her suggestion that under the more rigorous condition of matching subjects by VCI, the addition of PRI, WMI and PSI tasks did not result in decreases in the FSIQ of ADHD children relative to non-ADHD individuals.

The current study also replicated Friedman's (2006) finding that medication did not have a significant impact on FSIQ. There were no differences between the FSIQ's of

the medicated ADHD group and nonmedicated ADHD group. These findings are in contrast to previous studies which have suggested that the use of medication could have a significant effect on FSIQ score of ADHD children (i.e., Faraone, 2003; Gillberg, et. al., 1997). Friedman attributed the inconsistency to methodological differences, such as lack of random assignment to medicated/ nonmedicated group comparisons and possibly to small effect sizes of significant group differences. Further, Gillberg and colleagues (1997) used a long-term, placebo-controlled study that found improved results on the WISC-R with medicated ADHD children; however, the sample size was small, and type II error could not be excluded as a possible source of the differences. The current study utilized procedures similar to Friedman; therefore, differences between the current study and previous studies showing contrary findings may also be attributed to methodological differences.

At the Index level, this study found significant differences among the ADHD groups and their controls on WMI and PSI, specifically between the medicated ADHD group and their matched controls. On the WMI, although overall scores were not significantly different between the nonmedicated ADHD group and their matched controls, there were significant differences between groups on the LNS subtest. This finding is somewhat consistent with the literature (Kail & Salthouse, 1994; Kail & Salthouse, 1994; Kail, 2000; Kalff et al., 2002; Karatekin & Asarnow, 1998; Martinussen et al., 2005; Rucklidge & Tannock, 2001; Weiler et al., 2002), suggesting that ADHD is associated with problems in verbal working memory and slower retrieval speed. It is also consistent with studies utilizing the WISC-III (Anastopoulos et al., 1994; Mayes et al., 1998; Reinecke, Beebe, & Stein, 1999) and WISC-IV (J. Friedman, 2006; J. Friedman,

2006; Wechsler, 2003), which found WMI scores to be weaker in ADHD subjects than in non-ADHD groups. Although the current findings are in line with previous research, more significant differences were expected at the subtest level than were actually found, specifically on the Digit Span subtest. Previous research using the WISC-III found that ADHD subjects had lower mean scores on the Digit Span subtest (Kaufman, 1994; Mayes et al., 1998; Wechsler, 1991; Wechsler, 1991) than non-ADHD subjects. On the other hand, the WISC-IV standardization studies comparing the ADHD group with other groups found only small effect sizes for Digit Span (Wechsler, 2003). Furthermore, Friedman (2006) found some significant differences on the Digit Span subtest between the ADHD and non-ADHD groups, although further analysis found that this difference was related to performance only on Digit Span Forward.

Also, in support of the research implicating weaknesses in working memory in ADHD subjects, this study found that subjects with ADHD were more likely than their non-ADHD controls to exhibit Working Memory Index scores relatively lower than measures of other cognitive abilities such as the Verbal Comprehension and the combination of verbal comprehension and perceptual reasoning tasks in the form of the General Ability Index. This is consistent with previous research, using the WISC-III, which found that ADHD subjects scored relatively weaker on the Freedom from Distractibility Index than on other factors, such as the Verbal Comprehension and Perceptual Organization Index (Anastopoulos et al., 1994; Krane & Tannock, 2001; Reinecke et al., 1999). In the current study, cases of WMI scores greater than the VCI and/ or GAI scores occurred more frequently in the non-ADHD groups.

On the Processing Speed Index, the medicated ADHD group had significantly lower PSI scores than their matched controls. At the subtest level, there were significant differences between both the nonmedicated and medicated ADHD groups and their matched controls on the CD subtest, but only between the medicated ADHD group and their matched controls on the SS subtest. This is in contrast to Friedman's findings, which did not reveal significant PSI differences between groups. This finding is, however, in line with the results of the WISC-IV clinical study reported in the standardization manual (Wechsler, 2003), in which there was a moderate effect size for group mean differences on the PSI. The standardization study of the WISC-IV (Wechsler, 2003) also found one of the largest effect sizes at the subtest level for group mean scaled score differences on the Coding subtest. Other research (Krane & Tannock, 2001; Mayes, Calhoun & Crowell, 1999) also found significantly lower mean subtest scores for ADHD groups on the processing speed subtests of Coding and Symbol Search.

Further support of the research suggesting that processing speed is impacted in students with ADHD, is offered by the current finding that subjects with ADHD were more likely to display lower scores on the Processing Speed Index, relative to their scores on the Perceptual Reasoning and General Ability Index. Specifically, there were significantly more subjects in the medicated ADHD group with PRI scores greater than PSI scores. There were also significantly more medicated ADHD subjects than subjects in the control group who had GAI scores greater than PSI scores. This same finding, however, did not apply to the nonmedicated ADHD group when compared with their controls.

Related to the effects of medication on the cognitive functioning of ADHD students, previous studies evaluating the short-term effects of methylphenidate on WISC-III performance failed to reveal significant treatment effects for subtest, index, or VIQ and PIQ scores (Saklofske & Schwean, 1993; Schwean et al., 1993). In fact, Prifitera and colleagues (2005) speculated that, given the results of studies indicating the lack of medication effects on WISC-III performance, it would be unlikely that the WISC-IV ADHD clinical study, which included a large percentage of children being treated with medication, would find medication effects. The WISC-IV standardization sample, however, was not separated by medication effects; therefore, no conclusions could be drawn about medication effects of ADHD students on the WISC-IV. Friedman's (2006) study also did not find significant medication effects on the FSIQ, Index, or Subtest scores (with the exception of DSF) for subjects with ADHD. On the other hand, Faraone (2003) did report large effect sizes for stimulant medication on intelligence scores; however, other literature supporting this type of effect is sparse.

Consistent with much of the other literature, the current study did not find significant differences on overall measures of cognitive functioning between ADHD students who were medicated versus those who were not. There were no differences in FSIQ or Index scores. There were also no significant differences found between the medicated and nonmedicated subjects at the subtest level. Working Memory Index and Processing Speed Index performance was also compared in nonmedicated and medicated ADHD subjects to measures of other cognitive abilities such as Verbal Comprehension Index, Perceptual Reasoning Index and General Ability Index. Nonmedicated subjects were more likely than medicated subjects to display GAI scores greater than WMI. This

provides some support for the positive effects of medication on working memory, although much more research is needed to make this claim. On the other hand, no support for positive medication effects on processing speed was found in this study. In fact, the medicated subjects were actually more likely than nonmedicated subjects to display PSI scores lower than PRI scores. One reason for the lack of significant findings between the medicated and nonmedicated ADHD groups could be due to sample differences. For example, this study did not match medicated and nonmedicated subjects on any demographic variables or on severity of ADHD symptoms. Thus it is possible that underlying differences between samples on these variables obscured medication effects. The students taking medication often had other comorbid disorders and some were taking multiple medications, suggesting that perhaps their symptoms were greater to begin with. Also important to consider is that medication is more frequently prescribed for hyperactive/ impulsive symptoms than only for inattentive symptoms. Thus it is possible that the medicated ADHD group had more cases of ADHD-HIT or ADHD-CT, whereas the nonmedicated ADHD group may been more heterogeneous, containing more of a mixture of ADHD-HIT, ADHD-IT, and ADHD-CT. Thus both subtype of ADHD and severity of symptoms could be confounding variables. Finally, the number of cases in each group at each level was relatively small, which may also have limited the statistical findings. Further research evaluating medication effects while controlling for other potential confounding variables is needed to improve our understanding of the impact of medication on cognitive functioning.

Contributions to the Field

This study was one of a few that have matched controls to ADHD subjects on both demographic variables and verbal ability levels in order to examine the performance of ADHD students relative to non-ADHD students and to examine the performance of nonmedicated ADHD students relative to medicated ADHD students on measures of intelligence, working memory, and processing speed. Also evaluated in this study was performance on working memory and processing speed relative to measures of other cognitive abilities. This study both replicated and expanded Friedman's research, providing further evidence for the presence of some differences in the cognitive profiles of ADHD students versus students without the disorder. Although not as many significant results as expected were found, there was a clear trend (as evidenced both by significant results and by results approaching significance) of weaker Working Memory Index and Processing Speed Index scores in the ADHD groups relative to their non-ADHD counterparts and relative to their other cognitive abilities such as the abilities to reason with verbal and nonverbal information. In fact, in no cases did the collected data refute the expected pattern of ADHD groups as having greater Index score splits than non-ADHD groups. Although further research with larger sample sizes is needed to confirm this trend, this study coupled with Friedman's study, does suggest that relative weaknesses in working memory and processing speed may be a useful diagnostic marker, when combined with other corroborating data, for ADHD.

This study failed to find many differences between medicated ADHD subjects and nonmedicated ADHD subjects. Because medication has been found to lessen the symptoms of ADHD, it would make sense that this would carry over into improvement in some aspects of cognitive functioning. This study lent some mild support for positive

effects of medication on auditory working memory; however, this same finding did not hold true for processing speed. The finding that medicated ADHD subjects did not evidence less impaired processing speed than nonmedicated ADHD subjects could suggest that medication use does not, in fact, improve visual processing. On the other hand, these results could be related to limitations of the study design and sample characteristics that obscured medication effects. Much more research is needed to investigate processing speed and medication use in ADHD subjects.

Although the findings of this study seem to suggest that students with attentional disorders may be more likely to experience working memory and processing speed problems, it is important to consider individual cases in clinical practice. Not all ADHD students display this trend. Likewise, students with other types of disabilities could display this trend. Therefore considering cognitive patterns along with other data such as classroom behavior, developmental history, academic achievement, parent and teacher input, and executive function capacities is essential for differential diagnosis.

Limitations

Limitations of this study include the fact that cross-sectional comparisons of group data were analyzed rather than longitudinal data for treatment and control groups. Medication effects may be better ascertained by testing an ADHD group prior to and then after medication treatment begins and comparing pre-post differences with a control group.

Another limitation is the method of ADHD diagnosis. ADHD diagnosis of subjects could be made by the clinician assessing the child (i.e., school psychologists) or

through prior evaluation by another provider. Therefore consistency in diagnostic practice cannot be evaluated and may be variable across practitioners.

Only subjects with comorbid mental retardation or VCI scores below 80 were excluded from the study. Subjects with comorbid diagnoses such as learning disabilities, emotional disturbances, and Aspergers Disorder were not excluded in the current study, which raises the possibility that co-occurring disorders may have their own effects on reasoning, working memory, and processing speed.

Given the constraints of the data available, subjects with all three subtypes of ADHD (ADHD-IT, ADHD-HIT, and ADHD-CT) were included in this study and were grouped together. There is some evidence to suggest that ADHD-IT is distinct from ADHD-HIT and ADHD-CT (Barkley, DuPaul & McMurray, 1992), with ADHD-IT possibly having a more negative impact on cognitive processes. Thus, subtype of ADHD can present a confounding variable.

Gender differences were not explored because of difficulty obtaining large samples equally representative of both genders. Given the fact that greater intellectual impairments have been found in girls with ADHD than in boys (Biederman et al., 1999; Gaub & Carlson, 1997), results which include females may yield different results.

This study also did not analyze the impact of different types, name brands, or combinations of medication on cognitive functioning. The medicated ADHD group included students taking a variety of medications/ combinations of medications. Future research is needed to analyze the impact of different medications and combinations of medications on the cognitive functioning of ADHD individuals.

Finally, it is important to note that in conducting the analysis of factor score splits, the n values at each level were relatively small. With larger sample sizes, it is possible that more robust differences between ADHD and non-ADHD groups would be found.

Future Directions for Research

Further research with larger sample size is needed to investigate the VCI/ WMI, GAI/WMI, PRI/ PSI, GAI/PSI splits found in ADHD children. A focus on subtest score patterns will be particularly useful. If the findings of this study are replicated across other samples, clinicians can better understand the cognitive profiles of ADHD children.

There is also a need to evaluate the cognitive profiles both of younger and of older ADHD subjects to see if these findings hold true for all age groups. Research evaluating the cognitive profiles of ADHD females of all ages is needed to understand gender differences. Furthermore, an evaluation of different types and combinations of medication on cognitive functioning is needed to understand further, the impact of medication on cognitive functioning. Finally, an evaluation of the cognitive functioning of ADHD subjects, utilizing intelligence measures other than the Wechsler Scales is indicated. Current practice for diagnosing ADHD is based primarily on behavioral factors, which has many limitations. Understanding the cognitive indicators of ADHD will enhance the diagnostic practice of clinicians in the field.

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